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43RD WORLD SMALL ANIMAL VETERINARY ASSOCIATION CONGRESS AND 9TH FASAVA CONGRESS

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INTRODUCTION

Thank you for choosing the WSAVA Congress as part of your continual learning process and I know these Proceedings will continue to provide valuable teachings as you explore the breadth and depth of the information they contain, all thanks to our speakers who shared their passion and knowledge with us. The WSAVA World Congress is where the global community of companion animal practitioners gathers to share in the joy of learning, friendship, and collegiality.

So please take this unique opportunity to not only refresh your knowledge and passion for our great profession, but to also learn from the collective knowledge of colleagues from around the world. Hoping you leave with new memories, new learnings, and new friends.

Walt Ingwersen DVM, DVSc, DACVIM

WSAVA President

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WSV18-0109

DIAGNOSTIC IMAGING

HOW TO MAXIMIZE THE DIAGNOSTIC VALUE OF SPINAL AND PELVIC RADIOGRAPHS

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HOW TO MAXIMIZE THE DIAGNOSTIC VAUE OF SPINAL AND PELVIC RADIOGRAPHS

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Introduction

There are many indications to perform spinal and pelvic radiographs in dogs and cats, the most common ones including hindlimb lameness, mono- or paraparesis or -plegia, spinal and pelvic pain, trauma to the back or pelvic region. Survey radiographs are almost always warranted even if further imaging such as Computed Tomography or Magnetic Resonance Imaging is pursued. The goal of spinal radiography is to rule out traumatic lesions (fractures, luxations), aggressive lesions such as vertebral tumors and discospondylitis, and to determine if there are degenerative changes or changes to a disc space that could indicate disc disease. Pelvic radiographs are often performed to evaluate for fractures or changes to the coxofemoral joints. Good radiographic technique is of utmost importance in order to detect these lesions with confidence and on the flip side poor radiographic technique can obscure lesions, or just as detrimental, create the appearance of lesions where there are none. Obtaining the right projections is also very important depending on the indication. In this presentation examples of common diseases and pitfalls associated with spinal and pelvic radiography will be shown.

Spinal radiography

As a general rule, heavy sedation or general anesthesia is needed for radiography of the spine. It is rare that a patient is relaxed enough without sedation that the spine can be positioned properly. The exception to this rule is the acute trauma patient where muscle contraction around a fracture should not be compromised with sedation to avoid destabilization. Orthogonal (lateral and ventrodorsal) radiographs are the standard, however there are instances where lateral radiographs are sufficient for example to screen for larger trauma or discospondylitis. Trauma patient should be moved as little as possible and ventrodorsal projections should only be obtained if they can be performed with a horizontal beam. Main problems/artifacts that can hinder interpretation include patient rotation, too large of a field of view and in lateral projections insufficient padding of portions of the spine that naturally deviate towards the table such as the cervical spine and the caudal lumbar spine. Superimposition of structures such as the ear cartilages, the shoulder joints, and collars and harnesses can obscure lesions. Centering the beam to an area of suspected abnormality is very important as for example disc spaces are markedly distorted in the periphery of a large field of view due to the angled, divergent nature of the x-ray beam.

For good quality lateral cervical radiographs the patient should be positioned with the head extended and supported so that it is not rotated (for example by placing a foam wedge underneath the chin). The ears should be pulled forward so that they are not superimposed over the atlantoaxial junction. The thoracic limbs are pulled caudally to avoid superimposition of the shoulder joint over the caudal cervical spine. Padding is added under the caudal cervical spine to have the spine parallel to the x-ray table. In a perfect lateral view there is complete superimposition of the wings of the atlas. This unfortunately leads to poor visibility of the odontoid process of C2 and therefore a slightly oblique projection (head slightly rotated) is a helpful view when atlantoaxial subluxation is suspected as it projects the dens dorsally over the vertebral canal and fragmentation or blunting of the dens would be more easily visible. Extended and flexed views are not recommended when evaluating dogs for atlantoaxial luxation as it could result in spinal cord damage. For a ventrodorsal view the patient is ideally placed in a trough and straightened with the thoracic limbs also pulled caudally. Flexion and extension is occasionally used to evaluate presence of instability in the caudal cervical spine in dogs with suspected cervical spondylomyelopathy. Subtle lesions that involve the pedicles and transverse processes can be highlighted with oblique ventrodorsal projections, this projection also allows evaluation of the intervertebral foramina. Rotation and motion artifact are the main problems when radiographing the thoracic spine. Similar to the caudal cervical spine, padding should be added underneath the lumbar spine directly cranial to the pelvis to ensure that the spine is positioned parallel to the table. The last thoracic vertebrae or lumbosacral junction should be included in all views of the lumbar spine so that lesions can be localized to the correct vertebra. Lateral flexed and extended radiographs of the lumbosacral junction are taken to determine presence of instability at the lumbosacral junction in dogs with suspected cauda equina syndrome. When evaluating the lumbosacral endplates for defects such as discospondylitis or osteochondrosis, a ventrodorsal view with the pelvic limbs in flexed (frog-leg) position is helfpul as it highlights a slightly different portion of the endplate and endplate defects are often better visible than in extended hip views in general.

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Pelvic radiography

A routine pelvic radiographic series should include a lateral and ventrodorsal view. The lateral view is helpful to evaluate concomitant lesions of the caudal lumbar and lumbosacral spine, the pelvic canal and the caudal abdominal soft tissues. Periosteal reactions along the pelvic margins may be seen with prostatic cancer and are often best seen in the lateral view. The coxofemoral joints are more difficult to evaluate as there is superimposition with the pelvis. Ventrodorsal projections can be obtained with the pelvic limbs extended which is the standard view for evaluation of the coxofemoral joints. To evaluate for joint alignment and possible laxity it is very important to have proper positioning without axial rotation, otherwise the coxofemoral joints can artificially look better or worse than they are. Good positioning is recognized by symmetry of the ilial wings, similar size and shape of the obturator foramina and centered position of the spinous processes of the caudal lumbar spine. The femora should be parallel with the patella centered over the femoral trochlear groove. This is only possible in the heavily sedated patient as muscle tone otherwise prevents extension and internal rotation of the proximal pelvic limbs.

Flexed ventrodorsal projections are particularly useful when evaluating the lumbosacral junction for endplate defects, and in dogs and cats with suspected femoral neck fractures. Femoral neck fractures (traumatic or developmental as with slipped capital physes in cats) can be minimally displaced and difficult to detect in extended hip views as the fracture is reduced through positioning.

Symmetric positioning of the pelvis is also very important when evaluating patients for aggressive or traumatic lesions of the pelvis. Slight rotation can occasionally produce an unusual projection of a portion of the pelvis resulting in over- or underinterpreting changes in bone opacity. Other artifacts that can be problematic is superimposition of fecal material in the rectum. Gas lucency within the fecal material my result in the false impression of a radiolucent lytic lesion. It also hinders interpretation of the complex bone structure of the sacrum and cranial coccygeal vertebrae. Presence of gas in the anal sacs results in rounded radiolucent "lesions" superimposed over the ischium and should not be confused with a pathologic aggressive lesion.

Additional, specialized pelvic views to evaluate dogs for hip dysplasia include distraction methods (PennHIP for example) and dorsal acetabular rim views that are mostly used for surgical planning.

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WSV18-0043

SVA REHABILITATION

IS CANINE REHABILITATION AND WHY SHOULD I ADD IT TO MY PRACTICE?

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WHAT IS CANINE REHABILITATION AND WHY SHOULD I ADD IT TO MY PRACTICE?

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Canine rehabilitation is the application of physiotherapeutic techniques to evaluate and treat musculoskeletal impairments in our canine patients. It incorporates the use of objective outcome measures (goniometers, girthometers, etc.), manual assessments (including palpation, joint glides, and neurological assessment), gait analysis, and special tests brought from the field of human physiotherapy. This allows the therapist to tease out the specific structure and tissue type causing the impairments.

The therapist evaluates the presenting complaint, subjective information from the owner, and objective assessment carried out during the examination to create a problem list. Each item on the problem list is addressed in the plan of care.

Therapeutic plans generally involve a combination of manual therapies (joint mobilizations and soft tissue mobilizations), physical modalities (laser, therapeutic ultrasound, e-stim, shockwave), and therapeutic exercises. The modalities are generally used to prepare the tissues for the manual therapies and therapeutic exercises. Physical modalities should never be the sole therapeutic method applied to any patient.

Therapeutic exercise plans are based upon the weight bearing status of the patient, with early interventions focusing upon functional weight bearing exercises, later progressing to functional strengthening exercises. All exercise plans incorporate proprioception, balance, strength, flexibility, and endurance. Exercise equipment includes physioballs (shaped as rolls, peanuts, eggs, donuts and balls), cavaletti poles, therapy band, rocker/wobble boards, and treadmills.

Physical modality parameters are chosen based upon the acuity of the injury. They are used to prepare the tissues for additional therapy and can generally be applied by trained veterinary nurses. The most commonly used physical modalities include neuromuscular electrical stimulation, laser, therapeutic ultrasound,

extracorporeal shock wave therapy, and ice/compression units.Physiotherapy became 'mainstream' globally after WWI when tens of thousands of troops returned home needing assistance in regaining functional independence. At the same time, there was a polio epidemic. Equine rehabilitation became commonplace in the 1960's as horses transitioned from 'beasts of burden' to multimillion-dollar sport horses. Canine rehabilitation has been common throughout Europe and the UK since the '80's and in the U.S. since the '90's.

Veterinarians wishing to offer rehabilitation in their practices must pay attention to the legal issues involved. The terms **physical therapy** and **physiotherapy** are both protected terms globally. This means that only those people who are licensed to practice human physiotherapy may use these terms. Similarly, no one can use the term **veterinary** to describe their practice unless they are a licensed veterinary professional. There is a list serve for those interested in this field: Vetrehab@yahoo.com. This list is managed by Dr. Julie Mayer: drjulie@integrativeveterinarian.com. Veterinarians are encouraged to join the American Association of Rehabilitation Veterinarians (AARV: www.rehabvets.com), a global organization that maintains an archive of current publications related to the field of veterinary rehabilitation. The American College of Veterinary Sports Medicine and Rehabilitation, a specialty college providing board certification to qualified veterinarians was approved by the AVMA in 2010 and received full accreditation from the American Board of Veterinary Specialists earlier this year. There are currently ~240 diplomates in this college.The field of veterinary rehabilitation is being driven, in large part, by client demand. This is not unlike the growth of veterinary acupuncture in the 1980's. One of the largest drivers is the global phenomenon of canine agility competition. Enthusiasts are spending millions of dollars annually on training, equipment, travel and competition, and they are seeking out those veterinarians who understand their sport and offer rehabilitation services. There are many other organized canine sporting events, including puissance, dock diving, 'joring', pulling, ring sports, and flyball, with many of your clients participating in more than one sport with their canine companions. It behooves you to ask about their dog's 'jobs' and to be prepared to address their concerns. Sporting enthusiast make very valuable clients.Government awareness is also driving the demand for sports medicine and rehabilitation therapists. Increasingly, government agencies are seeking out the assistance of those who can help to keep their canine assets in the field, preventing injury, and speeding recovery from injury. Sports medicine specialists focus upon much more than musculoskeletal injuries—recognizing commonly used drugs that might impact the scenting ability of dogs trained to search for explosives or contraband,

understanding the impact of heat and dehydration on search and rescue dogs, helping handlers to choose optimal nutritional plans for working dogs in extreme weather conditions—and the list goes on.There are many models for sports medicine and rehabilitation practice today, everything from veterinarians opening rehabilitation services in conjunction with boarding and 'spa' facilities to large multispecialty practices adding these services to augment their existing surgery, neurology, and internal medicine practices. University veterinary schools are increasingly aware of the need for both clinical services and didactic training in this field. Many rehabilitation therapists are opting to run stand-alone rehabilitation practices while others offer mobile services, offering rehabilitation therapy to multiple practices in their region. Canine sporting events need rehabilitation trained veterinarians on site to care for the athletes. Emergency and critical care practices partner well with sports medicine and rehabilitation practices, allowing for 24-hour care.

Resources

- *American Association of Rehabilitation Veterinarians*
- *rehabvets.org*
- *American College of Veterinary Sports Medicine and Rehabilitation*
- *vsmr.org*
- *American College of Veterinary Surgeons*
- *acvs.org*
- *Veterinary Orthopedic Society*
- *vosdvm.org*
- *Canine Rehabilitation Institute*
- *caninerehabinstitute.com*

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TIPS FROM THE EXPERTS FOR THE APPROACH OF...

THE DOG WITH A HEART MURMUR

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Approach to The dog with a heart murmur
Adrian Boswood

A murmur is likely to be found during careful auscultation. Auscultation is best performed with both the patient and the clinician in a comfortable position. Careful auscultation takes a reasonable period to perform and should involve listening on both sides of the thorax, palpation of the thoracic wall and palpation of the arterial pulse.

In order to characterize a murmur adequately it is first necessary to thing about the normal constituents of the cardiac cycle and how the murmur relates to those normal characteristics.

Audible heart sounds are numbered and referred to as S_1, S_2, S_3 and S_4.

S_1 and S_2 are the only sounds normally audible in the dog. These make up the Lub-Dup normally heard on auscultation. S_1 corresponds to the sound generated in the heart and surrounding structures at the onset of systole on closure of the atrioventricular valves. This is usually the loudest heart sound and is heard best over the left apex. S_2 corresponds to the sound generated in the heart and surrounding structures on closure of the pulmonic and aortic valves. This represents the end of systole and will be loudest at the left heart base.

Between S_1 and S_2 is ventricular systole. Between S_2 and the following S_1 is diastole. Diastole is much more variable in length. The pulse will rise during systole.

S_3 corresponds to passive ventricular filling. The ventricle relaxes and blood passively flows in from the atria to the ventricle (approximately 75% of filling is passive).

S_4 corresponds to active ventricular filling as the atria contract. Blood is forced into the ventricle.

S_3 and S_4 when audible in small animals, are always indicative of an abnormality. The presence of an audible diastolic sound implies that the ventricle is not filling normally i.e. there is poor relaxation. This is described as a gallop rhythm and sounds like Du-Lub-Dup. One can only characterise if it is an S_3 or S_4 gallop by recording a phonocardiogram. (Differentiating whether gallop is due to S_3 or S_4 is not likely to be of significance anyway.)

Splitting of S_1 and/or S_2 indicates asynchronous closure of the AV or outflow valves. A split S_2 can occur if there is pulmonary hypertension for example. The pulmonic valve closes after the aortic valve leading to two audible sounds rather than one thus "splitting" the heart sound.

Heart murmurs

A murmur indicates the presence of turbulent flow within an area of the heart due to disturbance to the normal laminar flow of blood within the heart and surrounding vessels. This is likely to occur when there is an increased velocity of blood flow, an increased volume of blood flow, a reduction in the blood viscosity or when there is regurgitation of blood across an insufficient (leaking) valve.

When are you likely to hear murmurs?

The vast majority of murmurs in small animals are systolic. This is because systole is the most active period of the cardiac cycle when ejection occurs and ventricular pressures are highest. Aortic regurgitation gives rise to a diastolic murmur.

Systole	Diastole
AV valves closed M + T insufficiency	AV valves open M + T stenosis (low pressure)
Outflow valves open A + P stenosis	Outflow valves closed A + P insufficiency
Aortic pressure > PA pressure flow through PDA	Aortic pressure > PA pressure flow through PDA
LV pressure > RV pressure flow through VSD	LV pressure = RV pressure No flow through VSD

Abbreviations - M: Mitral, T: Tricuspid, A: Aortic, P: Pulmonic, PA: Pulmonary artery, PDA: Patent ductus arteriosus, LV: left ventricle, RV: Right ventricle, VSD: Ventricular septal defect.

Murmurs described according to
- Timing/Duration
- Intensity
- Location (point of maximal intensity)

Timing

The most important distinction as far as timing is concerned is systolic versus diastolic. Between S_1 and S_2 is systolic. Between S_2 and the following S_1 is diastolic. Some people try to define murmurs as early, mid or late systolic or late diastolic. This can cause a lot of confusion and it is not as important as differentiating systole from diastole. It is especially difficult to accurately time murmurs at higher heart rates.

It is possible to have a murmur throughout systole and diastole. These murmurs are described as continuous. The classic example of this is the PDA (although other potential causes exist). If the heart rate is fast and it is difficult to distinguish systole from diastole then palpate the femoral pulse. Pulse pressure comes up (rises) soon after the onset of ventricular systole.

Location of murmurs

The "heart base" area corresponds to the area of the outflow valves and is a fairly cranial position. In order to access this area you will have to move your stethoscope under the triceps muscle mass which may necessitate moving the animal's leg forward on that side. The heart

apex is the position on the left where the mitral valve will be most audible. On the right side the tricuspid valve is most clearly audible. VSD and PDA murmurs need not be at a valve position. PDA dorsal to the left heart base. VSD "diagonal" from the left heart apex to the right sternal border.

Intensity of murmur

Murmurs are graded out of six in terms of intensity

I/VI	Audible after a long time listening in perfect conditions. Quiet room, amenable patient, good stethoscope.
II/VI	Clearly audible as soon as the stethoscope is placed over the point of maximal intensity.
III/VI	Clearly audible and as loud as the normal heart sounds.
IV/VI	Audible louder than heart sounds but no thrill palpable, likely to radiate widely over the thorax.
V/VI	Thrill palpable over the point of maximal intensity at the skin surface
VI/VI	Audible with the stethoscope lifted off the chest.

Murmurs can also be described according to their radiation, pitch and shape. These characteristics are less important than the three already referred to.

Radiation

When a murmur is audible at a site other than the point of maximal intensity it is said to radiate. It will radiate more loudly in certain directions due to the direction of the turbulent jet giving rise to the murmur, or along structures adjacent to the site of origin of the murmur. Aortic stenosis murmurs tend to radiate up the carotid arteries and are sometimes audible over the head. Mitral murmurs radiate dorsally within the thorax.

Pitch

High pitch murmurs may be more likely to be ejection murmurs and low pitch murmurs may suggest regurgitant flow. Fairly loose terms and probably more useful when trying to establish if a murmur has changed or if more than one murmur is present in the same animal.

Shape

Description from phonocardiogram diamond shape, crescendo, crescendo - decrescendo etc. Decrescendo murmurs get less intense over time the classic example of this being aortic regurgitation.

What else are we listening for?

Intensity of heart sounds. May be muffled with pleural or pericardial fluid. May be marked if there is gross cardiomegaly.

Listen for heart rhythm. Compare to pulse rate and rhythm. Always take the pulse at the same time as listening to the heart.

Finally listen to the lungs. Respiratory disease is often mistaken for heart disease due to similarity of the signs – breathlessness, coughing etc. A lot of older small breed dogs with lung disease will have incidental heart murmurs. Listen for wheezes and crackles over lung fields. Crackles may be evident if there is pulmonary oedema present.

A murmur has been discovered – what now?

Following the discovery of a murmur there are a number of important questions that should be considered which will help narrow down the likely nature of the underlying disease process.

How old is the dog?

Animals younger than 3-4 years of age are more likely to have congenital disease rather than acquired disease.

What breed and size is the dog?

Many congenital diseases have strong breed associations. With acquired heart disease, degenerative mitral valve disease is more likely to occur in small breed dogs whereas larger dogs tend to develop dilated cardiomyopathy.

How audible is the murmur?

Louder murmurs often, but not always, signify the presence of more serious disease.

Is the animal showing any clinical signs consistent with heart failure?

Signs such as increased respiratory rate and effort, exercise intolerance, lethargy and collapse episodes may occur as a consequence of heart disease leading to inadequate function of the cardiovascular system. If such signs are present it is more likely that an animal's murmur indicates the presence of significant disease.

Are there other signs present suggestive of compromised function of the cardiovascular system these could include pallor, cyanosis, venous congestion, ascites, cold extremities, an audible arrhythmia etc.

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Further investigation

Many factors may determine whether or not a dog will undergo further investigation.

Factors to consider include the following.

Factor	More likely to investigate.....	Less likely to investigate if....
Age of patient.	Younger patient More likely to have definitive treatment and lengthy period of benefit	Older patient
Audibility of murmur	Loud murmur ≥ III/VI	Quiet murmur
Timing of murmur	Diastolic, continuous	Systolic
Presence or absence of clinical signs.	Clinical signs are present	Clinical signs are absent
Intended use of dog	Dog used for breeding or as athlete	Sedentary neutered dog
Level of owner anxiety	Owner anxious to know cause of murmur	Owner happy to wait and see
Anticipated cardiovascular stress	Patient likely to be anaesthetised or receive fluids in near future	Patient not expected to undergo any CV stress

What is the best method of investigation?

Usually the initial preferred diagnostic test for investigation of the presence of a heart murmur is echocardiography including Doppler echocardiography. This is often sufficient for a diagnosis to be reached although other tests may be required to determine the severity and impact of the patient's disease.

WSV18-0013

FELINE FOCUS (SIMULTANEOUS TRANSLATION INTO MANDARIN CHINESE)

FELINE URINARY TRACT HEALTH: METABOLISM AND STRESS

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Lower urinary tract disorders are common in cats. In previous decades, the focus of study has been on causes and management of crystalluria. As struvite crystalluria was successfully addressed through nutritional changes resulting in urine acidification, the frequency of calcium oxalate crystalluria increased. This encouraged emphasis on urine relative supersaturation (RSS), concentration and a pH neutrality. Nevertheless, cats still present with characteristic lower urinary tract signs (LUTS), namely dysuria, pollakiuria, hematuria, stranguria and periuria. The cause of approximately 65% of non-obstructive lower urinary tract disease is of unknown despite appropriate diagnostic testing. (Possible causes of LUTS are shown in Figure 1. A diagnostic approach to cats with lower urinary tract signs is shown in Figure 2.) These patients are described as having an idiopathic cystitis (IC). It is likely that this syndrome is multifactorial even within the same cat. The course of human interstitial/idiopathic, including interstitial cystitis, is known to be impacted by stress. There is evidence that there are immunological and neuroendocrine components in our feline IC patients as well.
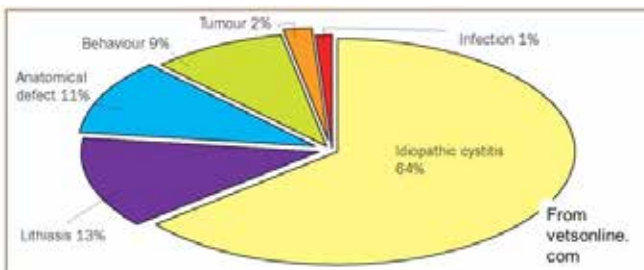
Studying feline idiopathic cystitis (FIC) is extremely challenging not only because of its multifactorial nature, but also because clinical signs are self-limiting. In approximately 91% of cats, evidence of discomfort resolves within 7 days without treatment. Subsequent episodes are also acute in nature and occur once or twice a year. As cats get older, the frequency and severity of the flare-up decreases. A small number of cats experience chronic persistent disease lasting weeks to months.

Inflammation associated with each incident may result in functional or mechanical obstruction. The first may be caused by urethra swelling, spasm, or reflex dyssynergia, while accumulations of inflammatory debris or the formation of matrix plugs can cause mechanical obstruction. Urachal diverticulae are a possible sequelae to FIC.

What causes the inflammation in non-obstructed LUTD? Many studies have attempted to answer this question yet results have been disappointing. Infectious agents, dietary causes (mineral composition, RSS and urine pH), neurogenic, anatomic, traumatic, neoplastic and iatrogenic etiologies are all implicated in some individuals, but the largest category remains idiopathic in origin.

Buffington and colleagues have investigated the problem from another angle asking whether a susceptible individual might develop FIC if they are in a provocative environment. Indeed, similar to the human model of IC, he found that affected cats have structurally altered adrenals, more reactive somatosensory spinal tracts and a larger pontine locus coeruleus (LC, the most important source of norepinephrine in the CNS) This suggests that patients with IC have increased sympathetic nervous system (SNS) activity even during periods without clinical signs. He has reviewed published epidemiologic data regarding the role of environment and its physiologic effects on risk for disease, especially in susceptible individuals. External influences include excessive body condition, decreased activity, being restricted to eliminate in a litter box, being strictly indoors, relocation of home, living with other cats and weather changes. Stressors (internal/perceived influences) that affect different individuals to a greater or lesser degree include an impoverished environment, lack of stimulation, noise, restraint, and lack of control over his/her environment (including meals). The stress response invokes changes in immune, neurologic and vascular status, all of which can cooperatively result in inflammation. With sufficiently severe stress, sensory input and inflammatory mediators stimulate the hypothalamic-pituitary-adrenal axis (HPAA) and the aforementioned pontine LC – norepinephrine system. With chronic stimulation, over time normal control is lost and affected individuals overreact physiologically to threatening or disruptive situations.
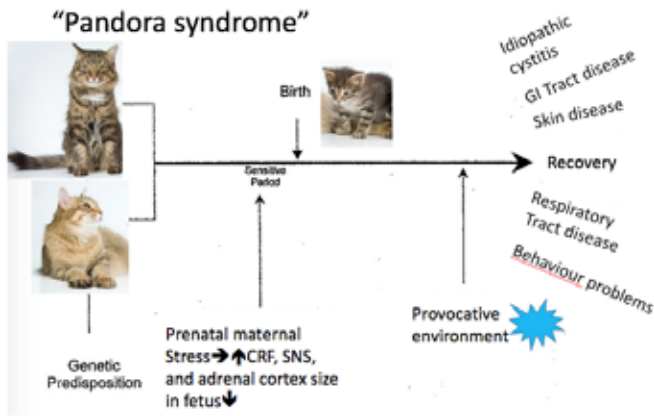
"Pandora syndrome"

Modified from Buffington CAT, Westropp JL, Chew DJ. From FUS to Pandora syndrome: Where are we, how did we get here, and where to now?. J Feline Med Surg. 2014;16(5):385-94.

Buffington and co-workers also identified that cats, as humans, with IC frequently have co-morbidities and has called this the Pandora Syndrome. He suggests that the bladder, rather than being the perpetrator of the LUTS, may be a victim of the systemic process associated with the sensitized central stress response system. Comorbid disorders include behavioural, endocrine, dermatological, respiratory, cardiovascular, and gastrointestinal problems. FIC does not necessarily precede the other conditions. In humans, the effects of chronic in utero stress on the health of the offspring are well documented. It may well be that genetic and similar epigenetic events contribute to the susceptibility of an individual making them at risk should they be exposed to provocative events.

MANAGEMENT OF CATS WITH FIC

Evaluating the efficacy of therapies for FIC is very difficult because of the waxing-waning nature of the disorder. Stress reduction appears to be a cornerstone for managing cats afflicted with FIC. Addressing environmental needs is essential (not optional) for optimum wellbeing of the cat. Environmental needs include those relating not only to the cat's physical surroundings (indoors or outdoors; in the home environment or at the veterinary practice) but also those affecting social interaction, including responses to human contact. Cats need to have multiple and separate locations for each resource (food, water, clean litter, toys, stable scratching surfaces, perches and resting areas). The overview of a therapeutic and management approach to a cat with LUTS is shown in Figure 3.

It is essential that cats are able to express their natural behaviours. Cats use olfactory and chemical information to evaluate their surroundings and maximize their sense of security, comfort and feel in control of their surroundings/environment. Depositing pheromones through cheek and paw pad marking as well as urine

is key for a cat's sense of control. In some situations, when a cat is marking with urine, it may be possible to get the cat to make a less offensive mark (from a human perspective). Cheek marking wall corners may be encouraged by using Feliway and not washing the cat's natural oils off walls and furniture. Likewise, providing secure, stable scratching surface placed in the location being urine marked, may result in the cat scratching and marking in that manner rather than spraying. The **AAFP and ISFM Feline Environmental Needs Guidelines** is an excellent resource freely available from: (jfm.sagepub.com/content/15/3/219.full.pdf+html).

Pheromone Use

FeliwayTM is a synthetic analog of a feline facial pheromone that is thought to increase emotional stability. Its use in the reduction of inappropriate urination needs to be studied further. Studies done to date have shown a reduction in urine marking of less than three months duration of over 96%. In cats who had been marking for four months or longer, there was a reduction of marking in 91% of cats after 35 days of environmental treatment. A third study showed that while there was a significant reduction in all households in which FeliwayTM was applied, 2/3 of the households still experienced some marking.

The product is sprayed directly on places soiled by the cat and also any prominent vertical locations in the environment. A daily application is necessary until the cat is noted to exhibit facial rubbing on the site. If the cat does not exhibit facial rubbing, then daily application to the environment should be continued for one month. Plug-in diffusers provide a constant, slow release of pheromone covering an area of 500 to 700 square feet (50-70 m2), but must not be covered, placed behind a door or under furniture.

Diet and Drugs

Feeding a diet that produces dilute urine with a neutral pH seems to help cats have fewer recurrences of FIC or any type of lower urinary tract disease. Canned food helps to ensure that the urine is dilute, making it less concentrated (hence, less irritating) and reducing the chance that crystals can form. Having plenty of fresh water available in multiple places in a form the individual cat likes will encourage drinking. Some cats prefer drinking from a recirculating water fountain, others prefer wide bowls. Feeding a diet that has omega-3 fatty acids along with anti-oxidants may also provide beneficial anti-inflammatory effects. Finally, being consistent both in time of feeding as well as diet being fed is very important in reducing stress.

Many drugs have been used to try to reduce the reoccurrence of FIC. Amitriptyline may be helpful in some cats if it is given on an ongoing basis. It is an antidepressant and agent that stabilizes mast cells

which may degranulate in some individuals with FIC. Glucosaminoglycans have also been studied and have variable, but generally poor, results. Best results appear to occur with diet, environmental and stress management rather than drug therapy.

SUMMARY

Lower urinary tract disorders are common in cats. Once appropriate diagnostics have ruled out direct causes, for most cases of non-obstructive LUTD, a more global approach needs to be taken, looking at and addressing the role of the cat's external and internal environments.
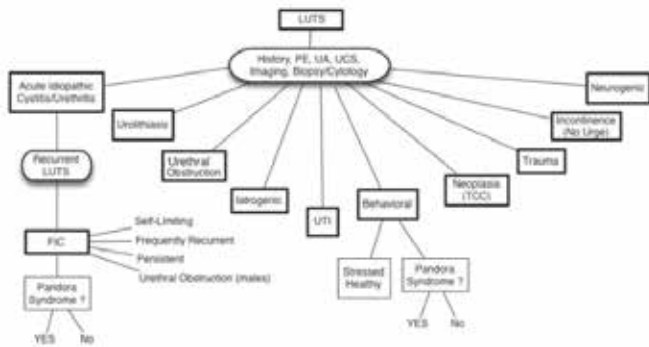
Figure 1: *Possible causes of lower urinary tract signs in cats with or without co-morbid conditions (Pandora Syndrome) (from Chew D, Buffington CAT, FLUTH Symposium 2014)*
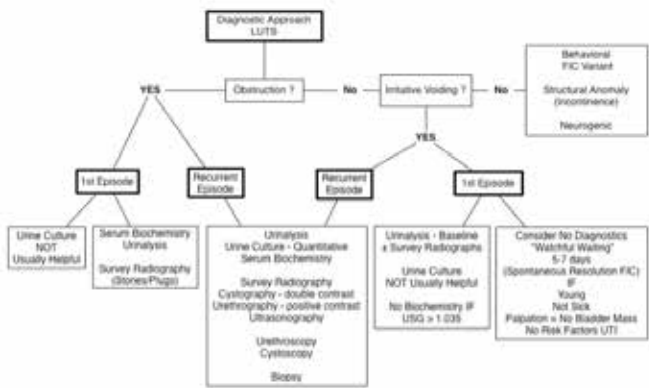
Figure 2: *Diagnostic approach to cats with lower urinary tract signs (from Chew D, Buffington CAT, FLUTH Symposium 2014*
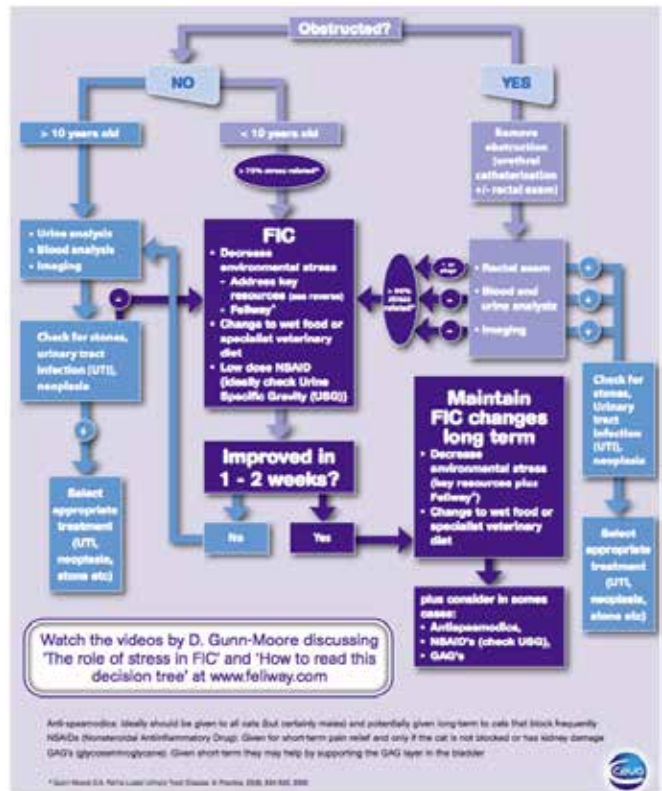
Figure 3: *Algorithm showing a therapeutic and management approach to a cat with lower urinary tract disease*

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CANINE CHRONIC HEPATITIS: CAUSES AND TREATMENT

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What is canine chronic hepatitis?

- 'Chronic hepatitis' = hepatic mononuclear or mixed inflammatory infiltrate with piecemeal necrosis and varing degrees of fibrosis. Fibrosis tends to progress to cirrhosis, but not inevitably.
- Cirrhosis describes progressive bridging fibrosis, inflammation and nodular regeneration which has classically been considered end stage and irreversible.

What isn't chronic hepatitis?

In addition, it is important to rule out secondary hepatopathies which are a much commoner cause of increased liver enzymes than primary liver disease. This differentiation is usually achieved through careful interpretation of clinical examination, history, clinicopathological tests and diagnostic imaging. Secondary hepatopathies are usually not clinically significant in their own right and resolve when the underlying cause is treated.

Causes of canine chronic hepatitis

Chronic hepatitis frustratingly usually remains a non-specific diagnosis in dogs and the cause is generally unknown. The potential causes of canine chronic hepatitis are reviewed in. They include copper storage disease and autoimmune disease in some dogs particularly English Springer Spaniels.

- Recognised in wide variety of dog breeds including cross breeds.
- Consistently increased incidences in certain specific breeds: middle aged dogs of a number of breeds including English Springer Spaniels; American and English Cocker spaniels (males more than females), West Highland White terriers (no apparent sex predisposition), Dobermans (strong female predisposition) and Labrador retrievers (female bias). Skye terrier hepatitis is now believed to be a congenital ductal plate abnormality
- Suggests a genetic basis for the disease but so far studies in dogs failed to elucidate specific genetic mechanisms apart from DLA associations in English Springer Spaniels and Dobermans.
- SOME cases are associated with primary or secondary increases in copper
- SOME cases may be autoimmune, but not all. The most likely breeds to have autoimmune hepatitis on current evidence are cocker spaniels, English Springer spaniels and some Dobermans.
- Potential low grade infections such as atypical leptospira could also potentially be involved but, again, these causes are not well investigated in

dogs. Chronic bacterial infections are typically associated with more of a granulomatous inflammatory response (- neutrophils and macrophages) and eubacterial fluorescent-in-situ hybridisation should be considered in these cases.

Potential Genetic reason for susceptibility to chronic hepatitis in dogs
Susceptibility to infectious causes of CH and/or to chronicity of infection rather than recovery
Susceptibility to autoimmune disease
Mutation of gene coding for protein involved in metal transport/storage/excretion *
Gene mutations resulting in hepatic accumulation of glycoprotein protease inhibitor
Increased susceptibility to chronic hepatic damage with toxic causes

Treatment of canine chronic hepatitis

The aims of treatment of any dog with chronic hepatitis are:

- To treat the underlying cause, if this can be identified
- To try to slow progression of the disease even if the cause if not identified
- To support liver function as long as possible and support the dog in positive calorie and nitrogen balance
- To treat the complications of liver disease which affect quality and length of life

The aim in all cases is to try to prevent the progression of disease to the end stage ie cirrhosis. Cirrhosis is often accompanied by the development of portal hypertension, where increased resistance to flow through the hepatic vasculature raises portal pressure resulting in splanchnic congestion, development of ascites and often acquired portosystemic shunts and hepatic encephalopathy (HE). It is, however, very important to remember that fibrosis of the liver is not inevitably progressive in one direction and that even early cirrhosis can be reversible if the underlying cause is removed, as has been clearly demonstrated in humans with alcoholic and viral hepatitis. The aim in treating canine CH should therefore be to identify and treat the cause wherever possible.

The clinician should use all the information available to them to optimise treatment for an individual case. On the clinical examination, blood results and imaging, the clinician needs to assess the degree of loss of liver function and identify signs of biliary stasis, portal hypertension, acquired portosystemic shunts, ascites, gastrointestinal oedema or ulceration or protein-calorie malnutrition. On the liver biopsy if available, the clinician should assess the amount and distribution of inflammation and the types of inflammatory cells involved, the degree and distribution of fibrosis and the presence of any obvious cause including build up of

copper. Combining all this information allows optimal treatment of the patient. It is not possible – and indeed is potentially dangerous – to give specific treatments for CH (such as copper chelators and steroid or other immunosuppressive therapy) without biopsy confirmation of disease.

Treating the cause of the disease

If a significant amount of copper is found in the biopsy, in proportion to the severity of the disease, copper storage disease should be strongly suspected and treated with chelation and dietary therapy. If copper storage disease is ruled out, the type and distribution of inflammation present may suggest a cause for the disease. There are more details in the liver biopsy lecture tomorrow.

Steroids should never be used without a liver biopsy because they are not indicated in non-inflammatory fibrosis and cirrhosis: if used in these cases in the presence of portal hypertension they have no clear benefit and conversely they increase the risk of serious consequences including increased water retention and gastrointestinal ulceration. None-the-less, corticosteroid therapy should offer the best chance of survival in dogs with true autoimmune CH so should not be withheld if the clinician and pathologist believe on the basis of liver biopsies that this is the most likely cause of disease in a particular dog. There are also some promising results from a study in the USA using cyclosporine for dogs with suspected immune-mediated chronic hepatitis.

TREATING CLINICAL SIGNS AND SUPPORTIVE THERAPY

Treating the clinical signs of disease non-specifically is a very worthwhile aim in CH because it improves the quality of life of the patient. Ultimately, most dogs with CH die because the owners request euthanasia due to poor quality of life, so improving the quality of life should also improve life expectancy in these patients. The factors to consider and treat are:

- Ascites: if present, this should be treated with spironolactone as the primary diuretic and addition of loop diuretics as necessary
- Vomiting, diarrhoea and evidence of GI ulceration: these are common in animals with portal hypertension and should be treated predominantly by careful little and often feeding to provide nutrition for gut wall healing and avoidance of potentially ulcerogenic drugs such as steroids. H2 antagonists such as ranitidine or proton pump inhibitors such as omeprazole could be used, together with sucralfate, although evidence for their efficacy in ulceration due to portal hypertension is lacking.
- Jaundice: when pre hepatic causes and post hepatic obstruction have been ruled out, this should be treated with ursodeoxycholic acid and anti-oxidants. Ursodeoxycholic acid has a large number of potential benefits in animals with CH including choleresis, displacement of toxic bile acids and anti-oxidant properties. There are no contraindications to its

use but, like all other therapies in canine CH, the evidence for its efficacy is very sparse. However, it makes logical sense to use it. It also makes sense to use anti-oxidants in this circumstance since refluxed bile damages mitochrondrial membranes and is a strong oxidant toxin. A combination of S-adenosyl methionine, silybin and vitamin E is advised. The clinician should try to choose neutraceuticals with proven bioavailability in dogs.

- Hepatic encephalopathy (HE): this is not as prominent or easily recognised in dogs with CH as it is in young dogs with congenital portosystemic shunt. None-the-less, it can be an important cause of confusion and unusual behaviour in these animals due to the development of acquired portosystemic shunts secondary to portal hypertension. It should be treated carefully: marked protein restriction is not indicated in these dogs, as they are likely to be suffering from protein-calorie malnutrition already. HE can be addressed by treating any underlying inflammatory trigger and also any precipitating gastrointestinal bleeding and giving a highly digestible, high quality diet little and often. Antibiotic and lactulose therapy may also be considered. Dietary protein restriction is very rarely necessary in these cases
- Treatment of protein-calorie malnutrition: many dogs with CH present in negative nitrogen balance. They are often thin with partial anorexia and vomiting and diarrhoea which contribute to nutrient malabsorption. It is therefore very important to prioritise nutrition in the treatment of these patients. Ideally, they should be fed a highly digestible, high quality diet little and often and this diet should not be protein-restricted.

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ONCOLOGY

UPDATE ON MALIGNANT LYMPHOMA IN DOGS

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UPDATE ON LYMPHOMA IN DOGS
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Initial diagnosis

- Fine needle aspiration (FNA) and cytology: From peripheral lymph node, organ, blood/ bone marrow or other site. Pros: quick, non-invasive. Immunocytochemistry may be done for immunophenotyping (T versus B cell). Definitive cytological diagnosis of lymphoma is reliable, however in situations where this is not possible additional diagnostics are required. Such situations include small cell/low grade lymphoma, reactive lymph nodes, splenic location and non-diagnostic samples due to cell rupture or blood contamination. In the latter situation, adjustments in technique such as non-aspiration, smaller needle gauge, and using gentle techniques in making slides can improve diagnostic quality

- Biopsy and histopathology: Histopathology with immunohistochemistry (IHC) is gold standard for canine lymphoma diagnosis and subtyping. Excision of a whole lymph node is recommended, but punch or needle core biopsies may also be diagnostic. Histopathology is indicated when cytology is inconclusive, FNA is low yield (e.g. minimally thickened intestinal wall), or additional information about subtype is desirable. Pros: generally relatively quick turnaround time, (IHC may take longer). Can give significantly more information if performed by a pathologist experienced in lymphoma subtyping. Cons: More invasive and expensive than FNA and cytology.

- PCR for antigen receptor rearrangement (PARR): Test for clonality, aim is to distinguish lymphoma from reactive lymphocyte population. Used where a definitive diagnosis of lymphoma cannot be made on cytology or histology alone. Pros: Can be performed on cytology or histologic preparations, usually without the need for collection of additional samples. High specificity. Cons: Can be long turnaround time, often expensive, variable sensitivity (approximately 70-90%, can be affected by the tissue sampled and primers used). Best used for diagnosis rather than immunophenotyping of known lymphoma. Most commonly applied in suspected small cell GI lymphoma, in combination with histopathology and IHC.

- Flow cytometry: Assess cell surface markers using specific antibody stains. Pros: Superior to PARR for immunophenotyping, may be suggestive for diagnosis (cannot be used to definitively identify a clonal population), can diagnose some specific subtypes (e.g. loss of CD45 in T-zone lymphoma), expression of additional proteins can be assessed (e.g. Ki67 or MHCII) which may have prognostic implications. Relatively non-invasive (fine needle aspiration), quick turnaround.

Cons: handling and shipping requirements may limit availability, as cells must be alive and in suspension.

- Newer biomarker tests (cRP, TK, haptoglobin) are available but their utility in routine clinical practice has not been demonstrated

Additional diagnostic tests: In my practice, I recommend additional diagnostic tests for a dog with known lymphoma if they alter prognosis, treatment options (what I would recommend or what the owner would choose to do), or there are abnormalities present that cannot be easily attributed to lymphoma.

- CBC, serum chemistry, urinalysis: Minimum database required if chemotherapy is considered. Thrombocytopenia, lymphocytosis and neoplastic lymphocytes on blood smears in dogs with multicentric large cell lymphoma are associated with bone marrow involvement (1, 2). Assess for abnormalities that may require alterations in chemotherapy protocol (e.g. liver function in dogs to receive vinca alkaloids), be directly associated with lymphoma (e.g. hypercalcemia implying likely T cell immunophenotype in large cell lymphoma) or other concerns. Proteinuria is common in dogs with cancer, including lymphoma (3, 4), and may require ongoing monitoring, further investigation or intervention.

- Staging:
  1. Involving a single lymph node, or lymphoid tissue in a single organ (excluding bone marrow)
  2. Involvement of multiple lymph nodes in a regional area
  3. Generalised lymph node involvement
  4. Liver and/or spleen involvement (+/- stage III)
  5. Blood, bone marrow and/or other systems

Each stage is subclassified into:

- Substage a: With systemic signs
- Substage b: Without systemic signs

In order to assign stage, full evaluation would involve, in addition to the minimum database, thoracic and abdominal imaging (usually chest radiographs and abdominal ultrasound) and sampling for cytology or histopathology, bone marrow evaluation and other evaluation depending on clinical presentation (e.g. MRI if neurological signs). The prognostic impact of stage beyond stage III in dogs with multicentric lymphoma has not been fully defined. Certainly, involvement of liver and spleen does not seem to have a significant impact on prognosis, and bone marrow may or may not. Therefore, if complete staging does not significantly impact prognosis or treatment options, for many oncologists and owners, it is preferable to spend money on treatment rather than additional diagnostic tests. However, if there are specific localising signs e.g. vomiting, coughing, further evaluation may be more warranted. As will be discussed later, large cell gastrointestinal (GI) lymphoma generally has a poorer prognosis and so identifying GI involvement may impact approach in cases with suggestive clinical signs.

- Immunophenotype: In large cell multicentric lymphoma, immunophenotype is one of the strongest

prognostic factors and therefore immunophenotyping is always recommended. IHC is considered the gold standard, however collection of biopsies solely for purposes of IHC in a dog with previously diagnosed lymphoma may be difficult to justify from the standpoint of invasiveness and cost, when less invasive options exist. In my opinion, flow cytometry would be considered the next preferred option, followed by immunocytochemistry and then PARR because of the limitations already discussed in turnaround time and sensitivity.

Canine lymphoma classification:

- Subtype: The WHO classification scheme is based on tissue architecture, cell size, mitotic rate, cellular features and immunophenotype. There are approximately 40 different subtypes. Pathologist expertise is important when classifying lymphoma and there is good, but not perfect agreement between pathologists (approximately 90% when only the 6 most common types are considered). From a practical standpoint, the important considerations are: identifying B versus T cell in large cell lymphoma, identifying T zone lymphoma separately from other T cell lymphomas, and identifying low grade B cell lymphomas, especially splenic marginal zone lymphoma. As well as considering the WHO subtype, clinical progression must be considered. T zone lymphoma, marginal zone lymphoma, mantle cell lymphoma and follicular lymphoma are generally considered indolent in behaviour. They often present as peripheral lymphadenomegaly (+/- lymphocytosis) which is slowly progressive. Although they are generally poorly responsive to standard maximum tolerated dose chemotherapy, prolonged survivals are common. Treatment is often not initiated at the time of diagnosis unless there are clinical signs or evidence of more rapid or advanced clinical progression. When treated, the most common initial protocol is chlorambucil and prednisolone. Within these 'indolent' subtypes, those of B cell origin generally do not have as good a prognosis as T zone lymphoma. There are some cases where an indolent lymphoma can undergo transformation to acquire more malignant behaviour and rapid progression. These are generally associated with a poor outcome, and reinforce the need to consider the clinical behaviour as well as the subtype.
- Anatomic location is also part of WHO classification, but some specific sites bear separate consideration
- Hepatosplenic and primary hepatic - generally aggressive and associated with lower likelihood of response to chemotherapy
- Splenic - often marginal zone and can do quite well with splenectomy alone, however case selection is important - i.e. confined to spleen, not large B cell lymphoma
- Rectal - mostly B cell and tend to have a good prognosis with chemotherapy based on the small numbers of dogs published in the literature (5)
- Gastrointestinal - High grade/large cell GI lymphoma in dogs is generally associated with a poor outcome. Recently, low-grade/small cell GI lymphoma has been recognised and seems to behave like the more common low-grade/small cell GI lymphoma in cats. Outcomes tend to be much better and recommended treatment is different (oral chlorambucil and prednisolone versus multi-agent CHOP or other chemotherapy protocol for high grade/large cell lymphoma)
- Epitheliotropic T cell lymphoma
    - Cutaneous: Most are diffuse and respond well initially to chemotherapy but progress within a few months. Alternative treatments such as isotretinoin or safflower oil may help in some cases. Solitary lesions may be associated with a better prognosis (Chan)
    - Oral/mucocutaneous: If lesions are localised to the oral cavity then outcomes with chemotherapy or radiation therapy can be very good (> 1-2 years), and again solitary lesions may be associated with better prognosis (6, 7)

Summary:

Canine lymphoma encompasses a group of diseases with varying clinical presentations, need for diagnostic tests, treatment options and prognosis. In my clinical practice, to treat a dog with lymphoma, the things I need to know are:

- Subtype as much as is reasonable i.e. large cell/small cell, immunophenotype and clinical behaviour at a minimum. Flow cytometry is my preferred option for immunophenotyping following a cytological diagnosis as it also enables diagnosis of T zone lymphoma. If a diagnosis has been made on histopathology, IHC is preferred for immunophenotyping
- Minimum database of CBC/serum chemistry/urinalysis
- Substage - assigned based on systemic signs
- In a dog with a confirmed diagnosis of multicentric lymphoma, additional diagnostic tests are not uniformly recommended in order to assign the case to stages I-V, unless it is felt that prognosis or treatment options would be changed by the result of these tests.

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GENETICS

WHAT PRACTITIONERS SHOULD KNOW ABOUT THE GENETICS OF BRACHYCEPHALY

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WHAT PRACTITIONERS SHOULD KNOW ABOUT THE GENETICS OF BRACHYCEPHALY

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Part of the welfare considerations practitioners deal with in our patients is the expression of extreme phenotypes or morphology. The "more is better" rule has led to extremes in size (large and small), hair, skin folds, angulation, and the most frequently seen is extreme brachycephaly.

Public preference for dogs and cats with pushed-in noses and bulging eyes has created a breeding environment that has crossed a tipping point in predisposing to disease. Brachycephalic obstructive airway syndrome (BOAS) is a disorder of breathing difficulty in short-snouted and "bully" breeds (especially the popular English Bulldogs, French Bulldogs and Pugs).[1] In Persian and Himalayan cats, BOAS causes chronic sinusitis.[2] These dog and cat breeds can also have issues with skin fold dermatitis, corneal ulceration, globe proptosis, dental malocclusion, and dystocia.[1]

Of the anatomical components of BOAS, stenotic nares and hypoplastic trachea are inherited. The length of the soft palate and therefore soft palate/epiglottis overlap is also inherited. Soft palate thickness (which exacerbates BOAS) is caused by hyperplasia secondary to chronic turbulence, as are everted laryngeal saccules. Laryngeal collapse and hiatal hernia (with regurgitation) are secondary to the effects of negative inspiratory pressure from restricted airflow.

THE INCIDENCE OF BOAS IS RAPIDLY INCREASING

BOAS is becoming a greater clinical issue due to the rapidly increasing popularity of the susceptible breeds. Registrations of French Bulldogs have increased 3,000% over the past 10 years.[1] As the clinical morbidity of BOAS does not usually manifest until middle age, these rapidly expanding young breed populations will continue to increase their presentation with symptomatic BOAS. Another welfare issue involves the increase of brachycephalic dogs relinquished to shelters and rescue organizations, and the difficulty rehoming dogs with breathing difficulties.

Compounding the matter is the fact that up to 60% of owners do not see their dogs (or cats) as being impaired by BOAS. The "normalization" of brachycephalic stridor also extends to veterinarians: "That's just how Pugs breath." However, BOAS dogs are regularly seen – especially in warm weather - with dyspnea, exercise intolerance, heat intolerance, abnormal and increased respiratory noise, sleep disorders, cyanosis, syncope and death.

Individual dogs in breeds with extreme brachycephaly die younger (median longevity 8.6 years) than dogs in moderate and non-brachycephalic breeds (median 12.7 years). A higher proportion of deaths in extreme brachycephalic breeds are due to upper respiratory disease (16.7%).[3]

PHENOTYPIC SCREENING AGAINST BOAS

The genetics of brachycephaly are present in all members of the involved breeds. Therefore the question becomes, "What is genetically different between dogs in these breeds exhibiting BOAS and those that do not? Part of this question also involves identifying diagnostic criteria that separates BOAS affected and non-affected dogs.

A group at the Royal Veterinary College in Cambridge, UK led by Dr. David Sargan has developed a closed chamber for Whole-Body Barometric Plethysmography (WBBP). A dog's airflow is measured at rest and after controlled activity. Physiological measurements of airflow volume and pattern consistently separate dogs and breeds with BOAS versus those without BOAS. Based on WBBP, approximately 50% of the study dogs in the three extreme brachycephalic breeds were BOAS affected.[1] While WBBP is not practical for widespread screening, its results have allowed studies of other measurements that differentiate between BOAS+ and BOAS- dogs. The data show that roughly 50% of BOAS variation is genetic and 50% is environmental; including exercise, feeding, temperature and humidity. There are several recorded measurements that are significant in different breeds and sexes. However stenotic airways and body condition score (obesity) are the most significant factors correlated to the clinical presentation of BOAS across all breeds.[4] Stenotic nares are primarily caused by the enlargement of the alar folds that impinge into the nasal opening. Veterinary surgeons demonstrate that surgically correcting stenotic nares relieves most dogs of the clinical signs of BOAS. Surgery removes the lateral alar cartilage. The rule of thumb is to open the nares to a cumulative 1/3 the width of the nose. According to WBBP almost all dogs improve their BOAS index after nares surgery and 40% become BOAS- non-affected.[1]

Dr. Rowena Packer at the Royal Veterinary College developed a measuring system for stenotic nares. She states that each breed has very specific nares conformation, and breed-specific values would need to be established for setting breeding goals. Studies show

75.4% of French Bulldogs have moderately to severely stenotic nostrils, while prevalence amongst Pugs (65.3%) and English Bulldogs (44.2%) is lower.[4]

The OFA in the US (ofa.org) has instituted a pilot database for computer-assisted measurement of nares openings from digital photographs in English Bulldog and other bully breeds. They also have an existing database for evaluating hypoplastic trachea from a lateral radiograph.

The Swedish and Finnish Kennel Clubs have developed standardized working (walking and jogging) tests to document labored breathing as a screening test for BOAS. These walking tests have also been evaluated with WBBP. Studies show English Bulldogs with more severe BOAS walked a shorter distance, more slowly and their recovery from exercise took longer than those with only mild signs of BOAS. Increases in body temperature during exercise were significantly higher in English Bulldogs than in controls.[5]

Continuing research on the phenotypic screening of BOAS is correlating WBBP, standardized walking tests, and nares and body measurements to more accurately define the BOAS phenotype. Some of these test results could in the future be combined into an estimated breeding value (EBV) to compare between prospective breeding dogs.

GENES RELATED TO BOAS

There are several studies into the genes causing BOAS. Groups of BOAS+ and BOAS- dogs can be compared through DNA analysis. Results show that many genes are involved in BOAS making it a polygenic disorder. Therefore the task is to identify if there are single genes that have a major effect on BOAS, or if a panel of genes provides a major difference in the liability to develop clinical BOAS. Such a panel would provide a genomic breeding value (GBV) to compare prospective breeding dogs, and possibly differentiate those with a genetic predisposition to become mildly, moderately, or severely affected with BOAS.

Several genetic studies have been conducted to identify genes associated with skull morphology, brachycephaly and BOAS. Identified genes include IGF1, THSB2, SMOC2, FGF4 and BMP3. However many of these genes are fixed (non-variable) in brachycephalic and BOAS liable breeds. Therefore they do not cause a genetic difference between BOAS+ and BOAS- dogs. Studies from the Cambridge group into a regulatory gene affecting SMOC2 expression show that it affects the facial skeleton in a dose-dependent manner and accounts for 36% of facial length differences.[6] This group has identified a panel of 11-13 genetic markers that account for 35% of the phenotypic variation in Pugs, 47% in French Bulldogs, and 51% in English Bulldogs. Based on this panel they have been able to predict the most severely affected BOAS dogs in each breed. They also find that those less likely to develop BOAS through GBVs have longer muzzles and wider nares openings.

CONCLUSIONS

While several breeds have a high incidence of BOAS, there is still considerable within-breed variation to enable breeders to breed away from genetically susceptable dogs. Current phenotypic screening tests to select against BOAS liability include standardized walking tests (usually administered by breed or kennel clubs), nares and trachea measurements, and selecting for a breed-appropriate but longer muzzle. Based on research models, genetic testing panels against BOAS liability should be available in the near future.

Part of changing the culture that has caused the rapid popularity of extreme brachycephalic breeds is to remove the social media fixation on them. In some studies, more than half of all advertising that includes a dog has a Pug, English Bulldog, or French Bulldog. The British Veterinary Association has called for a moratorium on advertisements containing extreme brachycephalic breeds.

Dog show judge's education is important to select against the breed extremes of short muzzles and tight nares and to reward moderation of phenotypic morphology. Veterinarians should educate breeders and owners on the morbidity of BOAS. Breeders should use genetic screening in breeding schemes, and prospective owners should seek health-conscious breeders who use genetic screening. Lastly, as environmental aspects influence approximately 50% of the clinical presentation of BOAS, owners can improve their dogs' health by keeping them slim and fit.

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WAVMA ORNAMENTAL FISH DISEASES

SUPPLIES AND EQUIPMENT TO PRACTICE AQUATIC VETERINARY MEDICINE

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Aquatic veterinarians can use much of the same equipment and medications used in any small animal veterinary practice. Most diagnostic tests in fish can be done using standard veterinary equipment, and surgeries performed with instruments typically used for ophthalmic work, such as iris scissors. Radiology equipment, especially digital or dental units, sonograms, endoscopes, and blood evaluating equipment used for other animals work well with fish patients, too. Many small animal drugs are also used in fish medicine, with only a few special medications needed to supplement other veterinary drugs.

Aquatic veterinarians often make house calls to examine fish, but there are also advantages of having clients bring in the fish to the veterinary hospital. Having a client bring a sick fish into the veterinary hospital allows treatment in a quarantine tank where all the conditions can be controlled and properly maintained. Regular daily observations can be made, and appropriate diagnostic tests and treatments performed. Sick fish are isolated from the remaining fish in the aquarium or pond. Once a diagnosis is made, the fish can be properly medicated, and the sick fish can be cared for until it is well enough to return home. Disadvantages include the lack of examination of the remaining fish and the aquatic environment (aquarium or pond) itself; having the owner transport the sick fish in plastic bags, buckets or ice chests to the clinic; and in many cases the unwillingness of the owner to catch and bring in their sick fish.

By making a house call, the fish can be examined in its own environment. The filtration units can be examined and water tests performed on the spot. Apparently healthy fish can be biopsied and checked for early signs of lesions that the owner might have missed. Suggestions on improving filtration, water quality and fish husbandry are easier to make when the facilities have been visited, rather than having just the owner's descriptions.

When making a house call, portable diagnostic equipment including microscopes, slides and coverslips, bacterial culturettes, and water test kits must be brought along. A "Doctor's Bag" of common medications and antibiotics can be made up for house calls. Some very important pieces of equipment to bring when visiting koi ponds are your own quality long-handled koi nets (many pond owners have only pool skimmer nets!), a plastic container for holding the fish for examination, and chest-high waders!

Drawing blood samples from larger fish, especially koi, can be done from the caudal vein below the spine in the caudal peduncle. Use a 1-ml tuberculin syringe with a 22 or 23-gauge needle of appropriate length. A butterfly catheter can be attached to the syringe to facilitate handling of the needle separately from the syringe. Fill the hub of the needle with a drop of lithium heparin to prevent the blood from clotting. This is preferable to ammonium heparin or sodium heparin, but they can also be used for hematology testing. The ammonium and sodium heparins will affect those blood values if used in samples for serology or electrolyte testing. Ethylenediamine tetra-acetic acid (EDTA) is not recommended to be used to prevent blood clotting in fish blood samples as it may cause erythrocyte lysis.

Some normal values for koi blood parameters, derived from Advanced Koi Care by Nicholas Saint-Erne (2002, 2010) and from Hematology and Clinical Chemistry of Cyprinid Fish by Groff and Zinkl (1999), are listed in the chart below.

KOI COMPLETE BLOOD COUNT (CBC):

Normal Range:
Red Blood Cells (Erythrocytes):
Red Blood Cells (10-13 µm cell length) 1-2 Million/µl
Hematocrit (Packed Cell Volume) 24-35%
Hemoglobin 8-13 g/dl
Methemoglobin 4.8-5.6%
Mean Corpuscular Volume 202 fl
Mean Corpuscular Hemoglobin 49.1 pg/cell
Mean Corpuscular Hemoglobin Concentration 0.24 g/dl

White Blood Cells (Leukocytes):
Total White Blood Cells 5-15 Thousand/µl
Neutrophils (10-15 µm) 750-1500/µl
Neutrophils (% of Total WBC) 12-20%
Band (immature) Neutrophils 0-4%
Small (Mature) Lymphocytes (6.6 µm) 3000-12,000/µl
Small (Mature) Lymphocytes 65-85%
Large (Immature) Lymphocytes (11.8 µm) 0-3%
Monocytes (10-16 µm) 100-600/µl
Monocytes 1-4%
Eosinophils (13.8 µm) 0-150/µl
Eosinophils 0-1%
Basophils (13.8 µm) 0-150/µl
Basophils 0-1%
Thrombocytes (4.6 x 7.7 µm) 50,000/µl

Serum Chemistries (Serology):

ALT(SGPT) 20-50 IU/L

AST (SGOT) 100-300 IU/L

Total Bilirubin 0.1-0.3 mg/dl

Alkaline Phosphatase 10-20 IU/L

GGT 1-3 IU/L

Uric Acid 1-2.5 mg/dl

Blood Urea Nitrogen 5-15 mg/dl

Creatinine 0.2-0.5 mg/dl

BUN/Creatinine Ratio 10-20

Creatinine Phosphokinase 14.5 IU/ml

Glucose 30-120 mg/dl

Cholesterol 200-400 mg/dl

Triglyceride 50-500 mg/dl

Amylase 25-50 IU/L

Lipase 25-50 IU/L

Total Protein 4-10 g/dl

Albumin 2-6 g/dl

Globulin 2-4 g/dl

A/G Ratio 0.7-1.2

Osmolality 220-420 mOsm/kg

Calcium 8.5-13.5 mg/dl

Phosphorus 10-15 mg/dl

Caciuml/Phosphorus Ratio 0.6-1.3

Magnesium 3-5 mEq/L

Sodium 100-140 mEq/L

Chloride 90-120 mEq/L

Potassium 4-30 mEq/L

Na/K Ratio 3-30

Radiographs of fish can be taken successfully with standard veterinary radiology equipment. The radiographs can be taken without anesthesia by briefly restraining the fish in a sealed plastic bag with a small volume of water. The fish in the plastic bag can be placed directly onto the film plate or digital sensor, and the bag taped down if necessary to hold the fish in the correct position. Fish can also be anesthetized and then taken out of the water and positioned for radiographs. Gas bladder (swim bladder) abnormalities, spinal deformities, abdominal masses, and occasionally ingested foreign objects can be evaluated using radiology. Abdominal viscera are not easily distinguishable in a radiograph without a contrast medium. A flexible rubber catheter can be inserted orally to place barium or iodinated contrast medium into the stomach or intestines to perform contrast studies on the intestinal tract. The dosage for barium is 5 to 10 mL/kg body weight and the iodinated medium is dosed at 1 to 2 mL/kg. Be careful not to leak barium into the oral cavity and onto the gills, which could impair oxygen diffusion through the gills.

Ultrasound imaging can be performed on fish confined in a small container of water, as the water serves to couple the transducer to the fish's body, eliminating the need for ultrasound gel. Transducers of 5 to 10 MHz work well for visualization of internal organs at depths up to 13 to 20 cm into the body, with lower frequency transducers producing images at greater depths of tissue penetration. If not waterproof, the transducer can be placed inside a plastic cover (e.g., plastic bag, examination glove, or condom) for protection. The transducer can be held several centimeters away from the fish if it is in the water, and the transducer repositioned until the desired image is obtained. Motion imaging can be used for guided tissue biopsy collection, abdominocentesis (coeliocentesis), or pneumocystocentesis.

Endoscopic examination of the oral cavity, gill arches, and the pharynx can be performed by passing the endoscope into the mouth or gill operculum of an anesthetized fish. Flexible endoscopes can be passed through the esophagus into the stomach or intestines. Koi and goldfish have no stomach, but the proximal intestine is elastic and can distend to hold ingesta. Laparoscopy (coelioscopy) can be performed in larger fish to visualize internal organs or take biopsy samples. A small surgical incision can be made through an anesthetized fish's body wall to insert an endoscope. Coelomic cavity visualization is used to evaluate the liver (hepatopancreas), the gonads to determine gender or reproductive organ development, the presence of adhesions or inflammation, the gas bladder position and status (inflamed, deflated, or fluid infused), or to collect an abdominal swab for bacterial culture. If the coelomic cavity has been insufflated with air during the procedure, the air must be removed to prevent buoyancy problems immediately after the procedure. The small incision can be closed with a simple interrupted absorbable suture, or sealed with methacrylate tissue adhesive.

Commonly Used Medications in Ornamental Fish

Amikacin – 5 mg/kg IM, IP every 3 days

Aztreonam (Azactam) – 100 mg/kg IM, IP every 2-5 days

Butorphanol – 0.1 mg/kg IM for pain control post-surgically

Dexamethasone – 1-2 mg/kg IM, IP q12h

Diflubenzuron (Dimilin) – 0.06 mg/L once weekly for 3 doses

Enrofloxacin (Baytril) – 10-14 mg/kg IM, IP q48h, or PO q24h

Epinephrine (1:1000) – 0.2-0.5 ml IM, IP, IC

Fenbendazole (Panacur) – 50 mg/kg orally for 2 days, 2 mg/L water q7d x 3 doses

Formalin (37% formaldehyde) – 25 mg/L (1 ml/10 gal) in pond every other day

Florfenicol (NuFlor) – 30-50 mg/kg IM, IP, PO q24-72h

Furosemide – 2-3 mg/kg IM, IP q12-72h

Gentamicin – 3 mg/kg IM once only due to kidney toxicity

Hydrogen peroxide – 250-500 mg/L dip to prevent fungal growth on eggs

Levamisole – 10 mg/L for 12-24h bath; 50 mg/L for a 2h bath

Metronidazole – 50 mg/L bath, daily for 3-10 days, 10 mg/g of food daily for 5 days

Oxytetracycline – 50-75 mg/kg BW, added to food daily for 10 days

Praziquantel (Droncit) – 5-25 mg/kg IM, IP, PO, 10 mg/L for 6-24h bath

Sulfadimethoxine-ormetoprim (Romet, Primor) – 50 mg/kg IM or added to food

Tetracycline – 250 mg/100 g of food

Trimethoprim sulfa – 30 mg/kg IM, IP, PO q24-48h

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DIAGNOSTIC IMAGING

INTESTINAL OBSTRUCTION IN DOGS: GET THE MOST FROM ABDOMINAL RADIOGRAPHS

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Intestinal obstruction in Dogs: get the most from abdominal radiographs

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Introduction

Radiography allows an overview of the abdomen while ultrasonography does not. A short cut to ultrasound in a vomiting animal could lead to misdiagnosis of a disease that could be readily identified radiographically. Therefore, radiography and ultrasonography should be considered complimentary diagnostic tests. Barium studies still have importance in assessing animals with chronic gastrointestinal obstructions.

Take home message of this lecture:

1. A three view radiographic procedure is required in every vomiting dog or cat
   a. Right and left lateral and a VD image
   b. Left lateral places gas in the pylorus and duodenum to find foreign material

2. Wooden spoon compression
   a. Compress the bowel with a wooden spoon to separate loops of jejunum from one another so that foreign material, linear foreign bodies and corrugations can be detected without superimposition

3. Barium studies and ultrasound should only be performed after 1 and 2 are performed

Ileus

Ileus is a failure of intestinal contents to be transported and is recognized radiographically by the presence of dilated bowel segments. Survey abdominal radiographs should always be performed in vomiting animals with vomiting. Ultrasound alone in such instances does not allow a global view of the abdomen, is much more time-consuming and non-gastrointestinal causes of the dog's clinical signs as well as any secondary abnormalities may be overlooked. The radiographic appearance of ileus is dependent on its duration, location and degree of obstruction. Acute or very proximal obstructions may

show little intestinal dilation radiographically whereas chronic or more distally located ones will show more generalized dilation of the small intestines. The two major types of ileus are obstructive (mechanical) and functional. Obstructive ileus may be partial or complete.

Partial Obstructions

Dogs and cats with partial obstructions tend to have a more chronic course of intermittent vomiting and diarrhea. Common causes include foreign bodies and strictures. Fasted (>12 hours) or anorectic animals should not have small bowel segments containing granular material resembling that of food content radiographically. Granular or more opaque small bowel contents may be detected in partial obstructions. The intestines in such cases may be mildly dilated (1-1.5 times the width of the second lumbar vertebral body) proximal to the obstruction or may even be of normal diameter. Because partial obstructions may be more difficult to diagnose radiographically than complete obstructions, complementary imaging procedures such as barium studies or ultrasound are often necessary for the diagnosis. Repeat radiographic examination has great diagnostic value when abnormal intestinal content is identified. If granular content is seen in the small intestine of an animal that is vomiting or anorectic for more than 24 hours, obstruction should be considered likely. If the same finding of focal granular luminal content is identified on follow-up radiographs, even if intestinal dilation is not evident, in a vomiting animal, the chances of obstruction are high and sonography or exploratory surgery is indicated. Ultrasonography is highly recommended in older animals to screen for intestinal masses prior to exploratory laparotomy.

Complete Obstructions

More severe dilation, usually with air, is seen in complete obstructions. The location of the obstruction can be either intraluminal (foreign bodies), extraluminal (adhesions, herniation, intussusceptions), or intramural (neoplastic wall infiltrations, granulomas). Dilation (1.5-2 times the width of the body of L2) is seen proximal to the site of obstruction and the segments distal to it usually appear empty and contracted. Due to this, the jejunal segments appear to have many varied diameters, some very dilated, others empty or small. This is called a "mixed population". This is due to the continued peristaltic activity in the distal segments. The dilated segments are often referred to as "sentinel loops". Proximal duodenal or pyloric obstructions may show no radiographic abnormalities. 24 hours following a gastric outflow obstruction, the animal has vomited out the intestinal contents and the intestinal content moves to the colon. Abdominal radiographs may show no abnormalities or gastric distention. Moreover, the entire gastrointestinal tract may actually appear completely empty after some hours due to recurrent vomiting.

The most common difficulty in diagnosing complete obstructions is in trying to differentiate small from large bowel when the colon is dilated and especially gas filled. It is recommended in such instances to perform a small volume barium enema in order to identify the colon and distinguish it from the small intestinal segments that may or may not be dilated. When the abdomen appears normal radiographically in a vomiting animal, either a barium study or an ultrasound examination should be the next diagnostic procedure.

Functional Ileus

Another form of ileus that can be detected is a generalized and uniform mild intestinal dilation due to lack of peristaltic activity. This is known as adynamic, functional or paralytic ileus and results from an inhibition of bowel motility. Functional ileus results in obstruction since the intestinal contents pool in the dependent areas of the gastrointestinal tract. Radiographically the gastrointestinal tract appears mildly dilated, can have a mixed content with some gas- and some fluid-filled intestines and colon have generalized fluid or gas filling. The distribution of the intestines in the abdomen is regular. Gas is often present in the stomach. Typically, granular ingesta in the stomach and bowel is not identified. The intestines appear to have a uniform diameter. Animals with this pattern typically have clinical signs of both vomiting and diarrhea. Such an adynamic intestinal pattern can be due to the administration of pharmaceutical agents such as parasympatholytics and sedatives. Other causes are peritonitis, blunt abdominal trauma, electrolyte imbalance and enteritis of various causes.

Complicated ileus

Complicated forms of ileus include bowel perforation with peritonitis, free air in the abdominal cavity, bowel ischemia due to thromboembolism, intussusception, or volvulus at the root of the mesentery. Linear foreign bodies can also lead to a complicated form of ileus. The presence of pneumoperitoneum together with abdominal effusion on an abdominal radiograph should alert the clinician that bowel perforation has occurred. The detection of free intraabdominal air may require the use of ventrodorsal horizontal beam radiography with the patient in left lateral recumbency. Free air can be detected just under the right abdominal wall and lateral to the duodenum. Volvulus or mesenteric thromboembolism is recognized by the presence of generalized, severely dilated and air-filled jejunal segments. Linear foreign bodies produce characteristic changes on abdominal radiographs in both cats and dogs. The small intestinal loops appear convoluted and gathered or clumped together at one site, usually in the mid-right abdomen and intraluminal gas bubbles appear asymmetrical and irregularly shaped.

Complications of obstructions

Gastrointestinal perforation affects the peritoneum of dogs and cats. Due to this, radiographic and sonographic features of perforation are characteristic of peritoneal disease. Clinical signs include fever, dyspnea, inappetence, vomiting, abdominal pain and possible diarrhea. Causes of perforation of the gastrointestinal tract in dogs and cats include foreign body perforation, perforating ulcer either due to benign or malignant diseases, non-steroidal anti-inflammatory therapy, bullet wound perforation, surgical dehiscence, intussusception, gastric dilatation volvulus.

Pneumoperitoneum

Free gas in the peritoneal space usually occurs with a perforated intestinal wall. However, chronic erosions of the wall due to neoplasm or chronic foreign body may be walled off and gas may not be evident radiographically. Free gas in the absence of recent laparotomy, trauma or abdominal perforation usually indicates intestinal perforation as the source. Presence of free air is in most all cases a surgical emergency. Rupture of the stomach usually leads to large amounts of air while if in the small intestine, the amount is smaller. Radiographic signs of pneumoperitoneum include loss of serosal detail as well as increased visualization of serosal margins due to outlining with gas. Horizontal beam radiography is indicated for suspicion of free gas when only small volumes are present. Free peritoneal air will tend to accumulate adjacent to the diaphragm, have triangular shapes, and may collect adjacent to the ribs and between the liver lobes. Large volumes of free gas can be more difficult to recognize radiographically than small volumes as they are so generalized over the abdomen they go unnoticed. The loss of serosal detail in the presence of free air is usually due to peritonitis secondary to the perforation. Radiographically, unless an intestinal mass or a radiolucent foreign body is identified, the site of intestinal perforation often is not identifiable.

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SVA REHABILITATION

INTRODUCTION TO PHYSIOTHERAPY TERMINOLOGY AND MANUAL THERAPIES

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INTRODUCTION TO PHYSIOTHERAPY TERMINOLOGY AND MANUAL THERAPIES

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The physiotherapeutic approach to patient evaluation places an emphasis upon proper and thorough soft tissue diagnosis. Problem solving is emphasized as are creating and meeting goals that are functional for the patient. As veterinarians, we have been trained to evaluate our patients, using imaging to help us evaluate bones and joints. When a dog presents with a lameness and the radiographs are negative, what can we do? This is where the skills used daily by physiotherapists can assist us. Determining specific soft tissue pathologies allows us to apply focused treatments to patients previously treated with "R&R" (Rest & Rimadyl).

Veterinarians have traditionally used "S.O.A.P. notes to record their thought processes on evaluating patients. Physiotherapists use a similar approach. Subjective data from the client/pet owner is combined with the medical history from the referring veterinarian as well as the objective data obtained through the physical examination. A detailed problem list is then created. From this list, an assessment is written. This is a narrative of the problem list. From this narrative, a list of **functional goals** is created. A detailed treatment plan is then written. As the treatment plan is pursued, the patient is reassessed to determine if goals were met. The treatment plan is then altered as necessary. The objective data collected includes the usual veterinary physical exam **plus** the following: Posture, Function, Strength, Gait, PROM/AROM, Flexibility, Joint Play, and Special Tests. Treatment plan development requires that the problem list be prioritized. The acuity and the primary tissue(s) of the injury are determined, leading to proper decision making regarding manual therapies, physical modalities, and therapeutic exercises. Treatments are then carried out, and the results evaluated before planning the next step.Determining the acuity of the injury requires understanding the

tissue responses during acute, subacute and chronic phases of healing. In the acute phase, there is initial tissue damage and an inflammatory response. Our treatment goals at this time are to decrease the acute pain, prevent exacerbation of the pain, and prevent resultant compensatory dysfunction. The subacute phase of healing begins when the inflammatory response has resolved. The injured tissues are still at high risk of reinjury and return to the acute phase. Treatment goals during the subacute phase include enhancing tissue healing, resolving compensatory pain and preventing exacerbation of the underlying injury. In the chronic phase, tissues are beginning to heal, scarring is taking place, but healing is incomplete. Here, the treatment goals are to complete the resolution of the underlying injury, recondition the tissues associated with the injury, and begin reconditioning of the entire body.As a review: The rehabilitation therapist gathers the subjective data, prior medical history, performs the evaluation, obtaining the objective data, and then creates a problem list. This problem list is translated into an assessment, from which functional goals are created. The treatment plan is developed, addressing each item on the problem list and working toward meeting the functional goals. Treatment choices are determined by the type of tissues that have been injured and the acuity of this injury.Treatment plan options fall into three categories: manual therapies, therapeutic exercises, and physical modalities. Manual therapies include stretching and joint mobilizations. Therapeutic exercises are chosen based upon the weight-bearing status of the patient. All exercise programs include work on proprioception and balance, strength, flexibility, and endurance. The type of exercise in each area is determined by weight-bearing status, which is defined as progressing from non-weight-bearing to partial weight-bearing to full weight bearing. Once an animal is fully weight-bearing on a limb, the types of exercises are changed from functional weight-bearing exercises to functional strengthening exercises. The patient's strength is then progressed from "<3 out 5" to "3 out of 5" to "5 out of 5". The goals of the therapeutic exercise program are determined by the goals of the client, but all programs will include work on proprioception/balance, strength, flexibility, and endurance.The parameters for each physical modality are chosen based upon the acuity of the injury. Physical modalities are generally used to prepare the tissues for manual therapies and therapeutic exercises. Most modality treatments can be carried out by a rehabilitation-trained veterinary nurse. Some manual therapies and therapeutic exercises must be done by the rehabilitation therapist (veterinarian or physiotherapist).

Physiotherapy Terminology

Physiotherapy brings a new set of terminology to the veterinary field. Some terms have been used regularly in veterinary practice, but others are new, and even the 'old' terms need to be more clearly defined.Range of motion (ROM) is determined by both osteokinematics and arthrokinematics and includes both active range of motion (AROM) and passive range of motion (PROM). Osteokinematics is defined as the movement of bony segments around a bony axis. There are two kinds of osteokinematics: AROM and PROM. Assessment of both AROM and PROM is an important component of the rehabilitation evaluation.Active range of motion (AROM) is carried out by the patient and is best assessed using slow motion video. Passive range of motion (PROM) is carried out by the therapist with no muscular effort on the part of the patient. This is measured using a goniometer aligned with specific bony landmarks. The goniometer has a stationary arm, a fulcrum, and a moving arm. The stationary arm is generally aligned with the proximal landmark. The fulcrum is aligned with the center of rotation of the joint, and the moving arm is aligned with the distal landmark. For example, in measuring elbow range of motion, the stationary arm is aligned with the greater tubercle, the fulcrum is placed over the lateral humeral epicondyle, and the moving arm is aligned with the lateral styloid process. Apps are available on mobile devices to measure goniometric angles on stop action video clips.AROM and PROM are used clinically to help tease out the source of impairment. For example, if a patient presents with limited AROM in shoulder extension with evidence of pain when moving into extension, but on PROM testing, full non-painful shoulder extension is achieved, what is your differential diagnosis? Pain on AROM with no pain on PROM leads to a diagnosis of muscle or muscle/tendon impairment. In this case, the primary differentials would be biceps or supraspinatus tendinopathy.Limited PROM can be the result of issues in muscle, tendon, intra-articular lesions, joint capsule shortening or swelling. Flexibility is different from ROM. Flexibility refers to muscle distensibility rather than joint arthrokinematics. To determine the source of loss of PROM, the therapist assesses end feels: the sensation or feeling in the therapist's hands when the joint is at the end of its available range.Arthrokinematics is the study of the movement of joint surfaces on one another. Normal arthrokinematics are essential before normal osteokinematics can occur. The two basic movements in all joints are roll and glide. Abnormal shortening of the joint capsule or associated ligaments will result in loss of normal arthrokinematics. The solution for this is joint mobilization that lengthens the shortened tissues. Joint mobilization is used to assess and treat limited arthrokinematics, and has the added advantage of modulating mechanoreceptors thereby reducing pain nociception.

WSV18-0101

POLYURIA / POLYDIPSIA

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Introduction

Polyuria and polydipsia (PUPD) is a common disorder in dogs and cats. As polyuria cannot be easily quantified, the diagnosis mostly relies on daily water consumption. Normal water consumption is ~50 ml/kg, however there is great variability among and within species. The definition of polydipsia is met when the daily water consumption is >100 ml/kg. In most of the cases, polyuria is the primary disorder and polydipsia is a compensatory mechanism to maintain normal hydration status. Less frequently, polydipsia is deriving the polyuria (e.g., primary polydipsia, neurological disorders, fever and pain). The most common mechanisms for polyuria include osmotic (including solute) diuresis, antidiuretic hormone (ADH) deficiency and conditions that alter the kidney response to ADH. In some disorders the pathophysiology of PUPD is multifactorial, involving both primary polyuria and primary polydipsia.

Establishing presence of PUPD

In some cases the owners do not recognize PUPD as a problem and in other cases PUPD may be the chief complaint. Incontinence, nocturia, pollakiuria or inappropriate urination should not be confused with PUPD, therefore accurate history is informative. Polydipsia can be confirmed easily by measuring daily water consumption. If the latter is not possible, urine specific gravity (USG) can be measured. PUPD is suspected when the morning USG is persistently low. The clinician needs to bear in mind that almost any USG can be normal, depending on the concurrent hydration status of the patient.

Diagnostic approach

For a rational diagnostic approach, the clinician needs to be familiar with the most common differential diagnoses in which PUPD is the presenting compliant. These include diabetes mellitus, chronic kidney disease (including pyelonephritis), hyperadrenocorticism, liver diseases, central diabetes insipidus (CDI), pyometra, and hypercalcemia. Other differential diagnoses that should be considered, however PUPD is less likely to be the main presenting compliant, include drug administration (e.g., phenobarbitone, glucocorticoids, diuretics), hypokalaemia, medullary washout, hypoadrenocorticism, congenital nephrogenic DI, post-obstructive diuresis, high salt or low protein diet. Rare differentials should be considered only when the diagnostic work up suggest that these are likely, or when other causes of PUPD have been excluded. These include primary renal glucosuria, Fanconi's syndrome and hyperviscosity.

History is an extremely valuable tool to further narrow down the differential diagnosis list, even prior to performing any laboratory test. For example, in a non-spayed female dog that was in estrus in the preceding weeks and also presents systemic clinical signs (e.g., lethargy/anorexia/voting), pyometra should be considered high in the differentials. If concurrent history also include polyphagia, diabetes mellitus, hyperthyroidism (in a cat), and hyperadrenocorticism should be considered. If the latter are also accompanied with weight loss, diabetes mellitus and hyperthyroidism (cat) should be considered. If the PUPD is accompanied with weight loss, CKD is more likely. Dogs and cats with some diseases are less likely to be bright alert and responsive (e.g., liver failure, hypoadrenocorticism, pyometra, hypercalcemia, advances CKD), whereas in others (e.g., CDI, primary polydipsia) animals are often bright alert and responsive, and PUPD is the only clinical sign present.

Physical examination findings may also aid in narrowing down the differential diagnosis list and occasionally be almost indicative of the diagnosis. Presence of anal sac mass in an old dog with PUPD, is highly suggestive of anal sac adenocarcinoma (promoting PUPD due to hypercalcemia). In most cats with hyperthyroidism, a cervical nodule can be palpated. Animals with hyperadrenocorticism, may present with concurrent dermatological disorders (alopecia, thinning of the skin, pot-belly), and animals with diabetes mellitus may present with varying degrees of cataract while small and irregular kidneys are suggestive of CKD.

To reach a final diagnosis, usually ancillary tests are required. Complete blood count, serum chemistry and urinalysis are indicated in animals presenting with PUPD. Urinalysis is an initial diagnostic test in such cases. The USG is usually iso- to hypo-stenuric, but may also be hyperstenuric in animals with diabetes mellitus. Unless USG is very now (<1.006), often times USG by itself does not narrow down substantially the differential diagnoses list. When the urine is extremely diluted, differentials like CDI, hyperadrenocorticism and primary polydipsia become more likely. Naturally, presence of glucose in the urine is highly suggestive of diabetes mellitus, however, glucosuria may also be present in face of normoglycemia in animals with primary glucosuria, Fanconi's syndrome, AKI or in animals with stress/drugs induced hyperglycemia. In the latter the glucosuria is expected to be transient. When bacteriuria and pyuria are present in urinalysis, pyolenephritis should be suspected, even in the absence of systemic clinical signs or changes in complete blood count (CBC) or chemistry, and urine should be submitted for culture and sensitivity.

Complete blood count is often times not rewarding, however it can be used to exclude some differentials (e.g., hyperviscosity) and in some diseases, CBC may increase the suspicion of one differential over the other. For example, stress leukogram and thrombocytosis may imply hyperadrenocorticism. Serum chemistry is extremely useful and in conjunction with the history, physical examination, CBC and urinalysis findings, further aid in narrowing down the differential diagnosis list. In animals with diabetes mellitus, hyperglycemia is expected. In diabetes mellitus and hyperadrenocorticism, increased activities of liver and biliary enzymes, hypertriglyceridemia, and hypercholesterolemia are expected. In cats with hyperthyroidism, liver enzymes are often elevated. In animals with hypoadrenocorticism, electrolyte disorders (hyponatremia and hyperkalemia) are common and may accompany hypoglycemia, hypocholesterolemia and hypercalcemia. In animals with kidney disease, creatinine and urea concentrations are often elevated. In animals with liver failure hypoalbuminemia, hypoglycemia, hyperbilirubinemia, hypocholesterolemia and decreased urea concentration may be identified. Hypercalcemia and hypokalemia can be easily identified as potential causes for PUPD.

Ultrasound examination often completes the initial diagnostic work-up. Hyperthyroid nodules, abnormalities in the adrenal glands, changes in liver and kidneys, should all be assessed.

The above diagnostic work-up is often sufficient to reach a diagnosis, or at least to substantially narrow down the differential diagnosis list. However, in certain occasions, additional tests are required to confirm the diagnosis (T_4, hyperadrenocorticism etc.).

In some animals with PUPD, CBC, serum chemistry, urinalysis and ultrasound examination are completely unremarkable. The main differentials to consider at this point include Stage I CKD (including pyelonephritis), hyperadrenocorticism, diabetes insipidus and primary polydipsia.

Presence of Stage I CKD can be further evaluated by symmetric dimethylarginine (SDMA) measurement, GFR (endogenous or exogenous creatinine or inulin clearance) and kidney biopsy. Hyperadrenocorticism should be considered even in the absence of any concurrent clinical signs or abnormalities in CBC, serum chemistry, urinalysis and ultrasound. If the index of suspicion for hyperadrenocorticism is low, urine cortisol to creatinine ratio (from voided urine sample obtained by the owners at home) can be used to exclude the disease. However, if the result is abnormally high, this test cannot confirm the diagnosis, and further tests like low dose dexamethasone suppression test and ACTH-stimulation test should be pursued.

In cases where all previously mentioned diagnostic tools were negative, the main remaining differentials include primary polydipsia and diabetes insipidus. In theory, calculated or measured serum osmolality can be used to differentiate these conditions, as in primary polydipsia, the polydipsia is deriving polyuria and thus serum osmolality is expected to be low, whereas in diabetes insipidus, the polyuria is deriving the polydipsia and thus the serum osmolality is expected to be high. In reality, compensatory mechanisms (i.e., drinking and urination) offset the primary mechanism for PUPD, and serum osmolality remains within the reference interval. Water deprivation test was previously suggested to differentiate primary polydipsia and diabetes insipidus and further to differentiate central vs. nephrogenic diabetes insipidus. However, this test is not risk free and thus, unless primary polydipsia is highly suspected (young, active dogs), this test is mostly replaced by an ADH trial. In this test, ADH is administered (orally or to the conjunctival sac) after the owners have quantified and established the daily water consumption. Water quantification should be continued daily after ADH administration. A 50% decrease in water consumption is confirmatory for the diagnosis of CDI. A common mistake is to rely on spot USG to confirm the diagnosis. One has to consider that often times, the change in urine USG on a spot sample may not be very different from the baseline, as the administered ADH is not active throughout the day, and when the serum ADH is low, the urine is quickly dilutes and a large volume of highly diluted urine masks the transient increase in urine USG.

WSV18-0008

FELINE CHRONIC PAIN SYNDROMES - MORE THAN MUSCULOSKELETAL

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Over the recent decade, there has been increased awareness of pain and attention to the alleviation of pain in cats. Investigation has focused primarily on chronic musculoskeletal pain. The purpose of this presentation is to address other types of chronic and neuropathic pain in cats. For an excellent review of all types of pain, the reader is referred to the WSAVA Guidelines for recognition, assessment and treatment of pain1.

INTRODUCTION

Pain isn't just about how it feels; it is also about how it makes you feel. It results in suffering and a feeling of hopelessness. According to the International Association for the Study of Pain, pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.

Pain may be nociceptive, associated with injury (thermal, chemical or traumatic/surgical), inflammatory or neuropathic. There is a lot of overlap, and, regardless of type, if adequately controlled and primary or secondary inflammation resolve, pain should not become irreversible.

Acute pain is associated with tissue damage and serves to change behaviour in order to minimize or avoid damage. It is beneficial in that it helps to optimize conditions in which healing can take place. It is self-limiting and stops once healing is complete. Chronic pain, on the other hand, persists beyond the expected healing process without a clear end-point. It is maladaptive and dysfunctional and does not support healing. It can have significant effect on physical wellbeing and psychology of the sufferer. Chronic pain may be considered a disease state. Chronic pain may be present as a result of ongoing medical inflammation (e.g., intestinal, lower urinary tract, oral/dental, musculoskeletal) or secondary to unrelenting nociceptive stimulation (injury and associated inflammation).

Neuropathic pain is a term that refers to pain that is directly caused, or instigated, by dysfunction of, injury to, or primary lesion in the nervous system. As damaged nerves fire spontaneously, they become hyper-responsive to even normal stimuli. This pathophysiology results from sequential changes occurring in the peripheral nervous system, spinal cord, brainstem and brain[1].

Neuropathic pain is often a result of surgery, especially amputations (e.g., tail, limb, onychectomy) or fractures when appropriate analgesic agents have not been used or used for a long enough duration. Nerve compression and diabetes also result in neuropathic pain as can any chronic, unrelenting pain regardless of cause2. Neoplasia, and probably interstitial/sterile idiopathic cystitis, are considered to be "mixed" as they have both inflammatory and neuropathic characteristics.

UNDERSTANDING PAIN

Tissue damage stimulates the nociceptors; this results in transduction (i.e., translation of the stimulus), transmission of the signal to the spinal cord where it is modulated (amplified or dampened) and then transmitted to the brain where the original stimulus is ultimately perceived (in the frontal cortex and limbic system). Therefore, it is likely that emotional and psychological elements play a role in cats as they do in people[3].

Acute pain must be treated until inflammation is sufficiently resolved that the pain pathway won't be aggravated anew. All patients need to be sent home with analgesic medication post-operatively, regardless of how "routine" the procedure is4-6. If inadequate analgesia was provided following surgery or other trauma, or wasn't administered for long enough, permanent changes to the central nervous system may occur resulting in the patient experiencing excessive and inappropriate pain. Persistent nociceptive input results in "wind-up", an increase in the excitability of the sensory neurons of the spinal cord. These hyperexcitable cells amplify the signal that is sent to the brain resulting in changes to receptors and a decrease in inhibitory signals descending from the brain. Thus, the patient has a lower threshold to pain, experiencing it at a lower intensity than is expected ("allodynia"), has a greater pain response, experiencing more pain than expected for a given stimulus ("hyperalgesia"), and may have pain over wider regions than expected. In other words, it is important, not only to provide pain relief, but to provide it for a long enough period[3]. (See Figure 1.)
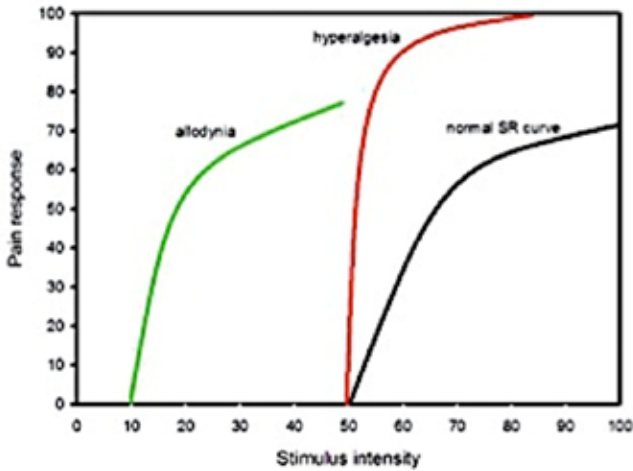
Figure 1: *Allodynia, hyperalgesia vs normal stimulus response curve. Image excerpted from Bergadano3*

Pain as an experience differs for each individual. Observe and adjust doses to make every patient comfortable.

CHRONIC PAIN: ESPECIALLY OLDER CATS BUT IN ANY AGE

Oral diseases such as periodontal disease, root exposure, resorptive lesions, stomatitis and oral ulcers and masses are all painful. Bacterial cystitis and pyelonephritis are more frequent in older cats but the prevalence of interstitial/sterile cystitis or inflammatory bowel disease does not differ from younger cats; inadequately addressed, these may cause on-going pain. The likelihood of neoplasia increases with increasing age. The need for analgesia MUST be considered as part of any treatment plan for the older cat. "Routine" procedures including blood collection, intravenous catheter placement, restraint of a thin or arthritic patient are uncomfortable.

Recognition of chronic pain and arthritic pain is relatively recent. The incidence of degenerative joint disease (DJD) appears to be much more common than previously thought and is probably a major cause of discomfort in ageing cats. In three studies retrospectively assessing radiographs taken of cats over 12 years of age7 or of any age8,9 the prevalence of findings suggestive of DJD was 90%, 22% and 34%, respectively with older cats showing radiographic changes. Only 4%, 33% and 16.5% had notation of restricted mobility in the medical record indicating that appropriate questions were not being asked of owners, that cats do not experience or that they don't show discomfort from these joint changes.

A recent study10 prospectively evaluated cats of all ages to determine the prevalence of radiographic signs of DJD. Most (92%) cats had radiographic evidence of DJD; 91% had at least 1 appendicular site affected and 55% had > 1 site of axial DJD. Affected joints in descending order of frequency were hip, stifle, tarsus, and elbow. The thoracic segment of the spine was more frequently affected than the lumbosacral segment. Grading the severity of each of the radiographic changes identified, they found that for every 1-year increase in age, the expected total DJD score increased by an estimated 13.6%. They concluded that radiographically visible DJD is very common in domesticated cats, even in the young and is strongly associated with age.

Yet lameness is not a common clinical sign of this problem in cats: signs are insidious or often attributed to ageing. They include inappropriate elimination (often adjacent to the litter box), decreased grooming, developing antipathy for being combed, reluctance to jump up or down, sleeping more, moving less, withdrawing from human interaction, and possibly even hiding. When activity monitors were attached to cats' collars, activity counts increased with meloxicam suggesting alleviation of musculoskeletal discomfort[11].

Wherever possible, the underlying cause of the pain should be identified and corrected.

IDENTIFYING CHRONIC PAIN

Because cats are solitary survivors, they are notoriously secretive in revealing discomfort and disabilities. When the presenting concerns from the client fail to include observations of pain, questions regarding behavioural or life-style changes may elicit clues. Changes in awareness, personality and interaction, an inappropriate activity level, reduction in playing, aggression, changes in sleeping patterns and litter box use may be present. Other indicators of on-going pain include a decrease in mobility or ease of jumping (up or down), inappetence or altered eating behaviours and a poor coat from lack of grooming12. Adults and older individuals are generally more stoic making it even harder to detect pain than in the kitten. Seriously ill or obtunded patients are especially difficult to assess for pain as they are less likely to display behavioural signs of distress when compared to an otherwise healthy injured cat[13].

Examination may reveal reluctance to being handled or having a particular body part palpated or manipulated and may result in self-defensive behaviour. Sedation/anaesthesia may be needed to properly assess oral and dental problems or for imaging. Radiographic, ultrasonographic or advanced imaging (MRI/CT) may be warranted to identify the underlying problem. Quantitated sensory testing may be undertaken to help localize the neuropathic lesion using different types of stimuli to identify the type (and therefore location) of nerve fiber affected[2].

Elimination trials may be undertaken to verify and alleviate pain. For example, a local block may be used to assess oral/dental problems or a regional block for a joint or paw. An analgesic trial, usually based around opioids with or without non-steroidal anti-inflammatory

drugs (NSAIDs) should also be considered when there is a suspicion of pain. The truest assessment of the presence of pain is response to analgesics resulting in return to normal behaviours[12].

There are no pathognomonic or unique clinical signs that characterize pain or that are present in every painful individual. Cats cannot directly communicate their discomfort to us. Several pain scoring systems exist, however they are either for assessing acute pain or have not been validated. The Feline Musculoskeletal Pain Index[14] is the exception and may be a starting point for non-musculoskeletal pain. Fear (especially in the clinic) may look like pain and some patients may be experiencing both. With patients and in a quiet, calm environment, it can be easier to identify signs of pain. Look for at least three indicative signs in the history, behavioural changes and examination[3]. (See Figure 2).

Figure 2: *Triangulation for identifying chronic pain. Adapted from Bergadano3*

The experience of pain is different for every individual, both in severity, duration and impact. An analgesic regimen, using single or multiple agents, needs to be tailored to the individual's needs through empathic and repeated assessment.

PREVENTING AND TREATING CHRONIC PAIN

Wherever possible, pre-emptive analgesia should be used to prevent stimulation of nociceptors and transduction of pain. Central sensitization can be prevented at the level of the N-methyl-D-aspartate (NMDA) receptors in the dorsal horn of the spinal cord. Ketamine is used, not only for its properties as a dissociative analgesic agent, but also specifically to block these receptors, thereby acting as an analgesic and as an anti-hyperalgesic agent.

When pre-existing inflammation, inadequate peri-operative analgesia with resultant neuropathic pain exist, or if the cause of pain cannot be treated, then an effective analgesic protocol must be developed in order to provide the patient with the best quality of life possible. Providing multimodal, balanced analgesia impacts multiple sites of the pain pathway while reducing the risk of negative effects from any one class of drug. This may be achieved through the concurrent use of an opioid with an NSAID and possibly amantadine (NMDA receptor antagonist) for maladaptive pain. Analgesic choices and doses for cats are listed in Table 1.

Analgesia for chronic musculoskeletal disease

The cat with joint pain is often an older patient who may have concurrent problems (e.g., renal disease) including some that may affect drug metabolism[15]. Like painful patients of any age, they may be in a physiologic state that affects drug disposition, the most common ones being dehydration, inadequate tissue oxygenation, electrolyte or acid-base imbalances and malnutrition. The most common concern regarding NSAID side effects is the possible consequence of using this class of drug in a dehydrated patient resulting in effects on gastric mucosal health or on renal function[16]. Dehydration may be subclinical and difficult to assess in the very young and in the older cat due to the unreliability of skin elasticity in these age groups. (Stool consistency [i.e., pellets rather than formed logs] can be helpful in evaluating hydration.)

Opioids are safe for pain relief in any age group and are excellent when used at the same time as other agents, especially NSAIDs. They are not, however, a first drug of choice for cats with arthritic pain as they are not very effective for DJD. This is not to suggest that they shouldn't be used for "break-through" pain or for comfort during diagnostic testing. If they produce adverse side-effects (e.g., euphoria, constipation and inappetence) in an individual patient they may be reserved for palliative hospice care.

Pharmacokinetic data is lacking for safe, long-term use of many NSAIDs in cats. Carprofen half-life varies from nine to over 40 hours in cats[17,18]. As most NSAIDs have long half-lives in cats when compared to other species, one precaution to avoid toxicity is to reduce the frequency of administration. Interestingly, despite having a short half-life of under 2 hours in blood, robenacoxib (Onsior®) its effect persists for 24 h in clinical studies.

Metacam® 0.5 mg/ml oral suspension has been granted a licence in the EU for the alleviation of inflammation and pain in chronic musculoskeletal disorders in cats. The registered dose is 0.1 mg/kg on the first day followed by 0.05 mg/kg orally once daily. This is the first NSAID licensed for long-term use in cats.

Numerous efficacy studies have been performed regarding both of these NSAIDs. In one, clients felt that cats treated for one month with meloxicam were more willing to jump achieving progressively higher heights during the study. Evaluation of the cats by the veterinarian at the end of the month showed a significant reduction of gait stiffness 19. Three studies have evaluated long-term safety of this agent in older cats; one concluded that this agent is safe, efficacious and palatable for musculoskeletal pain at 0.01-0.03 mg/kg PO q24h for a mean treatment duration of 5.8 months; no deleterious effect on renal function was detected in cats studied. Gastrointestinal upset in 4% of cats was the only adverse effect noted20. The second and third, reviewed

the medical records of cats over 7 years of age treated for a minimum of 6 months with a daily maintenance dose of 0.02 mg/kg meloxicam and concluded that this dose does not hasten progression of renal disease in aged cats or aged cats with pre-existent stable IRIS stage 1-3 renal disease21,22.

In 2015, a paper reported on the safety of robenacoxib (1–2.4mg/kg) for daily, month long treatment of DJD in cats including 40 with chronic kidney disease IRIS stages 2-4. There was no evidence of increased risk in the frequency of reported adverse events, or in deterioration in renal variables in the subgroup of cats with concurrent CKD23. Despite being similar to meloxicam (class, mechanism of action), by September 2016 it was licensed only for short-term use.

Excretion and metabolism of meloxicam have been studied in cats. After oral administration, the major route of excretion is fecal and the main pathway of biotransformation is by oxidation, rather than by their limited glucuronidation pathway. Additionally, 21% of the recovered drug was eliminated in urine (2% as unchanged meloxicam, 19% as metabolites) and 79% in the feces (49% as unchanged meloxicam, 30% as metabolites)24.

A comprehensive review of the long-term use of NSAIDs in cats was published in 2010. This document may be accessed free-of-charge at: http://www.catvets.com/guidelines/practice-guidelines/nsaids-in-cats in Spanish, French, German and Japanese25. In addition, an educational client brochure (Spanish, French) regarding the safe use of NSAIDs in cats is also available at the same web link. To minimize the risks of NSAIDs, it is important to:

- Select appropriate patients: individuals should maintain hydration and not be hypovolemic, hypotensive or in congestive heart failure.

- Obtain a complete list of medications the cat is receiving or has access to.

- Base the dose on lean body weight and consider titrating, once pain is controlled, to lowest daily dose that maintains comfort.

- Use a balanced approach: include nutritional, adjunctive and environmental components.

- Use gastroprotectants to treat or prevent gastric upset.

- Ensure communication with clients through verbal and written instructions.

- Recognize adverse reactions promptly and discontinue the NSAID.

- Monitor blood work q 2-4 months (high risk patients) or q6 months (low risk patients25.

- A washout period of 3-5 days should be used if transitioning from one NSAID to another; a longer washout period is indicated (7-10 days or longer) when switching to, or from, aspirin or a corticosteroid. Additional, alternate analgesic agent(s) should be used during the washout period.25,26

A suitable protocol for a cat with pain from musculoskeletal disease might be baseline NSAID with intermittent use of an opioid (such as burprenorphine) when "break-through" pain is evidenced by a decrease in appetite, mobility or social interaction. Gabapentin may be added for ongoing care.

Environmental modifications: Regular nail trimming helps by maintaining proper joint relationships. Ramps and steps to favourite sleeping spots are helpful. Warm, soft, padded sleeping places for stiff, painful, possibly bony joints should be considered. Raising food and water bowls may help the cat with cervical vertebral changes. Adding a litter tray to reduce the distance between boxes may reduce accidents as well as encourage regular voiding and defecation. The rim of the tray mustn't be too high, nor the opening into the box too small. It should be scooped several times a day to encourage use.

Feeding a diet that is supplemented with eicosapentaenoic acid (EPA), and docosahexaenoic acid (DHA) +/- green-lipped mussel (GLM) extract and glucosamine/chondroitin sulfate may be beneficial. Disease-modifying agents such as polysulfated glycosaminoglycan, glucosamine and chondroitin sulfate may improve joint health27. Additional modalities (therapeutic exercise, acupuncture, cold laser therapy) while no scientific studies have been done to support efficacy, may also play a role in providing comfort for a cat with musculoskeletal discomfort.

The author recommends that for chronic administration of NSAIDs in cats, it is good clinical practice to use the lowest effective dose based on lean body weight, tapering to the lowest effective daily dose (off-label) and to avoiding use in, (or use lower initial doses) in cats with renal disease. Ensure that the patient is hydrated and give the NSAID with food. Individual patients respond differently to the same agent and dose. In most cases, NSAIDs are most effective when used in conjunction with other treatment modalities.

ANALGESIA FOR NEUROPATHIC PAIN

Neuropathic pain may be caused by inadequately alleviated traumatic or surgery-induced pain, such as onychectomy (declaw) or amputation28. Clients may remark that their cat doesn't jump as high as before the procedure or walks as if on glass or eggshells. Alternately, they may note decreased activity, increased aggression, inappetence starting months or even years after surgery. While the initiating event may be known,

it is imperative that radiographs of affected paws be taken to rule out a bone remnant, a surgically treatable problem. When none is found, neuropathic pain is treated by addressing "wind-up" while concurrently providing analgesia. Analgesics alone are ineffective. Amantadine is used off-label to block the NMDA receptors in the spinal cord, however, because it lacks analgesic effects, an opioid plus NSAID are used concurrently. The regime suggested by Gaynor is outlined in Table 2.

Another condition, feline orofacial pain syndrome (FOPS) is a disorder of cats with behavioural signs of oral discomfort and tongue mutilation. There is suggestion that it is inherited in an autosomal recessive manner. It is believed to be neuropathic in nature and characteristically includes exaggerated licking and chewing movements, and pawing at the mouth; in some extreme cases mutilation of tongue, lips and buccal mucosa occurs. It appears to be triggered by mouth movements (grooming, eating)29. Like idiopathic cystitis, it occurs at irregular intervals with the cat appearing to be pain-free in between these episodes. In both, external factors can also influence the disease such as anything causing stress or anxiety as well as other illness29,30.

Therapy for FOPS includes ruling out other causes of facial and oral pain, any dental disease discovered should be treated. An attempt to identify and eliminate environmental stresses and triggers should be made. Pain relief requires multiple agents including NSAIDs plus phenobarbital, carbamazepine, gabapentin or amitriptyline. Treatment is long-term and may not be successful in some cases31.

NOVEL MEDIATORS OF PAIN

New mediators of pain have been identified and are being studied as therapeutic targets. These include nerve growth factor (NGF), piprants, neurokinin-1 antagonists, selective neurotoxins and cannabinoids.

• Nerve growth factor

This mediator of inflammatory and neuropathic pain is elevated in models of chronic and animal pain32. Hyperalgesia is alleviated by inhibition of NGF. Numerous approaches are being evaluated, (e.g., monoclonal antibodies) to negate its effect. Risks and benefits of Tanezumab have been studied in human medicine for interstitial cystitis, osteoarthritis, diabetic neuropathy and post-herpetic neuralgia. A felinized anti-NGF monoclonal antibody (NV-02, Frunevetmab, Nexvet Biopharma) has been developed; multicenter clinical trials are underway33-34.

• Pripants

Pripants and substances that antagonize prostaglandin E2 EP4 receptor, i.e., further down the inflammatory cascade than NSAIDs thereby not interfering with the "housekeeping" actions of COX enzymes. While not yet approved for use in cats, grapiprant (Galliprant®, Elanco) has been studied in this species and has received FDA approval for use in dogs with DJD35.

• Neurokinin-1 antagonists

This class of drug prevents substance P from binding to NK-1 receptors. Maropitant is typically used as an antiemetic but appears to provide visceral analgesia in dogs as indicated by reduced anaesthetic requirements during ovariohysterectomy.

• Selective neurotoxins

Two selective neurotoxins have been studied in dogs: resiniferatoxin and substance P-saporin. These selectively inhibit or destroy cells in receptors. It is too early to say whether these will play a role in veterinary analgesia.

• Cannabinoids

The use and benefits of medical marijuana for people continue to be investigated. Cannabinoids

interact with receptors in the endocannabinoid system. These include cannabidiol (CBD), cannabinol (CBN) and tetrahydrocannabinol (THC) receptors. Fractions that target CBD and CBN receptors (e.g., as in hemp oil) but not THC receptors might be of use; THC is dangerous for small animals. Research is lacking for use for cannabinoids in cats at this time.

Table 1: Analgesic choices for chronic pain in cats

Drug Class	Drug	Follow-Up Analgesia or Chronic Pain Mild-Moderate Pain	Follow-Up Analgesia or Chronic Pain Moderate-Severe Pain
OPIOIDS	Butorphanol	0.1-0.4 mg/kg IV, IM, SC q2h	
	Buprenorphine	0.01-0.03 mg/kg IM, IV, SC q6-8h; 0.01-0.03 mg/kg q6-8h bucally	
	Morphine*		0.1-0.2 mg/kg IV q1-4h or 0.1-0.5 mg/kg IM q2-6h
	Hydromorphone**		0.08-0.3 mg/kg IV, IM q2-6h
	Fentanyl		0.004-0.01 mg/kg IV q 20

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			min or 0.001-0.004 mg/kg/h CRI or fentanyl patch 12.5-25 mcg/h q 4-5 d	
OPIOID REVERSAL OR TITRATION	Naloxone: for reversal/titration of opioid dose:	Dilute 0.1 ml of 0.4 mg/ml naloxone in 5 ml 0.9% NaCl; administer at 1.0 ml/minute to effect	Same	
NSAIDs***	Meloxicam	0.1 mg/kg PO on d1, then 0.05 mg/kg PO q24h long term; consider titrating to lowest effective daily dose (off-label)	Same	
	Robenacoxib	1-2 mg/kg PO q24h for 3 days (US, Canada), 6 days (EU)	Same	
	Ketoprofen	≤ 2.0 mg/kg SC once, then < 1.0 mg/kg q24h for 4 days maximum	Same	
	Tolfenamic acid	≤ 4.0 mg/kg SC, PO q24h, for 3-5d	Same	
NMDA RECEPTOR ANTAGONISTS	Ketamine	0.5 mg/kg IV prn (q 30 min)	0.1-0.5 mg/kg/h IV CRI combined with morphine	
	Amantadine		3-5 mg/kg PO q24h for neuropathic pain	
SEDATIVES for chronic pain of various levels in combination with opioids (not analgesics; provide relaxation)	Midazolam	0.1-0.5 mg/kg IV, IM q8-12h	Same	
	Diazepam	0.1-0.5 mg/kg IV q12h	Same	
	Acepromazine	0.01-0.05 mg/kg IV q1-2h or 0.02-0.1 mg/kg IM, SC q2-6h	Same	
	Medetomidine	0.02-0.05 mg/kg IM q 4-6h or 0.01-0.02 mg/kg IV prn	Same	
TRICYCLIC ANTIDEPRESSANT	Amitriptyline for idiopathic cystitis	2.5-12.5 mg/cat PO q24h		
	Gabapentin****	5-10 mg/kg PO q12-24h for *mild-moderate* post-op, follow-up, chronic or neuropathic pain		

* Morphine: Pretreat with Benadryl if administering IV.

** Hydromorphone: Caution: doses of 0.1 mg/kg and higher can cause hyperthermia in some patients; severe hyperthermia is an indication to change analgesic class.

*** Meloxicam: The author and all cited studies are referring to Metacam® throughout this article. Compounded formulations lack third party, mandated and assessed quality control (QC); there is known potency variability between compounders and even between batches from the same compounder.

**** Gabapentin: Taper dose when withdrawing drug. Do not discontinue abruptly.

Table 2: *Off-label protocol for alleviating neuropathic pain from onychectomy (Gaynor)[24]*

Administer concurrently: Amantadine 3 mg/kg PO q24h X 21 days,

+ Buprenorphine 0.01-0.02 mg/kg buccally q12h X 2-3 days,

+ Meloxicam starting at 0.05 mg/kg PO q24h X 4 days => tapering to 0.05 mg/2 kg PO q24h X 4 days => followed by 0.05 mg/cat PO q24h X 4 days and finally => 0.05 mg/cat PO q48h X 5 days.[†]

[†]The ISFM and AAFP consensus guidelines: Long-term use of NSAIDs in cats21 recommends once daily dosing rather than other frequencies.

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GASTROENTEROLOGY / LIVER

PANCREATITIS, CHOLANGITIS AND 'TRIADITIS' IN CATS: ONE DISEASE OR MANY?

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What is triaditis?

'Triaditis' is the term coined many years ago in a lecture by David Twedt in the USA to denote concurrent chronic cholangitis, chronic pancreatitis and inflammatory bowel disease in cats. It is a frustrating term because in fact, a literature search will show you there are NO publications on 'triaditis' in cats. There **are** publications on 'triaditis' in humans but they mean something very different – inflammation of the portal triad - i.e. confined to the liver. The term as used for cats could therefore lead to confusion, particularly in comparative studies – but it has gained widespread acceptance as a short-hand way of reminding us that these three conditions can occur together in cats.

There is continuing debate about whether these three conditions are really related in cats. The first published report was Weiss and others in 1996 who reported an increased prevalence of inflammatory bowel disease (IBD) and pancreatitis in cats with cholangiohepatitis. More recent studies of cholangiohepatitis seem to support this: for example, in a study of MRI in cats with cholangitis or pancreatitis , 8 out of 10 cats had both histologically confirmed pancreatitis and cholangiohepatitis on laparoscopic biopsies. In another study of ultrasound in 26 cats with cholangitis, 17 had concurrent ultrasonographic abnormalities in the gut wall and 7 had concurrent pancreatic abnormalities.

The evidence does suggest, therefore, that a sub-set of cats has concurrent gut, liver and pancreas disease. There are also cats which suffer from one or the other but not all three.

Why does 'triaditis' occur in cats?

The short answer is nobody knows. There are a number of possible reasons but more research will be needed to identify which of these are important. Broadly speaking, chronic pancreatitis, chronic cholangitis and IBD could occur because of an infectious process; an autoimmune process or something physical (such as duct obstruction). An autoimmune disease could also involve food allergy (particularly in the gut) and an infectious cause could also encompass an unusual host reaction to their own microflora. In reality, particularly for chronic pancreatitis and chronic cholangitis, it is likely that cats suffer from several different diseases and not just one disease and that the aetiologies vary. It will be very difficult to define effective treatments until we know the causes and can separate out cats into their different groups.

Many authors simplistically suggest that cholangitis and pancreatitis occur because the pancreatic and bile ducts join before they enter the duodenum. Cats are like humans in this respect, whereas in most dogs the ducts don't join and enter the duodenum separately. Having a single outflow through the sphincter of Oddi would certainly pre-dispose to pancreatitis and cholangitis (although not explain the concurrent IBD) if both ducts were blocked concurrently. This is unusual in cats – in humans, the commonest reason for this to occur would be gall stones blocking the ducts near the sphincter of Oddi. This is very rare in cats. Cats do sometimes suffer from poorly defined sphincter dysfunction which may cause this syndrome: 'sphincter of Oddi dysfunction' has been described in both humans and cats and describes a spasm of the sphincter which can block both pancreatic and bile ducts. It produces a dynamic obstruction which can be very difficult to diagnose without dynamic imaging with stimuli for gall bladder emptying. However, it is diagnosed more often in humans with IBD and it is proposed that inflammation in the intestinal wall around the sphincter predisposes to it. Too few cases have been reported in cats for us to know if there is any association with IBD – but it is likely to be a condition which is under-diagnosed in cats because it will not be found unless you are looking for it.

Some cats may get ascending infection from the gut into both ducts, which potentially could be predisposed by vomiting or overgrowth of bacteria secondary to IBD, but it is not clear how often this happens. Bacteria could also pass across the mucosal barrier and into the portal blood circulation and then enter the liver (Twedt et al 2014). A second possible reason could be close proximity of the pancreas, bile duct and small intestine, such that inflammation of infection in one organ has a 'local' effect spreading to the others. This could be particularly true with pancreatitis as the bile duct passes through the pancreas so could be blocked during an episode of pancreatic inflammation and the neighbouring small intestinal wall might also be involved. David Twedt and Kenny Simpson have performed fluorescent in situ hybridization (FISH) on liver and pancreas from cats with cholangitis and pancreatitis and shown bacteria within the organs: but it is not clear whether these are a primary cause or secondary phenomenon.

The third possible reason, which seems very plausible in a number of cases, is that all three organs are affected by the same disease process, as can occur in humans. The bile duct, pancreatic duct and small intestine might all be concurrently affected by autoimmune disease, similar to IgG4+ cholangitis in humans. It is worth considering human biliary tract disease because it helps to understand how relying entirely on histology in cats may be leading us to put several diseases together. We need to develop better methods of imaging and blood tests to help us understand these feline diseases further.

A summary of human diseases with similar clinical and histological appearances

There are three (or four) diseases of the biliary tract in humans which all have a very similar appearance on histology and yet are clinically very distinct.

- Primary sclerosing cholangitis

- Primary biliary cirrhosis

- IgG4+ cholangitis

- Sphincter of Oddi dysfunction)

I will describe these as an example of how relying entirely on histology in cats may be leading us to put several diseases together. We need to develop better methods of imaging and blood tests to help us understand these feline diseases further.

In a recent, multi-centre, unfinished (!) project, we looked at 39 cases of chronic cholangitis to ask the questions: what type of pathology is present? Are any similar to primary sclerosing cholangitis in humans?

Based on the human classification systems, a human pathologist, grading the cats 'blind', categorised nine cases as secondary biliary cirrhosis – suspected chronic extra-hepatic obstruction; seven cases as PSC-like changes; five cases as vanishing bile duct syndrome; one case as PBC-like changes and the rest non-specific 'cholangiopathy'. Whatever is going on in these cats with chronic cholangitis, we undoubtedly have several diseases present and we are undoubtedly missing the diagnosis of extra-hepatic biliary tract disease in many cases.

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STATE-OF-THE-ART LECTURE FELV AND FIV IN 2018 - CLINICAL APPROACH AND MANAGEMENT

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FeLV and FIV in 2018 – Clinical approach and management

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Identifying FeLV or FIV infection is important to optimize individual health care and prevent new infections. Serology is the first line diagnostic test and positive results on serology should be confirmed. Interpretation of retrovirus test results requires integration of the clinical findings, patient risk factors and performance of the tests being used.

FeLV overview

FeLV is a gammaretrovirus that infects domestic cats and other felidae worldwide. FeLV is transmitted vertically and horizontally. Infection spreads rapidly within households through oronasal contact with virus-containing secretions, principally saliva. Vaccination and changes in management practices have reduced the global prevalence of FeLV infection. However, testing for FeLV is still relevant because FeLV carries a poor prognosis, there is no effective treatment and transmission of infection to in contacts should be prevented.

FeLV testing and outcomes

Screening point-of-care (PoC) tests detect viral antigen, p27, in whole blood, plasma or serum. These kits usual perfom very well; the IDEXX SNAP Combo showed 100% specificity and sensitivity for detecting FeLV antigenaemia recently.(1) Following FeLV exposure, cats initially test antigen positive. A stable outcome, influenced by age, immune status and virus dose, is reached within weeks. Around 30% of exposed cats develop **progressive infection** (persistent antigenaemia, high proviral load). Cats with progressive FeLV infection are the source of infection for naïve cats, and they have reduced life-expectancy that is often only days or weeks. FeLV-related diseases include anaemias, immunosuppression and lympho or myeloproliferative diseases. In the remaining exposed cats, antigenaemia is transient, but residual virus can be detected by quantitative polymerase chain reaction (qPCR, also called

real-time PCR). These cats have **regressive infection**, where integrated provirus (DNA) and, sometimes viral RNA, can be detected by qPCR, but at lower levels and with restricted tropism compared with progressively infected cats. The prevalence of regressive infection reported in the field ranges from less than 1% to 10%. Regressively infected cats pose minimal risk to other cats, although disease outcomes are still under investigation. Loss of immunological containment of regressive infection resulting in progressive infection seems to be rare. Blood donors with regressive infection can transmit FeLV infection with recipients developing fatal progressive infection. Therefore, feline blood donors should test negative on FeLV antigen **and** qPCR tests.

Abortive infection, only detectable by seroconversion, is rare and of no consequence to the cat or in contacts.

Interpretation of FeLV test results

Positive serology must be confirmed with qPCR. Most cats exposed to FeLV will test positive for FeLV antigen initially and retesting is important. p27 positive and qPCR positive cats should be isolated from negative cats and rested around 12 weeks later. If the cat develops progressive infection, it will remain persistently antigen positive. If the cat develops regressive infection the antigen test will become negative. Cats with known exposure to FeLV that test p27 negative should be retested in 30 days. Neither vaccination against FeLV or maternal antibodies affect FeLV p27 serology results.

FIV overview

FIV is a retrovirus in the lentivirus genus. Worldwide seroprevalence varies from <5% to >30% depending on the region and the population tested. Territorial aggression is the major mode of FIV transmission and free-roaming, entire, sick, male cats are at high risk from infection. FIV-infected cats are at risk from immune dysfunction although many have a normal life expectancy. Immune dysfunction can manifest as atypical and refractory bacterial, viral or protozoal infections and parasitoses. In second stage FIV, persistent cytopenias, high grade lymphoma, unexplained weight loss and neurological signs can occur. Confinement of uninfected cats is the most effective way to prevent transmission. A field study demonstrated a protective rate of 56% for Fel-O-Vax FIV vaccine (Boehringer Ingelheim).(2) FIV presents no known zoonotic risk

FIV serology

Serology is the first line test for FIV. PoC tests or laboratory-based ELISAs have high sensitivities and specificities for antibody detection. In patients over 6 months of age that have not been vaccinated against FIV, anti-FIV antibodies are a marker for FIV infection. Where vaccination with Fel-O-Vax FIV is available, test

selection is important because PoC kits vary in their ability to distinguish vaccine-induced from infection-associated anti-FIV antibodies. Witness FeLV/FIV (Zoetis) and Anigen Rapid FIV/FeLV (BioNote) showed high specificities for identifying FIV-infection in several studies.(3, 4) Interference with these tests by vaccination can still occur particularly after a recent booster. (5, 6) IDEXX SNAP FIV/FeLV Combo is not useful to discriminate vaccinated from infected cats. The reliability of a negative serology result is unchanged by the introduction of the FIV vaccine.

FIV molecular testing

Molecular tests target FIV DNA provirus and some also detect viral RNA in plasma by qPCR. Diagnostic sensitivity that is 5-15% lower than serology and specificity comparable with serology are reported for commercial qPCR tests.(7, 8) qPCR should not be used as a screening test for FIV. qPCR can be used as an adjunct to serology to determine the true FIV-status of cats in certain circumstances including; seropositive cats that may be vaccinated against FIV, seropositive cats less than 6 months of age and seronegative cats that have may been recently exposed to FIV. qPCR can detect viral RNA in blood within 1 to 2 weeks and DNA within 3 weeks of experimental infection but differences in virus strain, dose and assay sensitivities mean that it is hard to predict how long after natural exposure qPCR can reliably detect FIV infection.(9) A positive FIV qPCR results indicates that the cat is almost certainly infected, whereas negative qPCR results are inconclusive.

Interpretation and confirmation of FIV serology

Interpretation of the results of serological screening requires the integration of clinical findings from the individual cat to guide whether repeat serology or qPCR is indicated and what the timing of repeat testing should be. Negative serology results are generally reliable because the sensitivity of serological tests approaches 100%. The time to seroconversion following natural infection is variable and can be prolonged. A seronegative cat that may have been recently exposed should be isolated from FIV-infected cats or cats of unknown FIV-status and retested after 60 days. Rarely, cats in the terminal stage of FIV-infection have impaired antibody production despite high plasma viral loads. If advanced FIV infection is suspected, negative serology results should be followed-up with qPCR testing.Confirmation of positive serology results is recommended. The testing methodology used for confirmation will depend on assessment of the patient's FIV-infection risk, test availability, whether or not prior FIV vaccination can be ruled out and financial considerations. In cats with a low FIV risk, such as healthy, purebred, young, indoor cats, confirmation of positive serology is essential because the positive predictive value (PPV) of serology is reduced in low-risk groups. The reason for

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this is that the prevalence of FIV in a low risk population approaches the expected frequency of false positives. So for each positive test result obtained in cats at low risk, there is a higher chance that the result is a false positive. Maternally-derived FIV antibodies may result in positive serology in uninfected kittens. A positive serology results in a cat under 6 months of age can be confirmed with qPCR. Alternatively, serology can be repeated after 6 months of age. Negative serology in kittens is reliable. Where prior vaccination with Fel-O-Vax FIV is confirmed or cannot be ruled out, Anigen Rapid FIV/FeLV or Witness FeLV/FIV are useful screening tests. A positive result using one of these kits can be confirmed with the other kit or with qPCR depending on availability and finances.

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ONCOLOGY

CURRENT TREATMENT OPTIONS FOR MAST CELL TUMORS IN DOGS AND CATS

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CURRENT TREATMENT OPTIONS FOR MAST CELL TUMOURS IN DOGS AND CATS
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Learning objectives: Understand the perioperative management, staging and surgical oncology principles for cutaneous MCT in dogs and cats, and the indications for adjunctive radiation therapy or chemotherapy in conjunction with surgery. Understand treatment options for non-resectable MCTs.

Canine cutaneous MCTs:

Diagnosis:

- Cytology is usually diagnostic, and finding of suspected high grade MCT on cytology is highly predictive for histologic high grade.

- In my practice, complete staging involves evaluation of local/draining lymph nodes, abdominal ultrasound with liver and spleen cytology, and bone marrow evaluation. Bone marrow is typically reserved for cases with CBC abnormalities because it is uncommon (< 5%).

- Lymph node (LN) evaluation is always recommended because low-grade MCT may metastasise. MCTs affecting the muzzle and perioral tissues may be more likely to metastasise. LN mapping is ideal to identify the 'sentinel' node. Diagnosis of metastasis from both cytology and histology can be challenging, but criteria have been recently published that may assist (1, 2). Good outcomes can be achieved with aggressive treatment in low grade, stage 2 MCT so identifying metastatic LN prior to surgery is important. Abdominal ultrasound and liver and spleen cytology is recommended in cases of known or suspected high grade tumours or when grade is unknown and local therapy is likely to be radical.

- Histology: The 2-tier histologic grading scheme (low/high grade) has better inter-pathologist agreement than the 3-tier scheme, however combining the two may give additional information. Mitotic index (per 10 high power fields) is i prognostic, though the most useful value is not fully determined. The significance of additional prognostic factors (Ki67, AgNORs, c-kit staining pattern and mutation) is, in my opinion, not fully determined in terms of effect on prognosis.

Treatment:

- Surgery: For MCT with distant (beyond LN) metastasis, removal of the primary tumour is not likely to impact prognosis, though it may improve quality of life in painful/ulcerated tumours. General oncologic principles of local en bloc resection with a margin of normal tissue around the tumour apply for cutaneous MCT. MCT should be minimally manipulated during preparation and surgery to decrease degranulation. Many MCT will have definitive excisional surgery on the basis of a cytology result. As a general rule, 1cm lateral margins are adequate for Grade I MCT, 2cm lateral margins for Grade 2 MCT and 3cm lateral margins for Grade 3 MCT. These have not been re-evaluated for the 2-tier grading scheme. If the grade is not known, then a minimum of 2cm lateral margins is recommended. For cytologically diagnosed MCT where definitive surgery is feasible based on size and location, biopsy for pre-operative grading is not usually required. Deep margins are more qualitative than quantitative. The deep margin should comprise fascia or muscle. Careful pre-operative palpation and imaging (Ultrasound, CT or MRI) are useful to determine degree of fixation to deeper structures in planning the deep margin. The excised tumour specimen should be inked to facilitate histological margin assessment. If the MCT is greater than 1cm in diameter, then the skin surface should be cut at 1cm intervals to allow appropriate fixation.

- Adjuvant local therapy: In cases of incomplete or narrow histological margins (i.e. risk of local recurrence), additional local therapy with revision surgery or radiation therapy is recommended. Electrochemotherapy is a newer modality which may also be an option. It is important to remember that the 'safe' histological margin (i.e. sufficient to prevent local recurrence) has not been determined for MCT and is influenced by grade. Many incompletely or narrowly excised low-grade MCT do not recur, however additional local therapy with re-excision or radiation therapy has been shown to improve survival and so should be considered in all cases (3).

- Adjuvant systemic therapy: Chemotherapy is recommended following surgery in high grade MCT, even without visible metastasis, as adjuvant chemotherapy appears to improve survival over surgery alone. The ideal protocol for adjuvant chemotherapy in high grade/ high risk MCT is not fully determined. Vinblastine and prednisolone appears to be most commonly used, but protocols including CCNU alone or alternating with vinblastine are also reported. The adjuvant use of TKIs such as toceranib (Palladia) or masitinib (Kinavet/Masivet) is not well studied, though one retrospective study reported improved outcome in dogs with high risk MCT receiving adjuvant vinblastine and prednisolone compared to those receiving masitinib (4). The other major challenge with adjuvant use of TKIs is determining appropriate duration of treatment. The use of adjuvant corticosteroids or anti-histamines has not been studied and is not generally recommended.

Non-resectable primary tumours:

- Radiation therapy (RT): Palliative or definitive RT may be considered. There are no large prospective studies, but it appears that the addition of prednisolone +/- toceranib may improve response (approximately 80% versus approximately 50%) and control duration over palliative RT alone.

- Chemotherapy: Many different chemotherapy drugs alone and in combination (+/- corticosteroids) have been studied, including vinblastine, CCNU, TKIs, paclitaxel, cyclophosphamide and hydroxyurea, and metronomic chlorambucil. Response rates range from approximately 20% to 80% and response durations from weeks to many months. In general, combination therapies seem more effective. I usually offer three options for chemotherapy for non-resectable MCT: 1) 'traditional' chemotherapy with vinblastine and/or CCNU along with prednisolone 2) toceranib with prednisolone or 3) metronomic chlorambucil and prednisolone

- Alternative local therapies: Electrochemotherapy results in response rates of 60-80% in the small studies published to date, and is generally well-tolerated. Intra-lesional triamcinolone may be an effective option in some cases, though responses are generally relatively short-lived.

- Treatment of metastatic MCT: In cases of MCTs with LN but no distant metastasis (stage 2), aggressive local therapy, often along with adjuvant chemotherapy is generally recommended. For low-grade stage 2 MCT aggressive local therapy (surgery +/- radiation therapy) may be sufficient for long term control. In distant metastasis, systemic treatment options as for non-resectable MCT are typically attempted, with some retrospective data suggesting that using vinblastine and CCNU is more effective than toceranib.

- Supportive care: Anti-histamines +/- antacids are recommended in all dogs with gross MCT disease

Feline MCTs

- Cutaneous: The majority are benign, but a subset are more aggressive and identifying those is challenging. The 3-tier grading system for canine MCT is not useful, the 2-tier system has not been evaluated. Features that have been assessed for impact on prognosis include:

- Histologic subtype

  - Mastocytic - most common, further divided into compact and diffuse (pleomorphic/anaplastic) forms. Diffuse MCT tend to have a higher mitotic index and may be associated with more aggressive behaviour, however prediction of behaviour based on histologic subtype alone is difficult. A more recent study described a subset of well-differentiated tumours with prominent multinucleated cells which appeared to have aggressive behaviour.

  - Histiocytic - more likely to affect young cats, with Siamese being most affected in some studies. Generally benign, may spontaneously resolve.

- Proliferation indices: Higher mitotic index (per 10 hpf) is associated with outcome, though the appropriate

cutoff value is not known. Ki67 staining is also associated with prognosis.

- KIT labelling pattern appears to be associated with prognosis as for dogs
- Multiple lesions: Unlike in dogs, multiple cutaneous tumours are associated with splenic involvement and worse outcomes than solitary tumours in cats in some studies, though other studies found no effect on prognosis.
- HIstologic margins may not predict recurrence, though evaluation of this factor in a group of 'high-risk' MCT may be of more clinical significance.
- Recurrent tumours may be associated with a worse prognosis

I currently recommend evaluation of local lymph nodes in every cat and abdominal ultrasound and buffy coat assessment in cats with multiple or high mitotic index MCT. For tumours with aggressive growth, more aggressive surgery is warranted, but given the benign behaviour of the majority of feline cutaneous MCT surgical margins of 1-2 cm seem reasonable.

- Splenic MCT may be solitary or associated with cutaneous tumours. Involvement of the liver and bone marrow/peripheral blood is common. Splenectomy is the treatment of choice, even if liver involvement or mastocytemia is documented, as it is associated with the longest survival times (> 1 year median). The addition of chemotherapy should be considered for cats with evidence of more distant disease (liver involvement, persistent mastocytemia), though the best protocol, and whether or not it impacts outcome, is not known.
- Intestinal: Prognosis was previously thought to be poor because of extensive disease at diagnosis but a recent study suggests that the behaviour of these tumours is extremely variable. Based on this, my current recommendation is for surgical excision if possible. Chemotherapy could be considered for metastatic or unresectable tumours.

Systemic treatment in feline MCT:

Response rates of approximately 50-80% to CCNU and to toceranib are reported, and there are anecdotal/case reports of responses to other such as vinblastine, chlorambucil and imatinib. For cats with gross disease, it would seem reasonable to start with CCNU or toceranib and consider the other drugs as alternatives if a good response was not seen.

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1. Krick EL, Billings AP, Shofer FS, Watanabe S, Sorenmo KU Cytological lymph node evaluation in dogs with mast cell tumours: association with grade and survival Vet Comp Oncol 2009;7:130-138
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CURRENT TREATMENT OPTIONS FOR MAST CELL TUMORS IN DOGS AND CATS

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CURRENT TREATMENT OPTIONS FOR MAST CELL TUMOURS IN DOGS AND CATS

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Learning objectives: Understand the perioperative management, staging and surgical oncology principles for cutaneous MCT in dogs and cats, and the indications for adjunctive radiation therapy or chemotherapy in conjunction with surgery. Understand treatment options for non-resectable MCTs.

Canine cutaneous MCTs:

Diagnosis:

- Cytology is usually diagnostic, and finding of suspected high grade MCT on cytology is highly predictive for histologic high grade.
- In my practice, complete staging involves evaluation of local/draining lymph nodes, abdominal ultrasound with liver and spleen cytology, and bone marrow evaluation. Bone marrow is typically reserved for cases with CBC abnormalities because it is uncommon (< 5%).
- Lymph node (LN) evaluation is always recommended because low-grade MCT may metastasise. MCTs affecting the muzzle and perioral tissues may be more likely to metastasise. LN mapping is ideal to identify the 'sentinel' node. Diagnosis of metastasis from both cytology and histology can be challenging, but criteria have been recently published that may assist (1, 2). Good outcomes can be achieved with aggressive treatment in low grade, stage 2 MCT so identifying metastatic LN prior to surgery is important. Abdominal ultrasound and liver and spleen cytology is recommended in cases of known or suspected high grade tumours or when grade is unknown and local therapy is likely to be radical.
- Histology: The 2-tier histologic grading scheme (low/high grade) has better inter-pathologist agreement than the 3-tier scheme, however combining the two may give additional information. Mitotic index (per 10 high power fields) is i prognostic, though the most useful value is not fully determined. The significance of additional prognostic factors (Ki67, AgNORs, c-kit staining pattern and mutation) is, in my opinion, not fully determined in terms of effect on prognosis.

Treatment:

- Surgery: For MCT with distant (beyond LN) metastasis, removal of the primary tumour is not likely to impact prognosis, though it may improve quality of life in painful/ulcerated tumours. General oncologic principles of local en bloc resection with a margin of normal tissue around the tumour apply for cutaneous MCT. MCT should be minimally manipulated during preparation and surgery to decrease degranulation. Many MCT will have definitive excisional surgery on the basis of a cytology result. As a general rule, 1cm lateral margins are adequate for Grade I MCT, 2cm lateral margins for Grade 2 MCT and 3cm lateral margins for Grade 3 MCT. These have not been re-evaluated for the 2-tier grading scheme. If the grade is not known, then a minimum of 2cm lateral margins is recommended. For cytologically diagnosed MCT where definitive surgery is feasible based on size and location, biopsy for pre-operative grading is not usually required. Deep margins are more qualitative than quantitative. The deep margin should comprise fascia or muscle. Careful pre-operative palpation and imaging (Ultrasound, CT or MRI) are useful to determine degree of fixation to deeper structures in planning the deep margin. The excised tumour specimen should have the surgical margins inked to facilitate histological margin assessment. If the MCT is greater than 1cm in diameter, then the skin surface should be cut at 1cm intervals to allow appropriate formalin fixation of the tissues.

- Adjuvant local therapy: In cases of incomplete or narrow histological margins (i.e. risk of local recurrence), additional local therapy with revision surgery or radiation therapy is recommended. Electrochemotherapy is a newer modality which may also be an option. It is important to remember that the 'safe' histological margin (i.e. sufficient to prevent local recurrence) has not been determined for MCT and is influenced by grade. Many incompletely or narrowly excised low-grade MCT do not recur, however additional local therapy with re-excision or radiation therapy has been shown to improve survival and so should be considered in all cases (3).

- Adjuvant systemic therapy: Chemotherapy is recommended following surgery in high grade MCT, even without visible metastasis, as adjuvant chemotherapy appears to improve survival over surgery alone. The ideal protocol for adjuvant chemotherapy in high grade/ high risk MCT is not fully determined. Vinblastine and prednisolone appears to be most commonly used, but protocols including CCNU alone or alternating with vinblastine are also reported. The adjuvant use of TKIs such as toceranib (Palladia) or masitinib (Kinavet/Masivet) is not well studied, though one retrospective study reported improved outcome in dogs with high risk MCT receiving adjuvant vinblastine and prednisolone compared to those receiving masitinib (4). The other major challenge with adjuvant use of TKIs is determining appropriate duration of treatment. The use of adjuvant corticosteroids or anti-histamines has not been studied and is not generally recommended.

- Non-resectable primary tumours:
  - Radiation therapy (RT): Palliative or definitive RT may be considered. There are no large prospective studies, but it appears that the addition of prednisolone +/- toceranib may improve response (approximately 80% versus approximately 50%) and control duration over palliative RT alone.
  - Chemotherapy: Many different chemotherapy drugs alone and in combination (+/- corticosteroids) have been studied, including vinblastine, CCNU, TKIs, paclitaxel, cyclophosphamide and hydroxyurea, and metronomic chlorambucil. Response rates range from approximately 20% to 80% and response durations from weeks to many months. In general, combination therapies seem more effective. I usually offer three options for chemotherapy for non-resectable MCT: 1) 'traditional' chemotherapy with vinblastine and/or CCNU along with prednisolone 2) toceranib with prednisolone or 3) metronomic chlorambucil and prednisolone
  - Alternative local therapies: Electrochemotherapy results in response rates of 60-80% in the small studies published to date, and is generally well-tolerated. Intra-lesional triamcinolone may be an effective option in some cases, though responses are generally relatively short-lived.

- Treatment of metastatic MCT: In cases of MCTs with LN but no distant metastasis (stage 2), aggressive local therapy, often along with adjuvant chemotherapy is generally recommended. For low-grade stage 2 MCT aggressive local therapy (surgery +/- radiation therapy) may be sufficient for long term control. In distant metastasis, systemic treatment options as for non-resectable MCT are typically attempted, with some retrospective data suggesting that using vinblastine and CCNU is more effective than toceranib.

- Supportive care: Anti-histamines +/- antacids are recommended in all dogs with gross MCT disease

Feline MCTs

- Cutaneous: The majority are benign, but a subset are more aggressive and identifying those is challenging. The 3-tier grading system for canine MCT is not useful, the 2-tier system has not been evaluated. Features that have been assessed for impact on prognosis include:
  - Histologic subtype
  - Mastocytic - most common, further divided into compact and diffuse (pleomorphic/anaplastic) forms. Diffuse MCT tend to have a higher mitotic index and may be associated with more aggressive behaviour, however prediction of behaviour based on histologic subtype alone is difficult. A more recent study described a subset of well-differentiated tumours with prominent multinucleated cells which appeared to have aggressive behaviour.
  - Histiocytic - more likely to affect young cats, with Siamese being most affected in some studies. Generally benign, may spontaneously resolve.
  - Proliferation indices: Higher mitotic index (per

10 hpf) is associated with outcome, though the appropriate cutoff value is not known. Ki67 staining is also associated with prognosis.

- KIT labelling pattern appears to be associated with prognosis as for dogs
- Multiple lesions: Unlike in dogs, multiple cutaneous tumours are associated with splenic involvement and worse outcomes than solitary tumours in cats in some studies, though other studies found no effect on prognosis.
- HIstologic margins may not predict recurrence, though evaluation of this factor in a group of 'high-risk' MCT may be of more clinical significance.
- Recurrent tumours may be associated with a worse prognosis

I currently recommend evaluation of local lymph nodes in every cat and abdominal ultrasound and buffy coat assessment in cats with multiple or high mitotic index MCT. For tumours with aggressive growth, more aggressive surgery is warranted, but given the benign behaviour of the majority of feline cutaneous MCT surgical margins of 1-2 cm seem reasonable.

- Splenic MCT may be solitary or associated with cutaneous tumours. Involvement of the liver and bone marrow/peripheral blood is common. Splenectomy is the treatment of choice, even if liver involvement or mastocytemia is documented, as it is associated with the longest survival times (> 1 year median). The addition of chemotherapy should be considered for cats with evidence of more distant disease (liver involvement, persistent mastocytemia), though the best protocol, and whether or not it impacts outcome, is not known.
- Intestinal: Prognosis was previously thought to be poor because of extensive disease at diagnosis but a recent study suggests that the behaviour of these tumours is extremely variable. Based on this, my current recommendation is for surgical excision if possible. Chemotherapy could be considered for metastatic or unresectable tumours.

Systemic treatment in feline MCT:

Response rates of approximately 50-80% to CCNU and to toceranib are reported, and there are anecdotal/case reports of responses to other such as vinblastine, chlorambucil and imatinib. For cats with gross disease, it would seem reasonable to start with CCNU or toceranib and consider the other drugs as alternatives if a good response was not seen.

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GENETICS

WHAT PRACTITIONERS SHOULD KNOW ABOUT THE GENETICS OF HIP DYSPLASIA

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WHAT PRACTITIONERS SHOULD KNOW ABOUT THE GENETICS OF HIP DYSPLASIA

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Canine hip dysplasia is a complexly inherited disorder that is seen in wolves and across all purebred and mixed-breed dogs. It is the number one cause of arthritis in all dogs. We observe this disorder in clinical practice as; hip pain/stiffness, decreased range of motion, altered hind limb gait, and later arthritis.

The diagnosis of hip dysplasia is through the phenotypic evaluation of the hips. While palpation may reveal laxity or crepitus, the standard for diagnosis is through hip radiographs. The hip-extended ventrodorsal hip radiograph under general anesthesia is the accepted standard in most of the world. Radiographic evaluation includes; subluxation, shallow acetabulae, bony remodeling – especially at the dorsal rim of the acetabulum and femoral neck, and osteoarthritic changes. Different countries have different hip grading systems, including the OFA, BVA/KC, and FCI. PennHIP measures the difference between a hip compressed and hip distracted view and generates a distraction index (DI) representing passive laxity of the hip. The Dorsolateral Subluxation (DSL) test generates a score using a dorsoventral radiograph with the hips flexed and bearing weight at the stifles on the x-ray table. DSL is mostly used as a research tool and is not widely utilized in genetic screening.

The age of assessment for hip dysplasia is important, as it is not a congenital disorder and develops over time. PennHIP recommends evaluation for laxity beyond 4 months of age. The DSL test is recommended after 8 months of age. The BVA/KC and FCI require dogs to be 1 year of age. The OFA used to certify hips at 1 year of age, but found that 95% accuracy of diagnosis only occurred after 2 years of age. PennHIP describes an accurate early age diagnosis of hip dysplasia, but a study of early-age OFA preliminary ratings show a similar predictability. All of the different radiographic evaluations for hip dysplasia measure phenotypic aspects, and are all found to be correlated to each other.

Lifetime studies on the hip status of dogs show that maintaining a lean body condition produces overall better hip confirmation and reduced hip arthritis. Restricted calorie loads in "large-breed puppy foods" promote a more uniform growth rate where the boney and soft tissue components can develop and mature in unison. Excessive compaction on the hips (jumping and landing with full body weight on the hind limbs) should be avoided in immature dogs when the skeletal components of the hip are still cartilaginous and liable to deformation.

HEREDITY OF HIP DYSPLASIA

The evolutionary development of breeds has produced some with higher and lower liability for developing hip dysplasia, based on which dysplasia liability genes they have lost or retained over time. Different studies find that hip dysplasia is 20% to 40% heritable. This means that 20 to 40% of the variability of dysplastic development is due to genetic factors, and the rest of the variability is due to environmental factors. This classifies hip dysplasia as a moderately heritable disorder, comparable to other complexly inherited traits such as egg production in poultry, and milk production in cattle. Proper selection against hip dysplasia should result in a reduction of the frequency of affected dogs.

The inheritance of hip dysplasia is polygenic, meaning that the action of several genes must combine together to produce the disorder. The specific combination of genes that produce liability to dysplastic development will vary between breeds, familial clusters, and between individual dogs. Environmental variables that can alter the expression of the disorder can include dietary load and degree of activity/mechanical stress.

Genetic studies into hip dysplasia have broken down the phenotypic liability into several components that appear to be inherited separately. Some of these variables include; joint laxity, age of ossification, depth of the acetabulae and liability for osteoarthritis.

Breed differences in prevalence of hip dysplasia often have to do with breed defining characteristics. Breeds that were established on a racing phenotype had extensive selective pressure for good hip conformation. Those who did not excel were not used for breeding. Other breed differences have to do with conformational morphology. Lighter-boned tight-muscled breeds have a lower prevalence of HD compared to heavier-boned course-muscled breeds.

Chondrodystrophic breeds and dogs have in general poorer radiographic hip joint conformation. However, their lower hip scores do not necessarily correlate to increased clinical disease. In a study of Pembroke Welsh Corgis, all dogs showed radiographic signs of hip dysplasia, but this was not correlated to their susceptibility to develop later osteoarthritis.

Studies on the genetic control of polygenically inherited traits show that selection based on phenotypic measurements of individuals show less improvement when compared with selection based on familial data. OFA data show that hip conformation scores are directly correlated to the scores of the parents, grandparents, and their siblings. **Combined parent hip scores are linearly correlated to the production of offspring with hip dysplasia - showing its inheritance as an additive (quantitative) trait.**

Familial data can also be computed as estimated breeding values (EBVs), based on the phenotype of the parents, siblings, siblings of parents, offspring, and other relatives. By utilizing phenotypical depth and breadth of pedigree, EBVs utilize information that can more accurately reflect the cumulative genetic influences passed down to the individual dog.

An issue with the accuracy of calculating EBVs involves dogs with missing phenotypes – as most breeds have less than 10% of breeding dogs or their siblings evaluated. To provide the most power, EBVs require data on all normal and abnormal sibs within litters. Without this, the accuracy and precision of EBVs is low. In an applied setting, dogs with high EBVs may also become popular sires thus putting pressures on gene pools that can affect genetic diversity. EBVs for hip dysplasia have been developed for several breeds in the UK by Dr. Lewis at the Kennel Club, in the US by Dr. Todhunter at Cornell University, and by the Australian Kennel Club. EBVs should include power estimates based on pedigree completeness.

Genomic breeding values (GBVs) are based on DNA markers that segregate with hip dysplasia in experimental populations. These markers may or may not be correlated to specifically identified dysplasia liability genes. Todhunter's group at Cornell and Dr. Distl's group in Hanover, Germany are working on GBVs. At this time breed-specific genetic marker panels are specific to the populations being studied, but do not accurately predict phenotypic liability in larger populations of the same breed or in different breed populations. Therefore, current commercial hip dysplasia liability DNA marker panels should be viewed with caution as they may not correlate to other populations within the same breed, or other breeds.

GBVs have greater promise in producing improvement with hip dysplasia as they avoid the issues of missing phenotypic information required for EBVs, as well as phenotypic variation caused by environmental influence. However the development of accurate GBVs requires full extended pedigree phenotypic information. While EBVs and GBVs appear to hold the most promise for improved genetic selection against hip dysplasia, their specific clinical use and validation are a work in progress.

GENETIC SELECTION FOR NORMAL HIPS

Studies on the response to selection based on individual phenotypes show mild improvement with hip dysplasia. It is known that the greater the selective pressure (percentage of the population eliminated from breeding due to phenotypic score), the greater the improvement. However, the greater the percentage of the population eliminated from breeding, the greater the loss of genetic diversity of the population. Therefore, selection must be combined with consideration of the breed gene pools.

The phenotypic evaluation of individual breeding dogs should be uniformly applied, and based on a properly executed hip radiograph on an anesthetized or deeply sedated dog. This allows for the best evaluation of both boney conformation and joint laxity.

Selection of breeding stock should be based on familial data – depth and breadth of hip normalcy – not just the phenotype of the individual dog. **The dog's own hip rating represents its phenotype, but the relative's hip ratings are more representative of the dog's genotype.** For example: The individual dog's OFA web page, and associated vertical pedigree as demonstrated on the OFA website (ofa.org) provides a good representation of the expectation of hip quality that can be passed on from the individual. A dog with excellent-rated hips but with a preponderance of fair-rated relatives would be expected to produce more like its relatives than itself.

Breaking down the hip phenotype for individual dogs allows the breeder to focus on aspects that need improvement in the next generation. If a quality dog shows some subluxation or laxity, it should be bred to a dog with tight hips. If a quality dog shows a slightly shallow acetabulum, it should be bred to a dog with deep acetabulae.

Selection based on the best available phenotypic imaging and incorporating familial breadth and depth of pedigree data should improve the hip and elbow status of individual dogs and thus their breeds.

References available upon request.

WSV18-0277

WATER QUALITY ASSESSMENT IN AQUATIC VET MEDICINE

With any animal, environmental conditions can affect their overall health, but with aquatic animals such as fish, proper water quality is an important part of a successful aquarium. Without clean water, the fish will be stressed and more susceptible to diseases and parasites. This lecture will provide veterinarians with information regarding how to test aquarium water and what the various water chemistry characteristics mean for the health of the fish. Correcting water quality problems is also included in the discussion.

Water quality can be measured with test kits available through pet stores or pond supply companies, or from many aquaculture suppliers. The simplest tests are small plastic strips with chemical pads attached that are dipped into the water to be tested. The pads change color which, when compared to a color chart, indicates the level of that substance in the water. These are fast, easy to use, inexpensive, and relatively accurate (they indicate a range rather than a precise measurement). Dry tablet tests are also available where a small tablet is dissolved into a test tube containing the water sample. Its color is then compared to a chart to determine the results. Some test kits have liquids that are mixed with the water to produce the color reactions. More expensive test kits use a spectrophotometer to electronically compare colors and these give more accurate results. Effective electronic meters are also available.

Ammonia:

Ammonia (NH_3) in the water reduces the ability of the fish to excrete nitrogenous wastes from their blood through the gills. As ammonia increases in the water, so do the waste products increase in the fish's blood, causing toxicity, gill damage, and death. Ammonia is mostly converted to nontoxic ammonium (NH_4^+) at a pH level below 6.5, but above 6.5 ammonia can become toxic very quickly if allowed to accumulate. The higher the pH and temperature of the water, the more toxic ammonia becomes. The ammonia in the aquarium water is broken down by aerobic nitrifying bacteria into nitrite and then into nitrate. Properly operating biological filtration systems (after they have been cycled) should keep ammonia levels at 0.0 mg/L in the aquarium water.

In the event of a filtration system problem that creates high ammonia levels (>0.25 mg/L), Ammonia Neutralizing products can be used to bind the ammonia in a nontoxic form until water changes can be used to bring the ammonia level down. Failure to eliminate the ammonia through water changes will result in elevated nitrite levels a few days later.

Note: Some municipalities add chlorine or chloramine to the tap water to make the water safe for human consumption. Contact the local water service if unsure of the chemicals being used. Aquarists in areas that have chloramine added to the tap water need to use an ammonia remover as well as a chlorine remover to make the tap water safe for use in their aquarium.

Nitrite:

Nitrite (NO_2-) is produced by the aerobic bacterial nitrification of ammonia. It should also be maintained at a level of 0.0 mg/L. Nitrite is absorbed through the fish's gills and causes methemoglobinemia, which reduces the ability of the fish's blood to carry oxygen. Salt in the water at 0.1-0.3% salinity will block the absorption of nitrite by the fish's gills. This is one of the reasons some aquarists add salt to aquariums with water changes. Remove any nitrite from the system by performing a partial water change.

Nitrate:

Nitrate (NO_3-) is produced by the aerobic bacterial nitrification of nitrite. While high nitrate levels are dangerous to saltwater fish and invertebrates, freshwater fish are very tolerant of high nitrate levels. Most freshwater fish can tolerate levels of 200 mg/L for short periods of time without significant problems. If nitrate levels exceed 20 mg/L, water changes can be used to lower the concentration. High levels of nitrate also promote algae growth.

pH:

The pH is the "potential of Hydrogen," which is the measure of the hydrogen ions in a water (the acid/base balance). Most natural freshwater systems range from slightly less than 6.0 to about 8.5 in pH value. Most freshwater fish are highly adaptable to gradual changes within this range of water conditions. Rapid changes in pH are detrimental to fish, and it is very important that the aquarium has a stable pH. The stability of the pH is related to water Alkalinity (buffering capacity).

Alkalinity:

Alkalinity is a measurement of the negative ions (e.g., Hydroxide, Carbonate, Bicarbonate) in the water that buffer against pH shifts. Ideal alkalinity is in the 100-250 mg/L range for most freshwater fish species. Biological filtration in the aquarium uses carbonates, so over time the alkalinity level is reduced. As the alkalinity falls, the water in the aquarium may experience sudden, and deadly, pH shifts. To prevent this, increase the buffering capacity of the water to stabilize the pH. This can be done by adding carbonates to the water, or doing water changes with water that contains higher alkalinity.

Hardness:

Hardness is the measurement of metallic positive ions (e.g., Calcium, Magnesium) in the water. Water with high hardness usually also has a high alkalinity and pH. Hardness in aquatic systems is best at 100-250 mg/L, but some fish such as discus prefer softer water. African cichlids prefer hard water. Most fish will adapt to existing hardness as long as it is not too extreme of a change.

Chlorine and Chloramine:

Chlorine and chloramine are used by water municipalities to make the water supply safe for human consumption. These compounds are extremely toxic to aquatic organisms and no amount can be tolerated in an aquarium. In cases of rapid fish loss in an aquarium after a water change, chlorine should be the first thing checked. Be certain to test for chlorine prior to treating the system with a dechlorinator. This will enable you to determine whether chlorine was the issue. There should never be any chlorine detectable in aquarium water! Add sodium thiosulfate to the system whenever chlorine is detected.

Temperature:

Freshwater tropical fish have a preferred optimum temperature range of 22-26 degrees Celsius (72-78 degrees Fahrenheit), but may be able to survive at temperatures about 5 degrees below or above this range. Gradual changes in water temperature within a fish's optimum range seldom cause health problems. Ideally, water temperature fluctuations should be no more than 3°C change per day. Temperature shock can occur with rapid changes, especially from warmer water to cooler water. Increasing the water temperature will lower the saturation point of dissolved oxygen (warmer water holds less oxygen than cooler water). It will also increase the toxicity of dissolved substances such as ammonia, chlorine, and heavy metals.

Summary:

Water testing is one of the most important aspects of aquarium maintenance. It is an important key in determining how well the filters are functioning. Keep a log of your water test results to monitor water quality changes over time. Always check the water quality when determining what may have caused fish loss. Water testing is not something to be taken lightly.

The following chart has general guidelines for the safe ranges and optimal levels of these parameters in freshwater aquaria and koi ponds.

Freshwater Parameter	Acceptable Test Results:	Optimum Range:
Temperature	68-84°F / 20-29°C	73-79°F / 23-26°C
pH (acid/base balance)	6.5-8.5 [if stable]	7.2-7.8
Ammonia (NH3)	0-0.2 mg/L	0-0.02 mg/L
Nitrite (NO2-)	0-0.2 mg/L	0-0.1 mg/L
Nitrate (NO3-)	0-80 mg/L	0-20 mg/L
Chlorine	0-0.1 mg/L	0 mg/L
Alkalinity	50-300 mg/L	75-150 mg/L
Hardness	50-300 mg/L	50-100 mg/L
Dissolved Oxygen	6-9 mg/L	8-9 mg/L
Carbon Dioxide	1-10 mg/L	2-5 mg/L
Salinity (Freshwater)	0-0.5%	0-0.2%

43RD WORLD SMALL ANIMAL VETERINARY ASSOCIATION CONGRESS AND 9TH FASAVA CONGRESS

Periodic partial water changes (25% of total volume) using dechlorinated tap water will keep aquarium water values normal. The frequency of changes will depend on the water test results, but normally once per week in new aquariums and 1-2 times per month in established aquariums is sufficient. Examples of incidents requiring increased water changes include toxin contamination, abnormal pH or alkalinity values, high ammonia, nitrite or nitrate levels, or over-medication. Test the water after performing a partial water change; if necessary, repeat partial water change to correct water quality parameters.

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CASE ROUNDS

SOLVING LIVER AND PANCREAS PROBLEMS WITH AN INTERNIST AND A RADIOLOGIS

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TBA

WSV18-0279

SVA INTEGRATIVE MEDICINE & ACUPUNCTURE

VETERINARY ACUPUNCTURE: FROM SCIENTIFIC EVIDENCE TO CLINIC APPLICATION

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A. Introduction

Acupuncture is a medical technique that has been practiced for over 3000 years in China, and is part of traditional Chinese medicine (TCM) and traditional Chinese veterinary medicine (TCVM). The use of acupuncture is becoming more common in veterinary medicine, and it can play a role in the management of acute, inflammatory, chronic pain, nerve damage, and nausea and vomiting, as well as many other internal medicine problems.

In 1997, the United States National Institutes of Health (NIH) developed a consensus statement about acupuncture and its efficacy. NIH said that there was compelling evidence that acupuncture was usefully in the management of osteoarthritis and musculoskeletal pain. It can be helpful in treating many gastrointestinal problems, including inflammatory bowel disease, diarrhea, ulcerative colitis, peptic ulcers, dyspepsia, abdominal pain, nausea and vomiting. Acupuncture can help with management of pulmonary disease including colds and asthma. The immunomodulation of acupuncture can reduce inflammation, elevate WBC, and increase interleukin-2 production. Finally, acupuncture can help in treating reproductive disorders, decreasing uterine bleeding and regulating ovulation. In additions, the World Health Organization (WHO) also recognizes the use of Acupuncture in the treatment of a wide range of common illnesses. While most of these studies reviewed the effectiveness of acupuncture in human patients, much of the data was based upon animal experimentation. Moreover, the conditions for which NIH and WHO thinks acupuncture can be effective are the same conditions which veterinarians treat with acupuncture.

From a modern prospective, acupuncture represents a form of nerve stimulation and neuromodulation of the body. As such, to know acupuncture is to know the nervous system. Certainly, we know that for acupuncture to work, it requires an intact nervous system and acupuncture is not effective if the nervous system is damaged beyond repair. Recently, using functional MRI (fMRI), the basic tenets of acupuncture have been proven. Those are that acupuncture is based upon the point selected, the method of stimulation and the duration of stimulation. Stimulation of various acupuncture points result is specific special changes in the central nervous system (CNS). The change is mild when only acupuncture needles are used and become more pronounced if electrical acupuncture is added. While the change initially is more limited, over time, the entire neural axis becomes involved.

B. Acupuncture

Acupuncture may be defined as the stimulation of a specific point on the body, referred to as an "acupoint". Physiological changes in response to acupuncture point stimulation is the basis of clinical treatment. Some of these changes include release of endogenous opioids, immune system stimulation, and blood pressure regulation. Stimulation of an acupoint causes activation of $A\alpha$ and $A\beta$ nerve fibers to conduct electrical signals through the spinothalamic tract to the hypothamalus and cause release of β-endorphins. Acupuncture also causes activation of the descending pain inhibitory pathway which activates the periaqueductal gray matter to release more β-endorphins and the nucleus raphe magnus to release serotonins. Pain is blocked with the release of these endogenous opioids and neurotransmitters. Acupuncture can also activate T-cell lymphocytes and increase the number of white blood cells for the treatment of immuno-deficiency. Acupoint stimulation also affects the blood pressure receptors and can influence blood pressure. It can be used to increase or decrease blood pressure.

C. Acupuncture Points (Acupoints)

Most acupoints are located along the nervous system and have been identified to be one of four basic types of points (Gunn Cc. 1997):

1. Type I acupoints, which make up 67% of all acupoints, are considered motor points. The motor point is the point in a muscle which, when electrical stimulation is applied, will produce a maximal contraction with minimal intensity of stimulation. Motor points are located in areas where nerves enter muscles. For instance, SI-9 is located at the junction of the deltoid muscle and triceps brachii and is supplied by axillary and radial nerves.

2. Type II points are located on the superficial nerves in the sagittal plane on the dorsal and ventral midlines. For instance, Bai-hui lies in the depression between the spinous processes of the seventh lumbar and the first sacral vertebrae on the dorsal midline and is supplied by the dorsal branch of the last lumbar nerve.

3. Type III points are located at high density loci of superficial nerves and nerve plexuses. For example, GB-34 is located at the point where the common peroneal nerve divides into the deep and superficial branches cranial and distal to the head of the fibula.

4. Type IV points are located at the muscle-tendon

junctions where the Golgi tendon organs are located. For example, BL-57 is located at the junction between the gastrocnemius muscle and the calcanean tendon.

The anatomic nature of acupuncture points represents neural vascular bundles, containing
- Abundant of free nerve endings
- Small arterioles, veins
- Lymphatics
- Increased concentration of mast cells
- Contain higher conductance, lower impedance, and higher capacitance than adjacent tissues
- Close proximity to somatic afferent terminals, suggesting the likelihood of interaction and modulation of afferent signals by needle placement
- Most are also found in palpable depressions

D. Pain Control Mechanism of Acupuncture

Many of the systematic reviews supporting acupuncture efficacy are for painful conditions. There are a few mechanisms by which acupuncture affects analgesia. These mechanisms have been extensively investigated and are quite well understood, and are enhanced with the passage of small amounts of electrical current through the acupuncture needle (electroacupuncture). These mechanisms are (Melzack R. 1965):

a) Local Effects
- A form of counter-irritation effect
- Insertion of needle in an acupoint results in local tissue damage (called microtrauma), which activates Hageman's tissue factor XII. This in turn results in the activation of local coagulation cascade and the complement cascade, leading to the production of plasminogen, protein kinins, and prostaglandins.
- The microtrauma also causes mast cell degranulation, which releases histamine, heparin, proteases and bradykinin.
- These local reactions ultimately result in increase blood flow to the area and local immune responsiveness, that help relieve pain and reduce inflammation and edema.
- Connective tissues are stretched when needles are placed in acupoints rather, which relax the muscles and tissues in the local area.

b) Spinal Cord Effects
- When nerve impulses travel up the sensory nerves, it activates three centers (spinal cord, midbrain, and hypothalamus/pituitary) to produce analgesic effects.
- At the spinal site, enkephalin and dynorphin are used to block incoming pain messages.
- Electroacupuncture at 2 Hz and 100 Hz cause the release of enkephalin and dynorphin in the spinal cord, respectively.

c) Brainstem Effects
- The neuronal network that serves acupuncture analgesia is thought to activate structures of the descending inhibitory pathways and deactivate limbic structures within the ascending nociceptive pathway.
- The midbrain uses endorphin to activate periaqueductal gray, and use serotonin and enkephalin to activate the raphe descending system, which then inhibit pain transmission within spinal cord.
- The pituitary releases beta-endorphin into the blood and cerebral spinal fluid to cause analgesia at a distance.
- The hypothalamus uses beta-endorphin to activate the descending analgesia system.

d) Gate Theory
- Acupuncture activates the Aβfibers (larger and fast conducting nerve fibers than C-fiber). These fibers lead to inhibition (pre-synaptic) of information carried by the C fibers (pain), so that the C fibers could no longer travel to the CNS to cause pain to the body

e) Acupuncture on fMRI Function
- Acupoints that have analgesic properties associated with them tend to activate specific pain-associated brainstem regions. Non-analgesic acupuncture points do not activate these regions; rather they activate other regions of the brain. (Chiu JH. 2003)

f) Frequency of Electroacupuncture
- Low frequency (2-40 Hz) predominantly stimulates A-delta fibers and induces the release of beta-endorphin, met-enkephalin and endomorphin from the brainstem predominately.
- High frequency (100 Hz) predominantly stimulates C fibers and selectively increases the release of dynorphin predominantly.
- Higher frequencies (200Hz), predominantly stimulates serotonergic fibers and releases serotonin and epinephrine. (Fwk S.1994)
- A combination of the two frequencies (2 and 100 Hz) produces a simultaneous release of all four opioid peptides, resulting in a maximal therapeutic effect. This finding has been verified in clinical studies in patients with various kinds of chronic pain including low back pain and diabetic neuropathic pain. (Han JS. 2004)
- One review showed that electroacupuncture activates the nervous system differently in health than in pain conditions, alleviates both sensory and affective inflammatory pain, and inhibits inflammatory and neuropathic pain more effectively at 2–10 Hz than at 100 Hz. (Zhang R. 2014)

F. Conclusion

In the past 30 years there has been increasing evidence to support the use of acupuncture as a therapeutic modality in veterinary medicine. There has been extensive research proving its analgesic effects, therefore it is the primary clinical indication for its use in both human medicine and veterinary medicine. Due to the intrinsic nature of the anatomical structures associated with the location of acupuncture points there are numerous systemic, local, and endocrine effects. For that reason, there are a variety of valuable clinical indications from pain management to cognition improvement and others. As more research develops in the upcoming years it is likely that acupuncture will become mainstream and a treatment modality taught in veterinary schools across the country.

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WSV18-0278

TIPS FROM THE EXPERTS FOR THE APPROACH OF...

DERMATOLOGIC EXAM

S. Quek[1]

The skill to maximize a dermatologic exam would seem very basic, but yet often forgotten. We are always so eager to dive into examining the patient that we forget that a good part of a good dermatologic exam is actually the history of the patient.

A dermatologic exam would constitute the entire dermatology consult. How successful a dermatologic consult would be dependent on how effective the vet identifies the skin condition, prescribes the treatment, communicates that to the owner and if the owner is going to follow up with the treatment.

A good dermatologic exam starts off with a detailed history of the patient. Asking the right questions is very important to obtain correct information of the patient's skin condition from the owner. Questions such as:

- Is the patient itchy?
- If so, where is it itchy?
- When is it itchy? Seasonal or non seasonal?
- Age of onset?
- Acute or chronic?
- Previous treatment and response?
- Previous diet and response?

This information can help categorize the skin condition. Whether it is allergic skin disease, ectoparasite, infectious, hormonal related or autoimmune.

The next step to a dermatologic exam is examining the patient. Look at the distribution of lesions. Most allergic skin disease in dogs have a predictable distribution pattern. Lesions affecting periocular, perioral, ears, paws, cubital areas, axilla, inguinal and perianal areas would point towards a possible food adverse reaction or atopic dermatitis. Bilateral flank, lateral thighs and tail base would indicate a possible flea bite allergy. Ear margin dermatitis and crusting with positive pinna-pedal reflex would be suspicious of sarcoptic mange infestation. Nasal planum, mucocutaneous junction ulceration and crusts would indicate an autoimmune disease and the list goes on. It is important to be familiar with the distribution patterns of different skin diseases.

Once a differential diagnosis of skin conditions has been made, the next step is to take good samples. Missing a skin diagnosis occurs more often from not taking correct samples than actually not knowing about the disease. Hence taking good samples is important to confirming the diagnosis. Sample taking also helps determine what treatment to administer. If antibiotics or anti-fungals need to be prescribed. Vets should be familiar with taking proper samples such as deep skin scrapes, fungal

cultures. If vet nurses are tasked to take samples, it is important to ensure that they are well trained to perform those tasks.

No dermatologic consult is complete without providing the client with a treatment plan. If you fail to plan, you will plan to fail. The owner is the one who has to carry out the treatment plan. Whether is it administering medication or performing an elimination diet trial. It is hence important that the clients are clear on the treatment and follow up. Provide a clear schedule so that the owner understands what needs to be done and when they need to come back for a follow up. No diet trial will be successful if the owner does not come back for a rechallenge. Arrange for nurses to call clients and remind them of their follow up.

KEY LEARNING OBJECTIVES

- To remember to take a detailed dermatologic history of the patient.

- To be familiar with distribution pattern of different skin diseases.

- To be competent is taking skin samples and to ensure that nursing staff are also well trained to obtain samples.

- Provide the client with a treatment plan and follow up.

WSV18-0011

FELINE FOCUS (SIMULTANEOUS TRANSLATION INTO MANDARIN CHINESE)

RESPECTFUL CAT HANDLING VS. CAT WRANGLING: FROM THE CAT'S POINT OF VIEW

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PART 1: RESPECTFUL HANDLING: FROM THE CAT'S POINT OF VIEW

In many clinics, some veterinarians and other team members do not enjoy working with cats because they believe that cats are unpredictable and feel anxious about getting hurt. By understanding why cats feel that they need to defend themselves, by learning to identify the warning cues, managing the interactions in a positive manner, and making relatively minor changes to what the cat is exposed to, this fear can be reduced.

The basis for working cooperatively with cats is being empathic to their nature and behaviors and trying to imagine what their experience is like. Cats are a species with a social structure different from ours. We need to look at cats differently, slow down and adjust our interactions. Minor modifications to the physical facility help reduce the strangeness and threats that cats experience in the veterinary clinic.

The goal of these two presentations is to look at how to change the experience for cats thereby removing some of the obstacles to routine feline veterinary care. This is beneficial for cats and their human companions and will also result in clinic growth.

WHY CATS RESPOND THE WAY THEY DO

In the wild, the number of feral cats living together depends on the availability of resources. These are food, water, privacy and safety, toileting areas, and availability of sexual partners. Mice and small birds are single portions; they are not large enough to be shared. After weaning, cats are responsible for feeding themselves. The resource density determines the number of cats living in a given area. In order to reduce conflict and the potential for physical harm associated with fighting, cats have developed an impressive repertoire of signals to maintain distance and protect resources within their territory. This results in little competition and a social structure that does not require sharing or taking turns. Stress is minimal unless resources become scarce. Aggressive communication signals developed in order to keep distance between individuals and to prevent contact with outsiders. Cats need to avoid physical injury in order to be able to hunt and protect themself. When resources are plentiful, a colony will develop consisting of related female cats with their young, who they jointly defend and nurse. Males are relegated to the periphery and vie for breeding privileges; only one mature tom usually lives with the group.

Many of the behaviours cats show in a clinic situation stem from the fact that while they are predators of mice and small birds, they are prey relative to almost all other larger animals, including larger birds. When they feel threatened, they rely on "fight or flight" and will try to escape situations that they view as dangerous. When they can't flee, they fight (self-defense) or freeze. From the perspective of a cat, humans are, (and what we do is), dangerous. As a result, we see frightened and defensive cats every day. Cats try to avoid physical confrontation through by using intimidating sounds and postures. This small creature feels more threatened than we do; it is important to refrain from becoming frightened ourselves.

Reading and understanding the cues and signals that cats use is important to detecting incipient fear. This allows us to respond respectfully as well as redirect the progression of an emotion and reshape experiences. We can learn to avoid using signals that are hostile (e.g., scruffing, making shushing/hissing sounds, looking into their faces) when we know how cats communicate.

FELINE SIGNALING: READING THEIR CUES

Tactile Sense

Touch is very important to cats. They rub against each other (allorubbing), against us, and against inanimate objects. Whether a full-body rub or rubbing a flank, tail, cheek or other body part, rubbing is believed to be an affiliative behavior seen between members of the same social group, feline or human. Rubbing is not only tactile, but is also a means of depositing the colony (family) scent. Cats often rub against us; unfortunately, we often misinterpret it as a request to be fed.

Allogrooming (mutual grooming) may precede a playful attack, follow a stressful interaction, and appear to be conciliatory or may simply be grooming. Kneading and treading occurs in adults either as a kitten-regressive behavior or as a component of sexual interaction.

The neck bite/scruffing is used by cats in three contexts: for transportation of young kittens, for restraint during copulation, and for dominance in a fight. Our use of scruffing fits most closely with the last and does not promote shaping safe, respectful cooperation. (See AAFP and ISFM Feline Friendly Handling Guidelines.)

Olfactory Cues

The role of smell and scent in feline communication is something we human beings are ill-equipped to appreciate. It has been estimated that the size of the olfactory epithelium in cats can be up to 20 cm2, whereas humans have only 2 to 4 cm2 of olfactory epithelium. While olfactory signals may be left by several methods, the one that is most problematic for people is urine spraying. This is a potent and important method of communication that we fail to appreciate. Other forms of olfactory messaging are cheek marking an object or

individual, scratching to leave scent from glands below the footpads, and midden, (i.e., leaving a deposit of feces uncovered in a strategic place). All of these have several advantages over visual cues. The message persists over time and in the absence of the sender, allowing for remote communication without the potential for conflict that direct interaction risks. This is especially useful at night and in areas with poor visibility. These signals help cats spread out over space as well as time-share territory. The disadvantage of this form of communication is that the sender cannot change the message once it has been deposited; it cannot be altered or removed and no adjustments can be made in response to the recipient's reaction. So, urine marking in the home is an attempt to signal to the other cats when "I was 'here'" and to establish a routine so that the cats can keep a distance by time-sharing the same space without needing to come into conflict. Every time we remove the urine, we interfere with this communication!

We have less well-developed olfactory sense; we fail to "read" the signals a patient may be giving us and are unable to fathom the overwhelming olfactory messages from previous patients and substances used in the hospital that the clinic experience presents to cats.

Visual Cues: Body Language (Posture, Face, Tail)

Body language and facial expression are extremely effective at maintaining or increasing distance between individuals potentially competing for resources. This requires having an unobstructed view, adequate ambient light, and, unlike olfactory cues, that the two individuals are in the same space at the same time. Body posture cues the big picture of emotional state but facial expression (eyes, ears, whiskers, mouth, visibility of teeth) provides the finer details and changes more rapidly. In a clinic setting, for us to appreciate the mental/emotional state of an individual, to avoid provoking them and getting hurt, it is extremely important to watch and interpret facial changes.

As a species that generally leads a solitary existence, survival depends on speed, stealth, self-reliance, and outsmarting others. As a consequence, cats may "bluff". When they act aggressively, they are generally hiding fear; "stoicism" hides vulnerability; subtle changes in behavior mask pain or significant illness. Body postures communicate confidence and physical prowess that may not be present. Keeping a threat at a distance may eliminate the need for a physical confrontation. The arched back "Halloween cat" typifies this façade of confidence. Making oneself smaller, on the other hand, to minimize threat and evade attention is portrayed by a crouch and withdrawal. In these postures, the weight remains on all four paws so that flight or chase remains possible. A cat feeling less fearful does not need to be on his or her feet. However, an extremely fearful threatened cat will roll exposing his or her abdomen

with all four feet ready for self-defense. This cat may be screaming while showing all of its weapons (nails and teeth).

Cats have extremely mobile ears. When the ears are forward, a cat is listening and is generally relaxed or alert but not emotionally aroused. Turned laterally, flat "airplane ears" indicate that the cat is more fearful or feels threatened. When ears are back and tight to the head, the cat is feeling very threatened and frightened. This cat will have a partially or fully open mouth and be hissing, spitting, yowling, or screaming. Cats will protect themselves if we fail to reduce the level of perceived threat. Ears turned back but erect indicates the most reactive and aggressive state. In this case, the mouth will be closed and the cat will be emitting a low growl with or without swallowing. This is the cat to be apprehensive of.

Figure 1: *Interpreting a cat's body posture.*
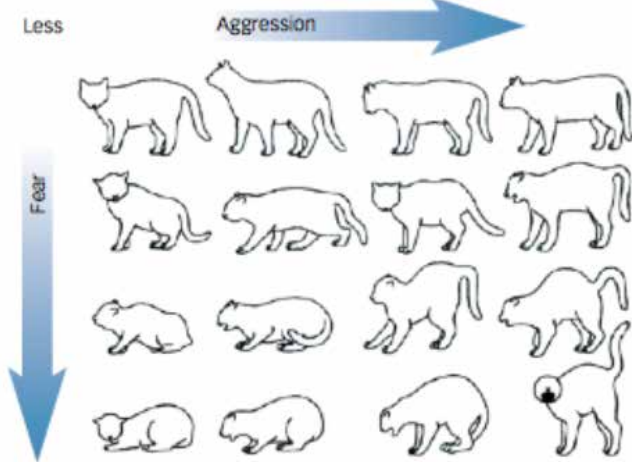
Figure 2: *Interpreting a cat's ear position and facial expression.*
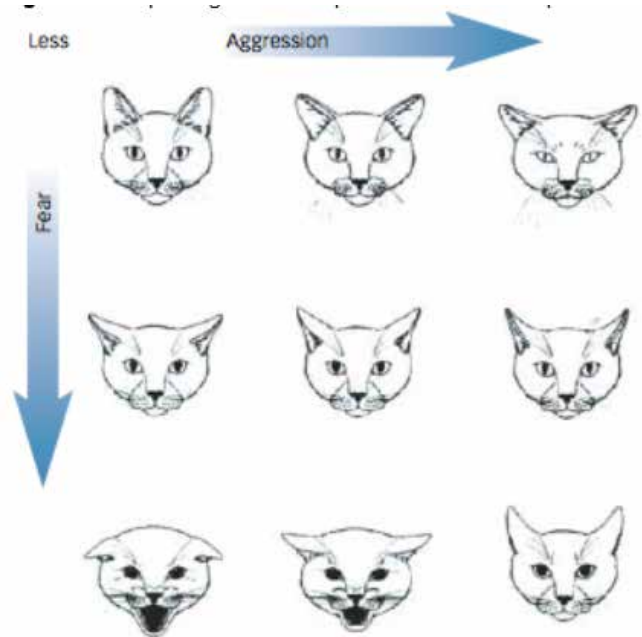
Figures from Rodan I: *Understanding the cat and feline-friendly handling. In Little SE, editor: The Cat: Clinical Medicine and Management, St. Louis, 2011, Elsevier, p5.*

Vocalization

This form of communication requires the direct presence of the recipient. It has the benefit of being easy to adapt from moment to moment. As with other signals, cats have a well-developed repertoire of sounds to convey a need or wish to increase the distance between individuals. The sounds made for encouraging socialization are a trill/chirrup, purr, puffing, prusten, chatter, miaow, and sexual calling. The cat that is open-mouth screaming is highly aroused but is probably less aggressive than the cat that is close-mouthed growl/wah-wah/mowling.

Cats use a combination of these different signals in any situation. We need to learn to look for all of them and interpret them together.

PART 2: RESPECTFUL HANDLING: PUTTING PURRSPECTIVE INTO YOUR PRACTICE

Making the clinic environment more "feline friendly" requires imagining how a cat perceives it. The exercise becomes one of identifying potential threats and removing or reducing their significance.

Reducing perceived threats in the hospital setting

It is important to reduce exposure to true predators (dogs, people, other cats) and to other perceived threats. Visual barriers in the seating/waiting area help to prevent cats from seeing dogs. Covering the carriers with a towel will also help so that cats don't see each other. Using chairs or ledges, keep kennels off the floor. If possible, have a separate cat-only waiting area. Reserve at least one examination room only for cats in order to reduce the smells of predators and to be able to furnish it with

necessary items for examining cats as well as to strive to achieve cat comfort.

Looking over our clinic/hospital environment, what can we do to reduce the stress and threat level of the physical and social environment? What things or events assault the five senses of a cat? How can we make positive changes to these? Table 1 shows a chart that can be completed by the clinic team. For example: Scary smells include alcohol, disinfectants, odours of other carried animals; this can be remediated by wiping the area to which alcohol had sparingly been applied with a damp cloth and using venipuncture sites far from the nose (medial saphenous) when possible. Disinfectant should be allowed to evaporate before a cat is placed/replaced in a kennel. Carry and examine all patients in their own, fresh towel rather than have their smells embed themselves in your clothing.

Table 1. Chart for assessing perceived threats to cats in hospital setting

Sense	Threat	Reduce threat by
Smell		
Hearing		
Sight		
Taste		
Touch		

Handling (examination, hospitalization, diagnostics, and treatments)

The goal is to handle our patients respectfully and provide an appeasing environment to build positive, long-term relationships. This is achieved by reducing threat and, thus, the cat's need to react defensively. Avoid doing things in a way that use threatening feline body language or tone. The aggressive cat is upright, stiff-legged, and large; sit down to examine cats.

Never stare a frightened cat in the face: examine cats from behind and, other than for ophthalmic evaluation, avoid direct frontal facial viewing. Using a sideways glance with hooded eyelids indicates a desire to cooperate. A slow blink is a reassuring signal to a cat similar to a human smile.

The aggressive cat growls and uses low tones; use light, upper register tones, perhaps chirruping as cats do when they are relaxed with conspecifics. Shushing a cat to try to calm her as we might a child is the equivalent to hissing at her. Short repetitive sounds should be avoided, since these may resemble spitting rhythms. Purrs, chuffing, trills, and chirrups are welcoming sounds.

When cats feel secure and safe, even just able to hide their faces in an elbow or a towel, they allow most procedures. Try to keep all four of their paws on the floor and avoid changing their body position as much as possible. A comprehensive examination, blood and urine collection, body temperature and blood pressure evaluation can all be done without changing the cat's position. Examine her in the base of her own carrier if the lid can be removed. Don't hang a cat's forelimbs over the edge of a table for jugular venipuncture. For the frightened individual, additional lack of support under the paws is not reassuring.

Reaching into a kennel to pick up a patient blocks the light; to the cat you appear as a looming, frightening stranger (smells, sounds, visual input). Instead, approach the opening of a kennel from the side so that some light still enters. Do not block every chance for escape; if the possibility to have some control over her environment and situation exists, she will be much more cooperative. Because cats rely on flight and fight for survival and are not reliant on others, when it comes to restraint, the mantra holds true: Less is more! Cats inherently resist intimate handling and restraint. By restraining them, we take away their sense of control and cause them to react. It is very easy to condition negative emotional responses. Scruffing is strongly discouraged as it is an act of dominance that cats may resent. Similarly, stretching is an inappropriate, disrespectful and unnecessary way to apply restraint. Every future experience builds on the previous negative (or positive) experience. Cat bags, masks, and gloves all carry the scents of similarly terrified patients plus other sundry smells (anal gland secretion, pus, blood, halitosis, etc.) A towel is all that is needed to wrap a cat in, in order to protect the handler. Remember, a cat would rather flee than attack.

Train all staff in respectful cat interactions and handling. An excellent and comprehensive resource is the American Association of Feline Practitioners (AAFP) and International Society of Feline Medicine (ISFM)'s Feline Friendly Handling Guidelines, downloadable at: www.isfm.net/wellcat/UK/FFHG.pdf. It is well worth reviewing and refining cat examination techniques as a clinic team for a consistent approach, the goal being to make them less threatening. Because value is "perceived worth" and because every visit is a valuable opportunity to educate the client, talk to the client and the cat throughout the entire procedure. Source and provide feline friendly medications, being sure to follow up one or more times with the client to find out how the patient is doing and if the client needs a refresher course on how to administer the medications. Be sure to send home an exam report with home care instructions for the client to refer to. Schedule recheck appointments or the next wellness visit before the client leaves the practice. The AAFP has created the Cat Friendly Practice program through which any interested clinic can raise its cat care IQ. (catfriendlypractice.catvets.com)

Meeting environmental needs improves health

Recently, it has been recognized that emotional well-being is highly dependent on meeting the environmental needs of cats. These include those relating to the indoor and outdoor physical environment, as well as a cat's social interactions, human and otherwise. In the AAFP and ISFM Feline Environmental Needs Guidelines, five pillars are described that form the basis of a healthy feline environment (Ellis, 2013). These pillars are:

1. A safe space
2. Multiple and separated resource stations (food, water, toileting areas, scratching areas, play areas, perches, resting and sleeping areas)
3. Opportunity for play and expression of predatory behaviors
4. Positive, consistent and predictable interactions with humans
5. An environment that respects the importance of a cat's sense of smell

When these are not met, cats become stressed to varying degrees. Some may express illness (such as inflammatory bowel disease, lower urinary tract inflammation), while others will manifest their distress through inappropriate elimination.

Other considerations

As cats age, they tolerate less time in the clinic. Siamese cats are especially prone to becoming depressed. Three days may be as long as a cat can stand the anxieties and indignities of hospitalization, even with daily visits from the owner. Consider capping intravenous catheters and send patients home, having them return for outpatient care. Even for in-hospital care, capping catheters off overnight (administering the overnight dose via the subcutaneous route) allows greater ease of movement, avoids alarms, which keeps patients awake. In either case, administer the overnight fluid volume subcutaneously.

Because cats "see" the world in overlapping clouds of smells, we should strive to provide familiar smells and reduce foreign, medicinal smells. Client-worn shirts or toys from home are helpful in cages. Feline facial pheromone may help to reduce stress. Because cats' sense of hearing is tuned more finely than ours, a quiet and reassuring environment is desirable. Cats should not be exposed to the sounds of predators, namely barking dog, but strange machines (faxes, printers, phones, dishwashers, centrifuges, etc.) should also be addressed. Reducing noises should be addressed when using certain induction agents as some enhance hearing (e.g., ketamine).

Avoid changing a cat's diet during hospitalization as is likely to result in inappetence and possibly the development of an aversion. If a change in diet is required for therapeutic reasons, try to make that change gradually in the safety of the home territory.

Taking a thorough history is especially important given cats' tendency to hide illness. Listening carefully to clients and their concerns is extremely important. Often clients detect changes that represent real problems. This is probably more common than the client who is blissfully unaware of significant health problems. By asking open-ended questions, we elicit a more detailed history than using only specific questions. For example, asking, "Have you noticed any changes in the contents of the litter box?" will probably evoke a yes or no answer. Whereas: "What does his stool look like?" followed by: "Would you describe it as hard pellets, moist logs, cowpie, or colored water? What colour is it? When did you first notice this?" will probably provide more useful answers. "Is there anything else?" is a very effective question.

Schedule a recheck appointment to evaluate the effect of any medical or nutritional therapy. Reassessing important variables (e.g., body weight, body condition score, previously abnormal laboratory results) and updating the patient history allows us to provide better care for our feline patients. Care of the client is essential to providing complete patient care. It is only through listening to, educating, and working with the client that we are able to offer the very best veterinary care.

Examples of practical applications

1. If a cat is uncooperative, a comprehensive physical examination can usually be done using only a towel as a protective barrier. Facing the cat away from you is less threatening for her. Confining the cat between your legs as you sit on the floor provides adequate persistent firm restraint that is reassuring rather than frightening.

2. Swaddling a cat's forelimbs and torso may help with blood and urine collection, placing the cat in lateral recumbency for cystocentesis and using the medial saphenous vein. This vein is also a superb choice for catheter placement and administration of intravenous medications. If the cat is allowed to have her front end in a sternal position while the back end is in lateral recumbency, she may struggle less.

3. Allow the client to be with the kitty as much as, and whenever, possible.

4. Recognize that a persistently elevated systolic value above 160 or 170 mm Hg probably represents true hypertension rather than the stress response. If in doubt, repeat the value later during the visit.

5. Feliway™ (Ceva Animal Health), a synthetic analog of a feline facial pheromone, may have a calming effect on cats. Spray (or wipe) it into kennels and carriers and even on your clothing before handling an anxious cat. Let the substance evaporate for a few minutes before placing the cat into the sprayed space. Feliway diffusers

plugged into treatment and hospitalization areas as well as reception and consultation rooms can help patients relax.

6. Elevated blood glucose and glucosuria may be a result of persistent stress. A diagnosis of diabetes, therefore, should be confirmed by finding an elevated serum fructosamine.

PART 3: RESPECTFUL HANDLING: IMPROVING CLIENT COMPLIANCE IN THE FELINE PRACTICE

While the number of cats kept as companions in North American homes continues to increase, the number of feline visits to clinics has been declining since 2001. Based on the AVMA's 2007 pet ownership and demographics survey, there were 13% more cats than dogs, yet cats failed to receive the same degree of veterinary attention. In small-animal practices, dogs represented 59% of office visits, cats only 39%. The 2011 Bayer Brakke study further noted three client-driven factors that limited the number of feline visits.

1. Inadequate understanding of the need for regular preventive health visits other than for vaccination.

2. Resistance to bringing a cat to the clinic because of the distress caused by putting a cat into a carrier and making the trip to the clinic.

3. The cost of veterinary care, in particular the frequency and size of price increases. (The state of the economy was a separate, external factor.)

In November 2012, Bayer conducted an online survey of 401 veterinary practice owners across the USA. The Bayer Veterinary Care Usage Study III: Feline Findings noted that 78% of veterinarians believed that better care for cats represented one of the most significant, missed opportunities for the profession. Yet, while 70% of those veterinarians were familiar with the earlier Bayer-Brakke studies, and while most recognized that cat owners consider a clinic visit to be stressful for themselves and their cats, nearly one-third of practices did not have staff trained on how to make visits less stressful for clients. Additionally, relatively few practices had adopted exam rooms used only for cats (35%), cat-only waiting areas that are physically and visually separated from dogs (18%), and cat-only days and appointment hours (11%). The study found that 46% of the surveyed clinics had recently started taking specific steps to increase visits among current feline patients, attract more cat-owning clients, and make their practices more "cat friendly".

Clearly there is a need to grow compliance among cat owners. However, part of the lack of awareness (at best) or reluctance (at worst) for making simple, inexpensive changes in attitude and facility is that many veterinarians and veterinary staff members prefer, or feel more comfortable, working with dogs than cats. The survey also identified that veterinarians find it easier to diagnose dog cases.

IMPROVING CLIENT COMPLIANCE

The verb "comply" means to act in accordance with a wish or command (Oxford), to conform, submit, or adapt (as to a regulation or to another's wishes) as required or requested (Miriam-Webster). For clients to comply with our recommendations, they have to fully understand, be willing and able to perform the actions that we are recommending. We need to enroll them so that they believe in the importance of these actions. But explanations aren't enough: on-going caring communication are needed to enhance client compliance.

Many clients believe that cats are self-sufficient, have very few needs, and are low maintenance pets. They don't understand that cats live as solitary hunters because their prey are too small to share. This means that they lack the resources of a supportive society. To avoid showing vulnerability they hide illness well. Additionally, cats are prey to larger birds and other species. This is critical in understanding why cats so easily feel self-defensive and how to work with cats. Educating potential and existing clients what these subtle signs of sickness are is a huge opportunity for increasing compliance. All veterinary team members also have to recognize that any admission of illness by a cat may signal a problem that has been going on for longer than recognized. Following are descriptions of signs that clients can be taught to look via direct emails, newsletters, the clinic website, and social media.

Subtle Signs of Sickness (adapted from www. haveweseenyourcatlately.com/Home.html and www. cathealthy.ca)

1. Inappropriate Elimination: Regardless of how "deliberate" it may seem to be, when a cat is avoiding or not using the litter box, they are trying to tell you something. The message may be that they are in physical discomfort or psychological distress. Physical problems include inflammation of the bladder or bowel, arthritis, hyperthyroidism, diabetes, dementia. Psychological distress may be due to social disturbance, anxiety due to other animals, children or adults, boredom, or a lack of opportunities to perform the full, natural repertoire of cat behaviours.

2. Changes in Interaction: Changes in how a cat interacts with people, other animals or his/her environment may indicate pain or distress.

3. Changes in Activity: A decrease in energy may be abrupt or gradual. The latter is often attributed to "just getting older", however, a healthy individual does not inherently "slow down" due to increasing age. A cause for such changes should be investigated. Dehydration, pain - from anything, including arthritis-, and hypokalemia are some of the problems that should be evaluated. The reverse is also true: an increase in energy in a previously

normal cat may be an indicator of incipient illness, most notably, hyperthyroidism or hypertension.

4. Changes in Sleeping Habits: This refers to pattern of sleeping (times of the day and night) as well as postures and locations. A cat with pain or with dementia may either sleep for longer or for shorter periods than previously. With FIV infection, the latter may occur. Night-time yowling suggests a decline in vision or hearing, hypertension, hyperthyroidism, pain or dementia.

5. Changes in Food And Water Consumption: This refers to quantity, preferences, as well as changes in behaviours associated with these activities (i.e., where, how often, attitude, amount at each instance, body posture, etc.).

6. Unexplained Weight Loss or Gain: As gratifying as it is to see rapid weight loss in a previously obese patient, even for those on appropriate dietary regimes, dramatic changes are neither desirable nor the norm. Oral pain may cause inappetence. Gradual weight and muscle loss may be related to ageing but should be monitored and investigated. Weight gain is most often from excess calories but could also be due to abdominal or thoracic fluid accumulation. Helpful tools include repeated assessment of body weight percentage weight change, body and muscle condition scoring.

7. Changes in Coat and Grooming: Excessive grooming may be caused by skin irritation (e.g., allergy, fleas, dryness), neuropathy, or be psychogenic (a way to reduce stress by releasing endorphins). A decrease in grooming is often associated with pain, often arthritic or oro-dental. Hairballs may be a sign of dermatologic or psychogenic problems, altered digestive motility or pain.

8. Signs of Stress: Along with aforementioned inappropriate elimination and overgrooming, signs of distress include hiding, chewing on non-food items, a flicking tail, ears placed further back than normal, unprovoked attacks.

9. Changes in Vocalization: These may be a change in tone, pitch, or urgency and frequency of vocalizing. See above regarding night-time yowling.

10. Bad Breath or Smelly Coat: Numerous oral and dental conditions result in halitosis; periodontal disease is extremely common in cats. The odour from sialoadenitis, infected ulcers, tumours, abscesses and anal gland secretions may be spread onto the coat via grooming.

However, even recognizing that their cat has a problem may not be enough to motivate the client to bring their cat in to the veterinarian. Screening to proactively identify disease early and to provide solid medicine can be an even harder "sell" because most people do not like bringing their cats in to the clinic. Many cat owners would rather provide care at home or even skip any form of consultation unless there is "something serious

going on"! The second significant opportunity to improve the lives of our patients and be of help to our clients is therefore enabling less stressful trips to the clinic.

Getting cats to the clinic

It is no fun bringing a cat to a veterinary clinic (for the owner or the cat)! All veterinary team members should be trained in coaching clients how to make the trip less stressful, from the experience at home, during transit, and once they arrive at the clinic. This conversation begins when the client calls to make an appointment or at the first visit with their cat. The American Association of Feline Practitioners (AAFP) has a free downloadable client handout entitled: Getting Your Cat to the Veterinarian (catvets.com/uploads/PDF/2011FelineFriendlyClientHandout.pdf). Clicker training can be used to help create positive associations with the carrier. Catalyst Council (www.catalystcouncil.org) has created excellent videos that clinic teams and clients can watch to learn how to accomplish this.

The frightening experience begins at home. Imagine the scenario from the cat's point of view: The carrier comes out, your caregiver is nervous, she chases you around and tries to force you into the carrier. You resist and may resort to self-defense. There are new or residual smells of human sweat, fear, maybe even blood. You may feel so anxious that you soil yourself! Eventually you are in the carrier. Everyone is exhausted. Then you are moved into a "car" that makes you move without any intention or action on your part. You may be a bit nauseous; certainly you are scared. You cry out repeatedly. You may vomit or soil yourself. Then the "car" stops and you get carried on a noisy and unfamiliar street and into a place with overwhelming smells and sounds! Help! And you are already aroused and anxious....look out!

We can reduce the stressors the cat encounters, or, in the case of a new cat, prevent them from occurring by teaching or habituating the cat to associate positive experiences with the carrier, the car, and even the clinic. By leaving the carrier out (or using a Hide-Perch-Go box/carrier) so that the cat sees it as non-threatening and enters it for meals, treats or other rewards, we reduce the initial tension and fight. Taking the cat on short car rides unassociated with the clinic helps to recondition the cat's negative associations with the clinic. Finally, taking the kitty to the clinic to be fussed over or only to get a treat will help teach the cat that the clinic isn't necessarily a horrible place.

Taking the household pet inventory

While there are a lot of cats who never receive veterinary care, there are a lot of cats living with existing clients we never see. We don't even know that they exist! If the cat is well or if the client has had a really bad experience in the past with a cat (or anticipates "bad behavior" from a cat), they are unlikely to voluntarily bring them in for

preventive care. By asking whether they have any other cats or pets when they any patient we can identify the un-served animals.

Improving the clinic experience

From the client's point of view: It wasn't fun to bring her, she isn't happy about being in the clinic and it isn't fun watching her be "manhandled". Once at the clinic, already stressed and frightened, it is extremely important to minimize or eliminating any further perceptions of threats. This requires that we imagine or try to see the clinic from the cat's point of view. The second and third of these presentations will speak in depth to these matters.

Making the environment more "feline friendly" can be as simple as having visual barriers in the seating/waiting area to prevent cats from seeing dogs. Covering the carriers with a towel will also help so that cats don't see each other. If possible, have separate cat-only waiting area. Restrict at least one examination room solely for cats to reduce the smells of predators and to be able to furnish it items needed for cat examinations and comfort.

Train all staff in respectful cat handling. An excellent and comprehensive resource is the AAFP and International Society of Feline Medicine (ISFM)'s Feline Friendly Handling Guidelines, downloadable at: www.isfm.net/wellcat/UK/FFHG.pdf. It is well worth reviewing and refining cat examination techniques with the goal of making them less threatening. Because value is "perceived worth" and because every visit is a valuable opportunity to educate the client, talk to the client and the cat throughout procedures. Source and provide feline friendly medications, being sure to follow up one or more times with the client to find out how the patient is doing and if the client needs a refresher course on how to administer the medications. Be sure to send home an exam report with home care instructions for the client to refer to. Schedule recheck or the next preventive healthcare appointments before the client leaves the practice.

The AAFP has created the Cat Friendly Practice program through which any interested clinic can raise its cat care IQ. (catfriendlypractice.catvets.com)

Facilitating compliance at home

Having a library of YouTube links or making your own clinic "how-to" videos is extremely helpful. YouTube videos made by lay people may have the advantage of being more convincing rather than those by healthcare professionals. Find ones that you and your staff think are best. Ask clients which ones they found and liked. There are many good links. Examples of useful illustrative clips to have on hand include how to:

- Give your cat a pill (see below)
- Give subcutaneous fluids: www.youtube.com/watch?v=OLOVw35w4Ns
- Administer insulin: http://www.youtube.com/watch?v=XeZgKLflJn4
- Measure blood glucose: www.veterinarypartner.com/Content.plx?A=605
- Use an inhaler for asthma medications: www.youtube.com/watch?v=INF1W8uaPEA
- Feed with a feeding tube: contact the author at hypurr@aol.com
- Change a KittyKollar (video) and Living with an E-tube (handout): www.kittykollar.com

You might also want to include syringe/assisted feeding. Cat caregivers like to show their skills and help others. Compile a selection of reading materials on the internet that you have vetted and feel comfortable with to guide those clients who want to learn more about their companion's medical condition. Superb client specific books are available from www.vetprofessional.com. They include: Caring For A Cat With Hyperthyroidism, Caring For A Cat With Chronic Kidney Disease, Caring For A Blind Cat among others.

Cornell University has a series of videos on a number of procedures and diseases at www.Partnersah.Vet.Cornell.Edu. They Include:

Brushing Your Cat's Teeth,

Giving Your Cat A Pill Or Capsule,

Giving Your Cat Liquid Medication,

Taking Your Cat's Temperature,

Trimming Your Cat's Nails.

Other free videos include: Caring For Your Diabetic Cat, Gastrointestinal Diseases In Cats, Cat Owner's Guide To Kidney Disease, Managing Destructive Scratching Behaviour In Cats and A Pet Owner's Guide To Cancer.

The ISFM has an excellent owner website: http://www.icatcare.org/advice. It includes handouts on medical conditions, general cat care, and videos. Websites specific for conditions include: chronic kidney disease: www.felinecrf.org and diabetes: www.felinediabetes.com, www.petdiabetes.com.

Follow-Up

In addition to reviewing discharge appointments of discussing future care, it is helpful to discuss reintroducing the patient to his/her home environment and housemates. Scheduling the next appointment before the client leaves, be it for a recheck evaluation, or for their semiannual or annual visit, encourages continuation of care. Especially useful in a first year of life program, it helps to lay the foundation for a culture of lifelong preventive healthcare.

Even with a motivated and educated client, the likelihood of a successful outcome depends on that client having a relationship built on trust, communication and knowing that the clinic team cares. Follow-up phone calls are a very effective investment of time.

Facilitating Finances

The Bayer study showed that clients want costs spread out over time. Fear of large bills is another significant factor preventing owners from bringing their cats to the clinic. Many practices have wellness plans. Check out the preventive healthcare protocols of Cat Healthy (cathealthy.ca) Directing clients toward pet health insurance that covers both preventive and accident/illness before it is needed is sound medical advice. This can save lives that might otherwise be lost because of hesitation to seek care or a decision to euthanize a pet out of financial concerns.

SUMMARY

Educating clients about the subtle signs of illness, reducing stressful travel and clinic visits, following up with personalized care, improves compliance. Taking a household pet inventory allows us to discover an un-served pet population. Cat Healthy (cathealthy.ca) has many resources to help you. Focusing on these unmet needs gives us the opportunity to:

- Provide preventive healthcare
- Detect disease early when we can prevent or alleviate suffering and save expense
- Protect life and enhance welfare
- Build trust with our clients
- Increase clinic visits.

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SOCIAL MEDIA FOR VETERINARIANS

HANDLING ONLINE REVIEWS

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"Practices need to get involved if they want to thrive in the future." - **Eric D. Garcia**

Whether your practice has been around for just a few days or a few decades, I can promise you that one thing is consistent between both:

Your online reputation is either driving people toward, or away from, your veterinary practice.

Unfortunately, if you haven't started to secure positive reviews online to both maintain and enhance your reputation, you run the risk of the many pitfalls that may follow. Something as simple as a Google Review can actually have profound implications on your business and whether or not you're securing the volume of pet owners that your practice is capable of.

While I once wrote specifically addressing the impact of Google on your online reputation as a whole (at simplydonetechsolutions.com/safeguarding-reputation), I will focus more on the power of Google Reviews and how to leverage this resource to enhance your business, maintain a stellar reputation and increase engagement from pet owners in the process.

When pet owners have a positive experience with your veterinary practice but don't leave a review, this encounter doesn't have the long-term impact it's capable of having.

Yes, the client was satisfied by their experience. But there's nothing over the long haul to prove it, enhance your reputation or otherwise garner a testimonial for future marketing. This can harm your overall visibility and consequently decrease the number of referrals you're receiving from Google. This is far from marginal, because Internet referrals from sites such as Google (or competitors like Bing and Yahoo!) **now account for one of the top three sources veterinary practices receive clients from.**

That's right, these online resources account for one-third of new business, which of course equates to vast amounts of annual revenue. While traditional word of mouth is still a powerful tool necessary for securing new clients, your online reputation is something like a **digital word of mouth**, which reverberates far and wide to pet owners who are searching for reliable care. There's no ignoring this reality, which leaves a more permanent footprint than traditional word of mouth and can't be underemphasized in the modern digital age. It's more important now than ever to begin securing new reviews as soon as possible.

This is partially because practices that have no reviews online are the most vulnerable. If this practice, who has not previously established their online reputation, receives a single negative review**, this is the only thing that prospective clients will see online.**

This can be detrimental for both new and existing business, as a negative review can cause doubt for new prospective client owners and even serve as a red flag to current clients of your veterinary practice.

That's why when veterinary practices approach me to ask where to start when it comes to their marketing efforts, I always tell them the same thing: **Start by looking at your online reviews!**

Your online reputation is the cornerstone of your success and will likely dictate future success as well. Even clients who ask me for a marketing strategy but haven't worked extensively on their online reputation receive the same reply. We may be able to create a fantastic brand with clear, beautiful messaging and a sleek, modern website, but it won't do anything if your practice doesn't have a great reputation online.

Practices must get involved with online reputation management, paying very close attention to Google Reviews, if they want to thrive both now and into the future.

The difference between a practice with a 3-star ranking and a 4-star ranking can make a major impact in your ability to market yourself and secure new business.

This is especially true when a single-star can impact your ability to appear in the map section of the first page of a Google search. A huge amount of traffic and attention goes toward the results that appear first, and practices with lower scores will get buried in online search results. This is because Google selectively filters the results of veterinary practices based off the reviews that they secure online.

It's actually in Google's best interest to do this, because providing results of higher-rated veterinary practices tends to enhance the search experience for pet owners, who are typically looking for the best resource available in their local area. If a pet owner searches (let's say, in the Tampa, Florida region for example) and only finds veterinary practices with 2 and 3 star reviews, they'll likely keep searching. Instead, Google wants to streamline search results and deliver the best results possible to the user on the very first try.

That's why practices with 4 and 5 star ratings often overwhelmingly appear first in search results!

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This may not occur if a rural area doesn't have a wide-selection of veterinary practices to choose from, but it will always be the case if there is competition in the marketplace.

Ok, let's try a little pop-quiz to see how it works. Let's say I find three veterinary practices during a Google search, and they have the following reviews:

Practice A.) 3-star rating with 10 reviews.

Practice B.) 4-star rating with 12 reviews.

Practice C.) No reviews.

In this common scenario, where would you go?

Statistically speaking, almost all those surveyed will choose **Practice B.** Practice B may not have the best veterinarians, the best service or even the best equipment. Still, their online reputation will land them countless new clients, as it's simply our nature to gravitate toward the most secure solution. Practice B has the highest rating overall, and this lets us feel secure in our decision when taking our pet there.

By taking ownership over your online reputation, you can leverage Google Reviews to garner new business and make sure your visibility is maximized online.

Get involved by following these simple steps:

(1) Claim your business with Google and list your hours, phone number, location and photos. This is your chance to ensure your listing is accurate and displays vivid imagery to attract new pet owners.

(2) Reply to positive reviews as they come in! Engage with pet owners who have taken the time to submit a review and show appreciation for those brand advocates for supporting you.

(3) Make sure to solicit new reviews, especially from pet owners who are delighted after their appointment. You can simply ask clients to leave a review, and send a follow-up email clearly explaining how to leave a review for your practice.

It's important to make leaving a review as easy as possible for your client, so feel free to print a step-by-step guide that ensures all types of pet owners know how to leave a review. There are also services available, such as AllyDVM, Vetstreet, ePetHealth, Rapport, Banyan, or Testimonial Tree, which can help you to secure reviews by integrating directly within your practice management software and sending surveys.

By enlisting the help of one of these recommended services, you'll gain the peace of mind that comes from knowing that only clients who leave great survey results, will be asked to go a step further by leaving you a review online. Those who had a less glowing experience will be asked if they're open to discussing their experience, allowing you an opportunity to address their concerns and enhance your veterinary practice accordingly.

No matter the route you choose, understanding and leveraging the impact of Google Reviews will allow you to increase customer satisfaction, enhance your practice and engage with new clients; contributing to a level of future success you may have once never imagined possible.

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SVA INFECTIOUS DISEASES

CANINE DISTEMPER UPDATE FROM RESEARCH TO CLINICAL PRACTICE

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CANINE DISTEMPER UPDATE FROM RESEARCH TO CLINICAL PRACTICE

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Canine distemper is a fatal infectious disease which distributed worldwide not only in domestic mammals but also spread through wildlife animals leading to depopulation in endangered species[1]. The causative pathogen is canine distemper virus (CDV); belonging to the genus Morbillivirus, family **Paramyxoviridae**; and affects the broad host ranges of the order Carnivora, including Canidae (dogs, foxes), Felidae (cats, lions), Mustelidae (ferret, mink, badger), Procyonidae (raccoons, kinkajous, lesser panda), Ursidae (bear), and Viveridae (civet)[2]. Recently, CDV expanded its susceptible hosts into the order Rodentia (rodent), and nonhuman primate (monkey)[3,4]. Additionally, there were some studies reported the detectable antibody titer against CDV in Artiodactyla (pig, dear) and Proboscidea (Elephant)[5,6]. Viral antigen was also accidentally detected in fleas (**Ceratophyllus sciurorum**) collected from the carcass of dead mink (**Neovison vison**). Nevertheless, domestic dogs have been suspected as a probable reservoir for free-ranging wildlife infections due to the potential for asymptomatic carrier animals. CDV has been known causing multisystemic disease and severe immunosuppression in infected hosts.

The main route of infection is via aerosol droplet secretions from the oral or nasal cavities of infected animals and by direct contact from infected to non-infected dogs. However, the role of the horizontal transmission vector, for example flea, remains elusive and need further investigations[7]. The infection is associated with all lymphatic tissues, epithelium of respiratory, gastrointestinal, and urogenital tracts, as well as the central nervous system (CNS); and optic nerves are also susceptible.

Clinical signs of affected dogs are often listless and have a decreased appetite. In milder cases, signs may be similar to other pathogens causing canine infectious respiratory disease complex (CIRDC)[8]. In subclinical infection, the viral shedding may also occur depending upon the level of specific humoral immunity in the host during the viremic period. Systemic signs are most common in unvaccinated dogs, particularly puppies aged less than 6 months, as maternal immunity wanes. Conjunctivitis, nasal discharge, cough, and fever are classic signs. Respiratory infection may involve the lower respiratory tract with possible primary viral pneumonia. Secondary bacterial infection is frequently associated and leads to fatal pneumonia. Vomiting and diarrhea may be present. Neurologic signs may be concurrent with epithelial signs, such as respiratory disease, conjunctivitis, vomiting, diarrhea) with encephalitis due to direct viral replication. Seizures and myoclonus are two of the more common signs, in which the latter may affect limbs or may manifest as chewing gum motion. Ocular disease may also present; lesions include anterior uveitis, optic neuritis, and retinal detachment. Infection during pregnancy may lead to abortion or stillbirth. Puppies infected prior to permanent dentition may have enamel hypoplasia. Digital hyperkeratosis may occur in some dogs.

Genome of CDV contains 15,690 nucleotides and possesses a non-segmented, negative sense, single-stranded RNA encoding six core-structural proteins. The proteins are phospho (P)-, nucleocapsid (N)-, large polymerase (L)-, matrix (M)-, fusion (F)- and hemagglutinin (H) proteins. Despite the L, N and P proteins serving together as a viral RNA constitute the ribonucleic protein (RNP), the H and F glycoproteins are well-recognized as a part associated with viral entry process by facilitating the binding and fusion to the host cells, respectively. Moreover, the H glycoprotein, which has a highly genetic variation, also acts as a protective antigen playing an important role of humeral antibody induction. The H gene is now contributed to be a part for strain classification based on genetic and related geographic diversities. To date, the CDV strains have been established at least 14 genetic lineages composing of America-1 and its vaccine strains, America-2, Europe Wildlife, Arctic, South Africa, America-1/Europe, South America-1, -2 and -3, Rockborn, Asia-1, -2, -3, and -4. The CDV Asia-4 strain, which was classified as a new lineage based on the H gene, was initially discovered in domestic dogs in Thailand and recently also detected in China [9,10].

Ante-mortem diagnosis can be done via virus identification in a clinical sample through use of convention or real-time reverse transcription polymerase chain reaction (RT-PCR) of whole blood, a swab of conjunctiva or nasal, cerebrospinal fluid (CSF), urine. Urine is a good choice for PCR testing in dogs with CDV encephalitis after resolution of epithelial signs. CDV may be detected in urine for a longer period than other sample types. Post-mortem diagnosis based on microscopic findings is used to confirm the infection.

The pathognomonic lesion is eosinophilic intranuclear/intracytoplasmic inclusion bodies in such infected as glial cells, neurons, epithelial respiratory cells, and cells of the gastrointestinal and urogenital tracts. Virus isolation is the gold standard for diagnosis and is useful in low levels of virus infection through observation of typical syncytial cell formation.

Attenuated live vaccines have been used for controlling the disease for many decades, yet a number of CDV infections in vaccinated dog were still observed. In Thailand, we identified circulating CDV strains into 2 clusters; Asia-1 and Asia-4. By using the restriction fragment length polymorphism (RFLP) techniques, we could effectively differentiate among individual wild-type and vaccine lineages presenting in Thailand.

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ONCOLOGY

APPROACH AND MANAGEMENT OF ORAL TUMORS IN DOGS AND CATS

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APPROACH AND MANAGEMENT OF ORAL TUMOURS IN DOGS AND CATS

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Learning objectives: Develop an approach to the diagnosis and staging of oral tumours in dogs and cats. Understand the principles of surgery for oral tumours, and indications for other treatments such as radiation therapy, chemotherapy or immunotherapy either as adjuncts to surgery or as sole treatments.

The most common oral tumours in dogs are melanoma, squamous cell carcinoma (SCC), and fibrosarcoma (FSA), though many other tumours can occur in the oral cavity including the tongue. In cats, 60-70% of oral tumours are squamous cell carcinoma with fibrosarcoma being the second most common. General principles of staging and diagnosis apply regardless of tumour type. Although cytology may be diagnostic, given the difficulty of FNA in a conscious patient, sedation/anaesthesia and incisional biopsy for histopathology is generally most appropriate. All the common tumours in dogs and cats have some risk of metastasis, so staging is indicated prior to surgery. CT is generally preferred to assess the primary tumour, local lymph nodes (LN) and lungs all at the same time. Abdominal imaging could be considered, especially for canine oral melanoma. LN palpation is not sensitive or specific for metastasis and so cytology/histopathology is recommended. However, identification of the 'sentinel' lymph node based on anatomy alone can be challenging. Metastasis to both ipsilateral and contralateral submandibular and retropharyngeal lymph nodes can be seen in dogs with malignancies of the head, including the oral cavity.

For tumours without distant metastasis, surgery (primary tumour +/- LN excision) is recommended where possible. However, oral tumours may be extensive at the time of diagnosis, especially those located more caudally in the mouth, precluding excision.

Surgery:

General surgical oncologic principles apply and oral tumours should be removed via wide en bloc surgical resection. For oral tumours associated with the maxilla or hard palate, this will be achieved by maxillectomy which can be partial or a complete hemimaxillectomy. Closure of the resultant oronasal defect is by a local mucosal flap raised from the ipsilateral gingiva. Most dogs have god to excellent functional outcomes and minimal cosmetic changes. Mandibular tumours require mandibulectomy (rostral, segmental or hemi). Post operatively, some animals experience 'mandibular drift' where the mandibular teeth do not align with the maxillary teeth. Cats can undergo mandibulectomy or maxillectomy but often require a longer period of adaption after surgery and oesophageal feeding tubes are required to provide nutrition in the post-operative period.

Removal of the draining lymph nodes is recommended in cases of oral tumours to provide accurate staging information. A recent description of a technique to remove all three draining lymph nodes through a single ventral cervical access incision has been published.

Lingual tumours can be treated by partial or subtotal glossectomy. The tongue is very vascular so appropriate haemostatis is required during surgery. If major glossectomy is required (e.g. for large or caudally located tumours), then the owners should be aware of the management and morbidity concerns before undertaking such a procedure.

Tonsillar tumours are most commonly SCC. They can be removed by tonsillectomy which is made easier with the use of a vessel sealing device. Surgery is considered palliative in these cases as tonsillar SCC is a highly metastatic and aggressive disease in dogs.

Following surgery, adjunctive therapy should be considered from two aspects - the need for additional local control due to narrow or incomplete histologic margins and the need for systemic therapy due to risk of metastasis. From a local control standpoint, assuming an aggressive first surgery has been performed, as discussed above, more extensive surgery is not generally possible in the oral cavity. Local recurrence can occur despite apparently complete excision (approx. 15-20% in canine oral tumours in general), but is very common following incomplete excision (>60%), and recurrent disease negatively impacts survival time. In the specific cases of feline oral SCC and canine oral FSA, local recurrence may be more common than other oral malignancies. In general, local recurrence is less common for mandibular tumours than maxillary tumours, likely due to the ability to obtain wider margins. Local recurrence is also greater for larger tumours, again likely due to incomplete excision. Where additional local control is indicated, radiation therapy (RT) is often very effective. With aggressive local therapy, outcomes for SCC and FSA in dogs can be very good, with median survival times of greater than 1.5-2 years (SCC generally better than FSA). Acute side effects from RT in the oral cavity are common but typically manageable with aggressive analgesia, and generally resolve within a few weeks. Feeding tubes may be required in some patients.

Adjuvant systemic therapy after local therapy is generally not indicated for canine oral SCC and FSA as they do not typically cause distant metastasis (varies with the study). If lymph node metastasis is documented, adjuvant chemotherapy may be warranted. In contrast, oral melanoma in dogs is considered to have a high risk of metastasis. The local disease (primary tumour +/- LN) can often be effectively controlled with surgery and/or RT, but development of metastasis is common. Systemic therapies including chemotherapy (carboplatin, dacarbazine, temozolomide) and immunotherapy (Oncept and other vaccines) have not shown a statistically significant improvement in survival over local therapies alone. Small tumours (< 2cm) may have fair to good outcomes with aggressive local therapy alone (median 1.5-2 years compared to 6-8 months for larger tumours).

For tumours where surgical excision is not possible, radiation therapy is generally the preferred option. Canine oral melanoma tends to be very radiation responsive. Hypofractionated/palliative type protocols are generally recommended for melanoma. As discussed above, although the local tumour can be effectively controlled, metastasis is common. There is some information about RT as sole treatment for canine SCC, with median control time of approximately 1 year reported with definitive-type protocols, though this may be better in smaller tumours. Canine FSA is generally thought to be less radiation responsive, though median control of 10 months is reported in one study. Palliative RT can be considered for any painful oral tumour.

Chemotherapy could be considered for macroscopic oral tumours if surgery and radiation are not feasible, or for metastatic tumours, however evidence of efficacy is sparse. Canine oral melanoma may respond to platinum agents, and dacarbazine & its related drug temozolomide have shown some efficacy in melanoma in humans. The Oncept melanoma vaccine may also have some benefit in some dogs, with occasional responses reported in macroscopic disease. There may be a potential benefit to combining Oncept with metronomic chemotherapy either in the adjuvant setting or with macroscopic melanoma. For canine oral SCC, responses are reported to carboplatin and piroxicam in some dogs, toceranib (+/- NSAID +/- cyclophosphamide), and even piroxicam alone. Canine oral FSA is likely to be relatively chemoresistant given responses of macroscopic soft tissue sarcomas in general, though some response to

doxorubicin or to metronomic chemotherapy may be seen.

There are three specific companion animal oral tumours that bear separate consideration.

- Feline oral SCC - these are often locally extensive at diagnosis, and if not surgical are very resistant to treatment, with median survival times of a few months. Even with surgery, recurrence is common, though for small tumours that can be treated with mandibulectomy and adjuvant radiation, survival times may be improved. RT alone does not appear to be effective in most cases. There is one small study evaluating systemic bleomycin chemotherapy along with radiation and surgery where outcomes may have been better, and toceranib (+/- NSAIDs +/- metronomic chemotherapy) may be of benefit in some cats. The use of NSAIDs is recommended based on COX expression in feline SCC for potential anti-tumour effects as well as for analgesia.

- Canine tonsillar SCC - unlike non-tonsillar oral SCC (with the possible exception of lingual SCC), tonsillar SCC is highly metastatic and aggressive in dogs. Often the diagnosis is made from a metastatic lymph node which is externally visible, rather than the primary tumour in the tonsil. Survival times are generally short (months), even with treatment, though combinations of chemotherapy (usually carboplatin) and radiation therapy may achieve reasonable palliation. However, in those where the disease is diagnosed early (no metastasis, one tonsil affected), prolonged survival may be possible with treatment.

- Canine hi-low (histologically low grade biologically high grade) FSA - a subset of canine oral FSA will appear non-aggressive (low grade sarcoma or even fibroma or reactive fibrous tissue) and yet have very aggressive behaviour with rapid local growth and extensive tissue invasion. This syndrome seems to be more common in the maxilla of large breed dogs. If amenable to aggressive therapy, outcomes can still be favourable.

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WSV18-0152

APPROACH AND MANAGEMENT OF ORAL TUMORS IN DOGS AND CATS

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APPROACH AND MANAGEMENT OF ORAL TUMOURS IN DOGS AND CATS

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Learning objectives: Develop an approach to the diagnosis and staging of oral tumours in dogs and cats. Understand the principles of surgery for oral tumours, and indications for other treatments such as radiation therapy, chemotherapy or immunotherapy either as adjuncts to surgery or as sole treatments.

The most common oral tumours in dogs are melanoma, squamous cell carcinoma (SCC), and fibrosarcoma (FSA), though many other tumours can occur in the oral cavity including the tongue. In cats, 60-70% of oral tumours are squamous cell carcinoma with fibrosarcoma being the second most common. General principles of staging and diagnosis apply regardless of tumour type. Although cytology may be diagnostic, given the difficulty of FNA in a conscious patient, sedation/anaesthesia and incisional biopsy for histopathology is generally most appropriate. All the common tumours in dogs and cats have some risk of metastasis, so staging is indicated prior to surgery. CT is generally preferred to assess the primary tumour, local lymph nodes (LN) and lungs all at the same time. Abdominal imaging could be considered, especially for canine oral melanoma. LN palpation is not sensitive or specific for metastasis and so cytology/histopathology is recommended. However, identification of the 'sentinel' lymph node based on anatomy alone can be challenging. Metastasis to both ipsilateral and contralateral submandibular and retropharyngeal lymph nodes can be seen in dogs with malignancies of the head, including the oral cavity.

For tumours without distant metastasis, surgery (primary tumour +/- LN excision) is recommended where possible. However, oral tumours may be extensive at the time of diagnosis, especially those located more caudally in the mouth, precluding excision.

Surgery:

General surgical oncologic principles apply and oral tumours should be removed via wide en bloc surgical resection. For oral tumours associated with the maxilla or hard palate, this will be achieved by maxillectomy which can be partial or a complete hemimaxillectomy. Closure of the resultant oronasal defect is by a local mucosal flap raised from the ipsilateral gingiva. Most dogs have god to excellent functional outcomes and minimal cosmetic changes. Mandibular tumours require mandibulectomy (rostral, segmental or hemi). Post operatively, some animals experience 'mandibular drift' where the mandibular teeth do not align with the maxillary teeth. Cats can undergo mandibulectomy or maxillectomy but often require a longer period of adaption after surgery and oesophageal feeding tubes are required to provide nutrition in the post-operative period.

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Following surgery, adjunctive therapy should be considered from two aspects - the need for additional local control due to narrow or incomplete histologic margins and the need for systemic therapy due to risk of metastasis. From a local control standpoint, assuming an aggressive first surgery has been performed, as discussed above, more extensive surgery is not generally possible in the oral cavity. Local recurrence can occur despite apparently complete excision (approx. 15-20% in canine oral tumours in general), but is very common following incomplete excision (>60%), and recurrent disease negatively impacts survival time. In the specific cases of feline oral SCC and canine oral FSA, local recurrence may be more common than other oral malignancies. In general, local recurrence is less common for mandibular tumours than maxillary tumours, likely due to the ability to obtain wider margins. Local recurrence is also greater for larger tumours, again likely due to incomplete excision. Where additional local control is indicated, radiation therapy (RT) is often very

effective. With aggressive local therapy, outcomes for SCC and FSA in dogs can be very good, with median survival times of greater than 1.5-2 years (SCC generally better than FSA). Acute side effects from RT in the oral cavity are common but typically manageable with aggressive analgesia, and generally resolve within a few weeks. Feeding tubes may be required in some patients.

Adjuvant systemic therapy after local therapy is generally not indicated for canine oral SCC and FSA as they do not typically cause distant metastasis (varies with the study). If lymph node metastasis is documented, adjuvant chemotherapy may be warranted. In contrast, oral melanoma in dogs is considered to have a high risk of metastasis. The local disease (primary tumour +/- LN) can often be effectively controlled with surgery and/or RT, but development of metastasis is common. Systemic therapies including chemotherapy (carboplatin, dacarbazine, temozolomide) and immunotherapy (Oncept and other vaccines) have not shown a statistically significant improvement in survival over local therapies alone. Small tumours (< 2cm) may have fair to good outcomes with aggressive local therapy alone (median 1.5-2 years compared to 6-8 months for larger tumours).

For tumours where surgical excision is not possible, radiation therapy is generally the preferred option. Canine oral melanoma tends to be very radiation responsive. Hypofractionated/palliative type protocols are generally recommended for melanoma. As discussed above, although the local tumour can be effectively controlled, metastasis is common. There is some information about RT as sole treatment for canine SCC, with median control time of approximately 1 year reported with definitive-type protocols, though this may be better in smaller tumours. Canine FSA is generally thought to be less radiation responsive, though median control of 10 months is reported in one study. Palliative RT can be considered for any painful oral tumour.

Chemotherapy could be considered for macroscopic oral tumours if surgery and radiation are not feasible, or for metastatic tumours, however evidence of efficacy is sparse. Canine oral melanoma may respond to platinum agents, and dacarbazine & its related drug temozolomide have shown some efficacy in melanoma in humans. The Oncept melanoma vaccine may also have some benefit in some dogs, with occasional responses reported in macroscopic disease. There may be a potential benefit to combining Oncept with metronomic chemotherapy either in the adjuvant setting or with macroscopic melanoma. For canine oral SCC, responses are reported to carboplatin and piroxicam in some dogs, toceranib (+/- NSAID +/- cyclophosphamide), and even piroxicam alone. Canine oral FSA is likely to be relatively chemoresistant given responses of macroscopic soft tissue sarcomas in general, though some response to

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ENDOCRINOLOGY

TIPS ON DIAGNOSIS AND MANAGEMENT OF CANINE HYPERADRENOCROTICISM

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ENDOCRINOLOGY: TIPS ON DIAGNOSIS AND MANAGEMENT OF CANINE HYPERADRENOCORTICISM

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Hyperadrenocorticism (HAC) is a common endocrinopathy in middle to older aged dogs. Clinical signs to make the clinician suspicious of hyperadrenocorticism include many "p's" polyphagia, polyuria/polydipsia, pyoderma, pot bellied appearance, and persistent urinary tract infections. Physical examination findings include hepatomegaly, truncal obesity, muscle wasting, panting, bilaterally symmetrical alopecia, thin, hyperpigmented skin, calcinosis cutis, and lipid deposits.Dogs get HAC in 2 ways: pituitary tumors (85%) and adrenal tumors (15%). Both produce an excess of cortisol that causes the disease entity. Three screening tests are available. The urine cortisol:creatinine ratio is very sensitive (100%) although not specific. Therefore if the urine cortisol:creatinine ratio is positive, another screening test will be necessary in order to diagnose the disease. A second screening test for HAC is the low dose dexamethasone suppression test. This should be to go to test unless there is significant concurrent disease such as diabetes mellitus. It is sensitive (89-92%) but with a decrease in specificity (75%). It is least specific in animals with concurrent disease. A third screening test for HAC is the ACTH Stimulation Test. It is less sensitive (75-83%) than the low dose dexamethasone test but it is more specific (85%). It is more specific in animals with concurrent disease and recommended for use in dogs with concurrent diabetes mellitus to decrease the possibility of a false positive response. The practitioner measures cortisol before and 60 min after injection of 5 ug/kg synthetic ACTH (cortrosyn). In diabetic patients, the owner should feed and administer insulin as usual. A simultaneous glucose curve should be performed in conjuction with the ACTH-stimulation test to ensure that the animal has blood glucose within the optimal range during testing. If ACTH-stimulation testing supports HAC, then treatment should be initiated. If HAC is still suspected but is not supported by the ACTH-stimulation test, then the test can be repeated 2-4 weeks later.

The practitioner should determine whether the patient has pituitary or adrenal dependent disease. This will direct therapy choice as well as help predict cost of treatment, explain potential neurologic disease, predict time of resolution of signs, and mitigate owner frustration with therapy (hopefully). An endogenous ACTH concentration can be evaluated. Dogs with adrenal tumors have low endogenous ACTH levels (<10 pg/ml). Mishandling of the plasma sample will result in artificially low ACTH levels, leading the practitioner to erroneously conclude the presence of an adrenal tumor. Abdominal ultrasound can be very helpful in identification of an adrenal tumor or bilateral adrenal hyperplasia. The practitioner should be cautious in interpreting results of an abdominal ultrasound in that adrenal masses may be nonfunctional, may be a pheochromocytoma, or the animal may have pituitary-dependent disease as well as an adrenal tumor. Bilateral adrenal hyperplasia may also occur in animals with concurrent non-adrenal disease.

Medical treatment of HAC includes trilostane (Vetoryl[R]) and mitotane (Lysodren[R]). Ketoconazole and deprenyl (Anipril[R]) have also been advocated, but are substantially less effective than trilostane or lysodren. Surgical options include adrenalectomy and hypophysectomy. Trilostane is a competitive 3B-OH steroid dehydrogenase inhibitor that inhibits the conversion of pregnenolone to progesterone in the adrenal gland. This blocks the formation of the end products of progesterone including cortisol and aldosterone. Trilostane can be compounded but a manufactured product is available (Vetoryl [R], Dechra Pharmaceuticals). Vetoryl[R] is FDA approved for veterinary use so that the veterinarian is legally protected. Vetroryl[R] is much more consistent in active ingredients than compounded trilostane and should be used when possible. The drug must be given daily or twice daily. The recommended dose is 1.5-3 mg/kg, with the lower dose given bid considered safer and more efficacious. Side effects of trilostane are similar to those of mitotane and prednisone should be sent home with the owner similarly to that done with mitotane therapy. Side effects of trilostane include glucocorticoid, mineralocorticoid or glucocorticoid + mineralocorticoid deficiency. Complete adrenal necrosis has been reported, although this a rare and idiosyncratic response. Over time adrenal glands will get enlarged and irregular, but no problems with this have been noted. Investigators have reported elevations of 17 OH progesterone indicating that trilostane has other actions that those expected based on just its pharmacology.

Trilostane therapy should be monitored 10-14 days after initiation. An ACTH-stimulation test measuring cortisol is performed 4-6 hours after the morning dose of trilostane is given. Results of the ACTH-stimulation are ideally a cortisol in the normal range pre- and post- ACTH with little stimulation, similar to what is desired with lysodren therapy. A sodium/potassium level should be measured as well. This often decreases with trilostane treatment because of increasing K+ due to loss of aldosterone function.Mitotane is a DDT derivative. It kills adrenocortical cells with a preference for the cortisol producing ones (zonae fasciculata and reticularis). The induction phase consists of administering 35 (big dog)-50 (little dog) mg/kg PO q.d. x 5-7 d. The maintenance phase is 35-50 mg/kg PO divided twice per week. The practitioner may work with the 500 mg tablet size or may have it compounded. Owners should be discharged with instructions to call the veterinarian if any of the danger signs including loss of appetite, lethargy, vomiting, diarrhea, or "just aint right" occur. The practitioner should also send home at least 2 doses of prednisone at 0.5 mg/kg to be given if the animal becomes ill from the mitotane. Side effects of mitotane include: adrenal cortical destruction producing a glucocorticoid-deficient addisonian or a full-blown addisonian. Often the adrenocortical deficiency is reversible. Another side effect is liver toxicity. This often resolves once off Lysodren, but it cannot be used again. Monitoring mitotane therapy should occur after the induction phase. An ACTH-stimulation test should be performed the morning after the last dose of mitotane during the induction phase. Ideally the cortisol should be within the normal range pre- and post- ACTH injection. ACTH should cause a minimal increase in the cortisol level. The practitioner should also perform bloodwork to evaluate liver health. Clinical signs of the animal should be discussed with the owner. In cases where the ACTH-stimulation test results do not agree with the resolution (or non resolution) of clinical signs reported by the owner, the clinical signs should be believed. Which therapy should the practitioner choose to treat HAC. In comparing trilostane and mitotane, both are as efficacious in controlling clinical signs and in the longevity of the treated animals with PDH or adrenal dependent disease. Both are sold commercially and cost is approximately the same. Some considerations are that trilostane must be given every day while mitotane can be given 2-3 times per week in a stabilized animal. Mitotane should not be given to animals with liver disease. The effects of adrenal gland hyperplasia/metaplasia by trilostane have not been fully investigated but appear to be unproblematic. The author recommends the use of trilostane initially as it is easier and safer for owners to administer; mitotane tablets are not coated and owners can ingest excess drug power if they do not wash their hands frequently. The dosing schedule for trilostane is also easier for owners to manage. If dogs do not respond well to trilostane, mitotane therapy can be instituted.

43RD WORLD SMALL ANIMAL VETERINARY ASSOCIATION CONGRESS AND 9TH FASAVA CONGRESS

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WSV18-0006

WAVMA ORNAMENTAL FISH DISEASES
CLINICAL ASPECTS OF DESIGN AND FUNCTION OF AQUASYSTEMS

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CLINICAL ASPECTS OF THE DESIGN AND FUNCTION OF AQUASYSTEMS

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The pet fish practitioner should have a thorough understanding of the clinically significant life support equipment found in aquasystems. Along with the physical exam and diagnostic workup of the aquatic species and water quality assessment, the assessment of the design and function of the aquasystem and equipment is an integral part of the case workup. This usually requires an onsite visit, especially for koi medicine, which will account for over 75% of the caseload of the pet fish practitioner. Along with examining both the mechanical and biological filtration, of critical importance is the understanding of the circulation, gas exchange and temperature control and the interactions between these factors. In reviewing ponds, a primary assessment involves the nutrient balance. Ponds may be divided into three groups: water gardens, characterized by small decorative fish species, an accent on natural appearance, having much of water surface covered by plants and no feeding of the fish; the goldfish pond, with small decorative fish species, the water surface largely open to view, and with regular feeding of the fish; and the koi pond, where koi and/or other large species are kept, the water surface being largely open to view, with regular heavy feeding of the fish. The next assessment involves the basic design concept, of which two types dominate. The first is the shallow dished pond. Its attributes are a traditional natural appearance, is the easier to build, with a cost about one-half that of a deep pond. It utilizes "in pond" settlement of waste, its biology dominated by a very high number of micro-organisms along the bottom. This type is best for a water garden or goldfish pond, as it has a high stocking density when koi are kept. Negative aspects are that it's prone to unstable water conditions (temperature, oxygen levels) and requires regular clean-outs when koi are kept (yearly cost). Heron predation can be a problem. The alternative is the deep pond. Here the circulation and filtration systems are designed for efficient high water quality production with more stable conditions. This is the best type for koi ponds, as it has a lower (half) stocking density of a shallow pond of the same footprint. The equipment creates a system that

is self-cleaning (no yearly maintenance costs). Heron predation is minimized. The negatives are the equipment and installation cost double that of the first and a more formalized appearance. Most large ponds of 50,000 gallons (200,000l.) or more are of the first type. They tend to have a low stocking density. Circulation and gas exchange remain important nevertheless. This is often accomplished via fountains or wetlands filters.

The process of gas exchange is critical and is assured by air diffusers and stones, waterfalls and spillways, surface skimmers, fountains, venturis and pond deicers. Water temperature is moderated by ambient air temperature (poor control), via a greenhouse, which is better, but still variable and with heaters. Mechanical filtration occurs through sedimentation (no filter), via Internal box filter (submersed near the pond bottom), external box filter (outside the pond), prefilter solids separator, either with a sedimentation chamber (vortex) or via a pressurized unit or a pressurized canister filter, of either downflow design (may contain sand, pebbles or plastic media) or upflow design, with floating bead filters. As to biological filtration, these can be independent units, but most are combined with mechanical filtration. It is important to always identify the biological portion of the filter. Concerning circulation are the mechanism, pattern and standard parameters to know. As to the mechanism, active circulation is created via pumps (either external or submersible), fountains and air lift via a bubbler. These last two methods interfere with the settlement of suspended solids and are less desirable when filter systems are used. Circulation can also be passive, where a pond deicer is used in winter. This method creates a vertical circulation, so should be placed directly over where the fish congregate and should not be combined with any other circulation mechanism. The parameters to know are the flow rate, which is described as the "turnover time". It is the system volume divided by the true output of the pump. This is the advertised output minus loss due to total dynamic head (discharge height + pipe friction). It is important to determine the true output (GPH) by examining the return(s). For warm season flow, the rate in shallow ponds should be ½- 1 hr., in deep ponds, 1-2 hrs. In winter, the turnover time should not be less than 10 hrs. As to the circulation frequency, it should be continuous.

WSV18-0196

USE OF ACUPUNCTURE POINTS IN EMERGENCIES

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USE OF ACUPUNCTURE POINTS IN EMERGENCIES

Introduction
Animal emergencies can be life-threatening and require immediate treatment or attention.

Objective
In extremely serious cases, the objective is to quickly resuscitate the animal or bring it away from danger.

Methods
One can consider the use of acupuncture at various acupuncture points in an emergency on a case-by-case basis.

Results
In most emergency cases, animals respond well to the emergency treatment via acupuncture.

Conclusion
The examples show that acupuncture can be very effective during emergency situations and is safe when used in the correct manner.

WSV18-0124

TIPS FROM THE EXPERTS FOR THE APPROACH OF...
OPTHALMOLOGIC EXAM

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Tip from experts to maximise your ... Ophthalmic exam

An ophthalmic examination should not be a daunting experience. Interpretation of examination findings may be challenging; however, the examination follows a logical,anatomical order.

In my opinion a basic ophthalmic exam is similar to performing a minimum database ie stool exam , urinalysis and so being I am going to focus on a number of basic and essential examinations in sequential order.

In this presentation I will place more emphasis on simple and objective assessment tools , and non ophthalmic devices that would be applicable the moment you get back to your practice. Following which I will briefly run through an ophthalmic exam using a number of advanced ophthalmic tools for those interested to refer cases to a veterinary ophthalmolgist.

I. OPHTHALMIC EXAMINATION

A. Distance Examination.

We first start off the examination paying close attention to the animal at a distance walking into brightly lit examination room with the client. We pay attention to the size, discoloration, symmetry of the eye and its periocular tissues. The behaviour and mentation of the pet is taken into consideration. Not to mention;, a good history should always be taken.

Ie: prophylactic remedies, ivermectin , etc

B. Vision Evaluation

A blind animal may exhibit high stepping, collision with objects, a stare-like expression, or reluctance to move in a strange environment. The owner's impression that the animal "sees" well at home must be interpreted cautiously. Animals can "memorize" their own environment. The animal is permitted a few minutes to adjust to the room and observed as the history is obtained.

The patient's vision can be further evaluated by noting the response to hand movements (menace reflex), bright lights (dazzle reflex) or to cotton balls tossed into the visual field (tracking reflex). The menace response and the visual placement reaction can also be performed to evaluate the vision. I recommend that each contralateral eye should be evaluated separately by covering the ipsilateral eye it with one hand.

The vision examination should be performed in normal light, then in dim light to assess day and night vision respectively. If you can see the cotton balls or the obstacles of the maze test, the dog or the cat should be able to see them better than you since their night vision is more developed than ours. Cats generally do not menace test well, but respond well to bright light stimulation, laser lights, and cotton ball testing.

C. Ocular Examination

Based on the sensitivity of each test , an orderly sequence of diagnostic tests must be followed based on the special requirements of each test. For example ;

The evaluation of the tear film (Schirmer tear test) must be done before the eye is manipulated or any drugs are instilled.

Cultures of the external ocular structures must be done before extensive cleaning is done and before drugs are instilled.

The use of mydriatics is necessary for examination of the lens and posterior segment, but should not be given prior to measuring the intraocular pressure (IOP). The intraocular pressure evaluation requires topical anesthetic and must be recorded before excessive manipulation or before the patient becomes restless and excited.

D. Periocular examination: Orbit and adnexa

Examinations of anatomic structures should begin with the orbit and other periocular tissues. Orbits are evaluated for symmetry, eye-orbit relationship, deformities or enlargements. Because of marked variations in eye position of different breeds, one should be acquainted with the various breed characteristics. The extremes of variation in eye position can be represented by the relative enophthalmia of the collie and the exophthalmia of the Pekingese.

The presence or absence of strabismus and nystagmus is noted. Esotropia (crossed-eyes) is inherited in Siamese cats but in dogs may represent severe intraocular or neurological disease. Nystagmus occurs frequently in Siamese, apparently not always associated with clinically detectable vision defects, but in dogs may result from congenital intraocular diseases, or acquired vestibular or cerebellar diseases.

The eyelid position may be helpful in determining relative globe size. Looking from over the top of the animal's head helps to estimate globe position. Additional evaluation of the orbit consists of examination of the mouth (floor of the orbit), palpation of orbital rim, retropulsion of the globe, and evaluation of nasal patency, if necessary.

Special examinations such as standard skull radiography, orbital angiography, ultrasonography, CT and MRI, and surgical exploration may be necessary for a thorough evaluation.

*** For the rest of the exam MAGNIFICATION is extremely important to identify changes or pathology. Without magnification impacted meibomian glands, ectopic cilia, distichia, corneal vessels and other subtle changes will be missed. A simple otoscope head with a magnifying lens and bright light source works great. Other magnifying glasses or headloupes are also available.

E. Eyelids

The eyelids are examined for abnormalities of position, function and structure such as lagophthalmos, ptosis, trichiasis, ectropion, entropion, blepharitis, lid neoplasms, etc.

The blink reflex should be evaluated. The efferent limb of this reflex requires the integrity of the facial nerve (CN VII) and the orbicularis oculi muscle. The afferent limb may be a menace (CN II), corneal sensation (CN V) or touch sensation to the periorbital skin (CN V). Rapidity and completeness of the blink should be evaluated.

The lower and upper eyelids should touch the globe. Lower lid-globe contact is important to prevent accumulation of tears and debris. The lower "lacrimal lake" may be grossly distorted by anesthetics and tranquilizers. Cilia or eyelashes occur mainly on the dog's upper lid in three irregular rows. The lower eyelids of dogs and both eyelids of cats are usually void of cilia. The eyelid contours are regular and gently curved, partially exposing the openings of the tarsal or Meibomian glands (gray line). The duct orifices are frequently raised and nonpigmented. Aberrant cilia (distichia) may emerge from the spaces among the Meibomian gland ducts, or the actual duct orifices. Ectopic cilia emerge from the within the palpebral conjunctiva of the upper lid and are frequently the same color as the dog's hair coat. They can escape detection without careful examination.

F. The Conjunctiva and the Nictitating Membrane

The palpebral conjunctiva is examined by manual eversion of the upper and lower eyelids. Excessive lymphoid follicles, increased vascularity, foreign bodies, ectopic cilia, obstructed tarsal glands, hemorrhage, lacerations, abnormal growths and edema (chemosis) may be abnormalities observed. Coloration of the conjunctiva can be used to assess the presence of anemia and icterus. Because the palpebral conjunctiva is transparent, chalazia or impacted Meibomian glands appear as slightly raised yellow masses.

Examination of the palpebral (outer) and bulbar (inner) surfaces of the nictitans is important for diagnosis of several common external ocular conditions. Frequent abnormalities are eversion of the cartilage of the nictitans, prolapse of the gland (cherry eye), foreign bodies, follicular conjunctivitis, enlargement of the secretory gland, foreign bodies, follicular conjunctivitis, and enlargement of the bulbar lymphoid tissue.

G. The Sclera

The sclera should be scrutinized for change in color, abnormal masses, and tears or lacerations. Small vessels in the episclera are usually visible and occasionally a large vortex vein (especially the dorsolateral vein) can be seen. Enlargement and congestion of the episcleral veins occur commonly with glaucoma. This venous enlargement remains even after the glaucoma is "controlled". Hyperemia of the episcleral vessels occurs in association with inflammatory conditions. The "ciliary flush" or limbal hyperemia from iridocyclitis is usually less affected by topical phenylephrine while that associated with the conjunctivitis will usually blanch. The perilimbal scleral vessels are small straight and immovable vs larger mobile and branching conjunctival vessels.

H. The Cornea

Corneal sensitivity (corneal reflex) is tested by a small wisp of cotton gently touched to the cornea. (This must be done prior to topical anesthetic instillation). If the animal sees the stimulation, you will get a false positive.

The cornea is normally transparent, avascular, moist, and unpigmented with a smooth, even contour. It should be carefully examined for loss of transparency (edema or infiltrates), opacity, vascularization, pigmentation, dryness, growths, foreign bodies, lacerations, changes of contour, and ulceration.

Two types of vascularization occur in the cornea: superficial and deep. Superficial vessels occur in the anterior one-half of the corneal stroma, are usually continuous with visible conjunctival vessels, are "tree-like", and associated with external corneal diseases. Deep vessels appear as small, fine vessels in the corneal stroma that extend from the anterior sclera or deeper limbal vessels (paint brush border), and are associated with intraocular inflammation.

Examination of the cornea is incomplete without utilization of topical ophthalmic stains. Fluorescein is used to demonstrate the presence or absence of corneal ulcers. For topical use, fluorescein impregnated paper strips are preferred to fluorescein solution to insure sterility.

Because the water-soluble fluorescein stains the preocular film, a faint green may occur on the corneal surface.

The corneal epithelium is lipid-selective and prevents any appreciable corneal penetration by fluorescein. In the presence of a corneal epithelial defect, the dye rapidly diffuses into the corneal stroma. An area of fluorescein retention by corneal stroma is indicative of an epithelial defect (a corneal ulcer/erosion).

Rose bengal is a valuable stain in the evaluation of

the health of the corneal and conjunctival epithelium. It produces a brilliant red coloration of any dead or degenerating cells, and indicates defects in the mucin layer of the tear film. Rose bengal is retained by the cornea and conjunctiva in early fungal keratitis, keratoconjunctivitis sicca, pigmentary keratitis, exposure keratitis, viral keratitis, and certain other corneal ulcers.

I. The Anterior Chamber and the Iris.

The cardinal signs of uveitis is the "aqueous flare ".Increased protein in the aqueous humor, when viewed with a focal light source, gives the appearance of a light beam passing through smoke. Its appearance results from the optical Tyndall phenomenon.When checking for flare also compare the depth of the anterior chamber between the two eyes.

The iris is examined with a focused beam of light and magnification for color, shape, pupil size, surface, and movement. Iridal color in dogs varies from dark brown to blue, and generally 3 "zones" of color are evident (pupillary margin, iris collarette and the iris base). Light brown irides occur in many breeds, such as the Brittany Spaniels, German Short Hair Pointers and other breeds. Iridal heterochromia is not uncommon in white cats, St. Bernards, Great Danes, Beagles, merle Collies, Australian Shepherds, Old English Sheepdogs, Dalmatians and the merle Sheltie. Iris color in cats varies from blue to yellow-green to brown. In acute iritis, the iris may appear congested and swollen with loss of detail, and it may become darker in appearance with chronicity.

J. The Lens

The lens, which is normally a transparent avascular structure, should be examined for opacities (cataracts), position, presence, and size. Focal cataracts should be localized within the various parts of the lens as prognosis and etiology may be suggested by location. Nuclear cataracts are usually stationary while those affecting the equator or posterior cortex are often progressive. By slit lamp biomicroscopy, the canine lens may contain focal imperfections that are not "cataractous." Early cataract formation, evidenced usually as focal crystallization, vacuoles and water clefts, can be detected long before visual disturbances occur.

Localization of focal cataracts is performed by "retroillumination " of the fundus. Refractive light retroilluminated that is blocked out refers to a cataract formation. Location of a cataract may give clues about its cause i.e., inherited or associated with PRA.

Nuclear sclerosis of the lens begins to develop in dogs around 6 years. Biomicroscopic examinations can detect refractive changes between the lens nucleus and cortex as early as three years of age in dogs. Refractive light retroilluminated that is NOT blocked out and is complete and round confirms nuclear sclerosis. This is frequently mistaken for cataract formation in older animals by owners and veterinarians.

K. The Vitreous

The vitreous humor is normally a clear gel. The anterior portion can be examined using focal illumination and some magnification. The posterior aspect of the vitreous is examined by ophthalmoscopy or the slit lamp biomicroscope with added lenses. Frequently seen vitreous abnormalities include vitreous strands, asteroid hyalosis, hemorrhage and infiltration with inflammatory cells. Small remnants of the hyaloid vasculature (seen as white strands) are frequently encountered behind the central posterior lens capsule in the vitreous immediately posterior to the lens. Liquefaction of the vitreous is called syneresis, and opacities that occur in the liquefied state are called "synchysis scintillans". These opacities often rise and fall in the vitreous as the eye moves.

Differentiation of lens and vitreous opacities may pose a problem for the clinician. Localization of intraocular opacities can be achieved by noting direction of movement in relation to the center of the globe, or by slit lamp biomicroscopy. The first procedure is convenient and assumes the center of rotation of the eye is the posterior aspect of the lens nucleus in the dog. Opacities which are anterior will move with eye movement; for example, an anterior cortical cataract will move left when the eye turns left. Opacities posterior to the center of rotation will move in the opposite direction. In the horse the optical center of the eye is the posterior pole of the lens. The stability of the opacity may also help to differentiate lens from vitreous. Lens opacities are fixed and remain stationary when the eye stops moving. Vitreous opacities tend to move slightly or oscillate within the gel vitreous after eye movement ceases.

L. The Fundus

The ocular fundus is examined last and requires direct and/or indirect ophthalmoscopy. Although the fundus can be viewed without drug-induced mydriasis, dilation of the pupil greatly facilitates examination of the complete ocular fundus. The ocular fundus is examined for changes in the normal appearance, detachment of the retina, chorioretinal hypoplasia or dysplasia, vascular patterns, attenuation, congestion, hemorrhage, colobomas, scars, alteration in coloration, changes in pigmentation and foci of inflammation. The optic disc should also be examined for size, shape, color, masses, and pits or colobomas. Swelling and inflammation of the optic disc occurs with optic neuritis, which is characterized by blindness. Myelination of the disk must be differentiated from swelling of the disk.

II. Special Diagnostic Procedures

A. Pupillary Light Reflexes (PLRs)

The size of the pupils are evaluated and the direct and consensual pupillary light reflexes are tested. This should be done with a bright light in a dimly lit room. The pupillary light reflexes are affected by the psychic state of the animal, room illumination, age, many topical and systemic drugs and the intensity of the light stimulus. Older animals may exhibit slow and incomplete pupillary light reflexes resulting from atrophy of the iris sphincter muscle. This is common in small dogs, especially poodles. The pupillary margin may have an irregular or scalloped appearance. Incomplete iris atrophy may give an irregular pupil shape.

The rapidity of pupillary light response, extent of miosis and ability to maintain miosis to constant light stimulation are evaluated. The consensual pupillary reflex is normally equal to the direct. The pupillary light reflexes require integrity of retinal neural cells, optic nerves, optic chiasm, optic tracts, midbrain (Edinger-Westphal nuclei), parasympathetic fibers via the oculomotor nerve, ciliary ganglia and the iridal sphincter musculature. The reflex is subcortical and should be considered an evaluation of the retina and optic tracts, not of vision.

Drug induced mydriasis is not used indiscriminately. The instillation of mydriatics is avoided in animals with predisposition to, or overt glaucoma, and lens luxation. Young puppies dilate slowly, often incompletely, and may require multiple drops. Mydriasis produced by darkening the room may permit a cursory but not complete examination of the ocular fundus. 1% Tropicamide (Mydriacyl-Alcon Laboratories) provides mydriasis within 15 to 20 minutes in a normal eye.

B. Corneo-Conjunctival Cultures And Cytology

Corneo-conjunctival cultures and cytology are especially valuable in chronic, severe and non-responsive external ocular conditions. The cultures should be done before any administration of drops, since many of the drugs contain bacteriostatic agents. Topical anesthetics are used prior to the collection of cytologic material.

To obtain a specimen for cytologic examination topical anesthetic is instilled 2-3 times over a few minutes and the animal's head and muzzle are held firmly by the assistant. To obtain a conjunctival scraping, the lower eyelid is everted and the ventral conjunctival surfaces are vigorously rubbed with a stainless steel or platinum spatula. The collected material is distributed onto glass slides. Ideally, conjunctiva should be scraped vigorously enough to obtain basilar cells without inducing hemorrhage. To obtain a smear of exfoliated cells, a moistened dacron tipped applicator is rubbed along the conjunctival cul-de-sac and then rolled on glass slides. The specimens are stained with new methylene blue, Gram's, Wright's, Giemsa's, or modified Sani's methods.

C. Nasolacrimal System and Tear Production

The nasolacrimal system and preocular tear film are evaluated by considering both the secretory and excretory components.

Schirmer Tear Test

The precorneal tear film is essential in maintaining normal corneal health. Measurement of tear production is an important diagnostic test when deficiency of the lacrimal system is suspected.

The tear-producing system is evaluated qualitatively by the Schirmer tear test. The diagnosis of "dry eye" or keratoconjunctivitis sicca (KCS) may be missed if the Schirmer tear test is not routinely used. The Schirmer tear test measures only the aqueous aspects of tears. Currently, aqueous tear production is most commonly measured using the Schirmer tear test.

Schirmer Values:

Dog: 21.9 +/- 4.0 mm wetting/minute

Rabbit: 5.3 +/- 2.9 mm wetting/minute

Cat: 20.2 +/- 4.5 mm wetting/minute

The round end of the test paper is bent while still in the envelope and positioned without contamination in the lacrimal lake at the junction of the lateral and middle thirds of the lower eyelid. The animal usually closes its eyelids during the test. After one minute the paper is removed and measured on a millimeter scale on the paper envelope. The STT strip should be left in position for one minute. It is not a linear test, so if you obtain a value of 7 mm/30 seconds this does not mean it will be 14mm/min!!!! If you get an abnormal value <15mm in less than one minute the test should be repeated leaving the strip in for a full minute.

Phenol Red Thread (Prt)

The Phenol Red Thread Test is a new, fast and equally accurate method to assess tear production.

In the PRT tear test, the thread is 75 mm long and is impregnated with phenol red, a pH-sensitive indicator. A 3 mm indentation at the end of the thread is inserted into the inferior conjunctival sac for 15 seconds. The alkaline tears turn the pale yellow thread red.

A test time of 15 seconds is required compared to the 5 minutes needed for the STT in humans or the 1 minute in dogs.

Anesthesia is not necessary for the PRT tear test because the subject has little or no sensation from the thread. It is theorized that the minimal sensation and short test time give a more accurate indicator of the volume of residual tears in the inferior conjunctival sac of the eyes.

Mean length of absorption for the PRT tear test in cats is 23.0 mm ± 2.2 mm/15 seconds. The normal range in cats for the PRT tear test is 18.4 to 27.7 mm/15 seconds.

In dogs the mean length of absorption using the PRT tear test is 29.7 to 38.6 mm/15 seconds.

Tear Drainage

The excretory component of the nasolacrimal system is evaluated by the presence or absence of medial canthal tearing; passage of fluorescein instilled onto the eye; nasolacrimal flush; catheterization of the entire system, and by dacryocystorhinography. The nasolacrimal drainage apparatus consists of two puncta and canaliculi, a poorly developed nasolacrimal sac and the nasolacrimal duct. The oval puncta are situated in the upper and lower medial eyelid margins about 1 to 2 mm in the palpebral conjunctiva. A partial to complete ring of pigment may surround the puncta and facilitates their detection.

Passage of fluorescein from the eye to the external nares is a reasonable test for patency of the nasolacrimal system. A strip of fluorescein is moistened with a few drops of sterile eyewash and touched to the upper bulbar conjunctiva. The dye usually appears at the external nares in 3 to 5 minutes. Both sides should be performed at the same time to compare passage times. Ultraviolet light enhances detection of the dye. Fluorescein passage in brachycephalic dogs and is not reliable as the dye may exit more readily into the nasopharynx. The animal's tongue and saliva should be examined with a UV light in these cases.

The nasolacrimal flush determines patency of the system and the treatment of many of its disorders. The upper punctum is cannulated with a 22-23 g blunt lacrimal needle or 22-24 gauge teflon catheter under topical anesthesia. Tranquilization or general anesthesia is seldom necessary for the dog but often necessary for the cat. A 2 to 3 ml plastic syringe with sterile saline is used to inject the solution through the upper punctum, canaliculus, nasolacrimal sac, lower canaliculus and out the lower punctum. Once this "arc" is established, the lower punctum is compressed digitally and the solution is forced through the nasolacrimal duct and out the external nares. If the dog's head is positioned upward, the dog will swallow or gag on the solution. Excessive pressure should be avoided to minimize the danger of rupturing the N-L system above an obstruction.

D. External Ophthalmic Stains

Fluorescein

Examination of the cornea is incomplete without utilization of topical ophthalmic stains. Fluorescein is used to demonstrate the presence or absence of corneal ulcers. For topical use, fluorescein impregnated paper strips are preferred to fluorescein solution to insure sterility.

Rose Bengal

Rose bengal is retained by the cornea and conjunctiva in keratoconjunctivitis sicca, early fungal keratitis, pigmentary keratitis, exposure keratitis, viral keratitis, and certain other corneal ulcers.

E. Intraocular Pressure Measurement (Tonometry)

Intraocular pressure (IOP) is estimated digitally, and measured by Schiotz tonometry or applanation tonometry. Subtle elevations in intraocular pressure, repeated measurements of glaucomatous eyes under medical treatment, or after surgical intervention require instrument tonometry.

Applanation tonometers (especially the Tonopen type) are very accurate and easy to use. Applanation tonometers are becoming more common in practices. The Tonopen applanation tonometer has made it much easier to diagnose and treat the animal glaucomas.

IOP is 16.8 ± 4.0 mm Hg in dogs;

20.2 ± 5.5 in cats; and

23.2 ± 6.9 in horses.

F. Ophthalmoscopy

1. Direct Ophthalmoscopy

Direct ophthalmoscopy is used more frequently by practitioners than indirect ophthalmoscopy. However, both techniques have advantages that complement each other when used together. The method is termed "direct" because a condensing lens is not interpositioned between the ophthalmoscope and the patient's eye. The examiner has a direct optical image of the patient's eye. The fundus image is real, upright and approximately about 17 to 19 times magnified in dogs and cats. The fundus area visualized is about 10 degrees or approximately 2 disc diameters.

The direct ophthalmoscope head also offers a range of lenses to enable focusing at various depths within the eye. These lenses are calibrated in diopters. A lens with a power of 1 diopter will focus light from an infinite source (parallel rays) at 1 meter. The higher the diopters, the more converging power the lens possesses. Negative diopters denote diverging lenses. When an emmetropic eye (observer) looks into an emmetropic eye (patient) with an ophthalmoscope the retina of the patient should be in focus at the 0 diopter setting. Minor lens corrections are usually needed to focus on the patient's fundus. Within the eye, a distance of 3 diopters equals 1 mm.

In performing ophthalmoscopy, the patient's body and head are minimally restrained by an assistant. The examiner holds the muzzle and/or lids with one hand and with the other hand holds the ophthalmoscope to make the necessary diopter changes. It is preferred to view the tapetal fundus several inches from the patient and then

move to 1 to 2 inches from the patient's eye when the optimum focus is achieved and the animal has adapted to the restraint. The diopter setting is usually started at "0" and adjusted to between +3 to -3 diopters to provide the sharpest image possible. By using more positive lenses the lens can be seen at +8 to +12 diopters and the cornea at +20 diopters.

Direct ophthalmoscopy has certain limitations. Penetration of cloudy or partially crystallized media is limited. Because of magnification, there is a small field of view. Examination of the peripheral fundus is difficult. There may be difficulty in compensating for refractive errors and eye movements. Stereopsis is absent, and depth of focus is limited. The small working distance between examiner and patient may be hazardous to certain species of animals.

The PanOptic ophthalmoscope is available and provides an intermediate level of magnification to the direct and indirect techniques.

2. Indirect ophthalmoscopy

Indirect ophthalmoscopy complements direct ophthalmoscopy. To perform indirect ophthalmoscopy a fairly bright light source is directed into the eye. A condensing lens is interposed between the light source and the eye. Incident light is condensed to illuminate the fundus. The reflected light then is condensed by the same lens to form a virtual, inverted, and reversed image between the lens and the light source.

The advantages of binocular indirect ophthalmoscopy are penetration of cloudy media, large field of view (hence an excellent survey instrument), examination of the peripheral fundus, ease of compensation of refractive errors and eye movements, stereopsis, greater distance between examiner and patient, two to three simultaneous observers and the ability to readily examine the more intractable patients with less hazard to the examiner. The disadvantages include less magnification for studying particular areas, and the need for drug-induced mydriasis.

Indirect ophthalmoscopy can be employed with only a light source and a lens. Several commercial indirect ophthalmoscopes are available. Regardless of the light source used, the power and type of lens used determines the ease and accuracy with which the fundus exam will be conducted.

The indirect ophthalmoscope is adjusted so the light is slightly off center of the examiner's visual field (to reduce glare). The patient's muzzle is held gently and the lens is positioned three to five cm from the cornea and the upper eyelid retracted. The lens is usually held close to the cornea initially to permit observation of the ocular fundus and then moved away from the eye until the image is maximum size. When the hand lens is

interposed between the light source and the eye, the fundus is visualized. Image magnification (2X to 4X) is dependent on the dioptric power of the hand lens. The +20 lens is the most versatile. Occasionally, an annoying light reflection occurs and is remedied by slightly tilting the hand lens.

Image magnification is dependent on the dioptric power of the hand lens. The +20 D lens is the most versatile.

H. Ultrasonography

Ultrasonography has become increasingly useful in the diagnosis of intraocular disease. High frequency sound waves are directed through the eye. A portion of these sound waves "echo" off tissue interfaces. These echoes are amplified and projected onto an oscilloscope. Echoes from the corneal surfaces, the anterior and posterior lens surfaces, the retina, and any abnormal intraocular material will project an image which aids intraocular diagnosis. This is especially useful when dense corneal opacity or mature cataract obscures the view of the fundus.

WSV18-0012

FELINE FOCUS (SIMULTANEOUS TRANSLATION INTO MANDARIN CHINESE)

RESPECTFUL CAT HANDLING VS. CAT WRANGLING: PUTTING PURRSPECTIVE IN YOUR PRACTICE

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Cats are becoming increasingly kept as companions around the world. There are 75 million pet-owning homes in Europe. In these homes are kept 72 million cats, 63 million dogs 40 million caged birds, 22 million small mammals and 5 million ornamental fish. (FEDIAF Facts and Figures 2014) Despite more cats than dogs being kept as companions, feline veterinary visits lag behind those for dogs in numbers/pet, total numbers and amount spent per animal. Most people bring their cats to the veterinary clinic in the first year after adoption. After the initial visit(s), most people only bring their cats in when they are ill with less than half bringing them in for vaccines. This leads to two questions:

1. Why don't cats visit the veterinary clinic as often as dogs?

2. What can we do to improve the experience for the cat, the client and the clinic team?

Clients may consciously or subconsciously be reluctant to bring their cats to the clinic because they:

1. Falsely believe that cats are low maintenance and that indoor cats do not need regular preventive health care.

2. Don't recognize how subtle the signs of sickness are in this species.

3. Feel guilty, stressed or even fearful about travelling with their cat, seeing their cat upset in the veterinary clinic, worrying about their cat's response when they return home and fear about their ability to perform prescribed treatments.

4. Don't like paying for something they don't really want to do.

In addition, many clinic team members are less comfortable handling cats and fear being hurt by a self-defensive patient. This often translates into escalation of defensive handling, use of sedatives and fewer recommended recall visits.

In order to change this negative picture, we have to recognize the essence of a cat. Cats are prone to self defensive behaviours because they are not just predators (towards small birds and rodents) but are prey to any other animal. Because they have evolved to eat small frequent meals, they hunt alone and eat alone. As a consequence, they need to define and maintain a resource-based territory. Being self-dependent makes them very vulnerable: should they become injured, no one will nurse them or feed them. Elaborate and dramatic body language and vocalizations encourage potential threats (other cats, predators and people) to keep their distance...in order to prevent the cat from physical harm. Rather than being aggressive, our patients are being self-defensive. When a cat is showing fear-based behaviours, distract him/her, identify and reduce the (likely) perceived threat.

Cats need to feel in control of their situation in order to feel safe. If frightened, they will flee, freeze or fight. When handling them, allow them to feel that they have the chance to flee by not blocking them completely, by keeping as many paws on the ground as possible and giving them space. Assess the clinical facility to identify what smells, sights, sounds, textures and tastes are threatening to cats and make small chances to reduce these threats. Be aware of the patient's (and client's) experience and adapt accordingly. Allow the cat to acclimate to a safe and quiet consultation room. Perform diagnostic tests in this safe space rather than taking the cat "to the back", a new, frightening environment.

Wellness programs are useful ways to educate and provide a culture of lifelong preventive healthcare. A first year of life program sets the stage for good preventive care, but prescheduling subsequent annual visits through adulthood and screening visits for senior life-stages provides appropriate ongoing care. All cats require vaccination and prophylactic parasite treatment whether they have access to the outdoors or not. Many clients have other animal companions at home that we are unaware of. Perform a pet inventory by asking them if they have any other pets; this allows us to provide preventive healthcare for those animals as well.

Educate regarding the subtle signs of sickness: (http://www.haveweseenyourcatlately.com/Health_and_Wellness.html)

1. Inappropriate elimination

2. Changes in interaction

3. Changes in activity

4. Changes in sleeping habits

5. Changes in eating and drinking (manner as well as amount and frequency)

6. Unexplained weight loss or gain

7. Changes in grooming as well as hairballs

8. Signs of stress/distress

9. Changes in vocalization

10. Bad breath

Travelling to the clinic does not have to be stressful.

Teach all clients to have a carrier that they feel comfortable keeping in the house where the cat can use it as a safe and favoured space. Transport the cat in the foot well of the car for optimal security, keep the carrier horizontal with minimal rocking/sliding, cover it with a towel and keep it off the floor of the clinic to avoid interactions with other animals. (www.cathealthy.ca)

The more relaxed the cat and clinic team are, the more relaxed the client will be.

SOCIAL MEDIA FOR VETERINARIANS

TOP 10 BEST SOCIAL MEDIA TIPS

E. Garcia[1]

[1]*Simply Done Tech Solutions, CEO- Digital Strategist, Tampa, USA*

TELL YOUR STORY

People are often under the impression that Facebook is solely about peer-to-peer interactions. This, however, couldn't be further from the truth. Facebook is a platform that's become as universal as the water cooler itself. Successful veterinary practices around the **world leverage Facebook as a place to tell their unique story.** Your veterinary practice has a narrative; a year it was founded, a founder (or two, or more) and a style and perspective that makes it entirely unique.

Use Facebook to tell your story and not only capture, but captivate your audience!

Tell us about your success stories: the pets that you care for and the difference that you've made today. All of these things foster community, trust, interactions, and keep your trusted pet owners coming back for more.

These success stories are technically known as:

Case studies – a story particular to a specific pet, place and time.

These case studies are of crucial importance for a multitude of reasons, but primarily because they help your audience to see firsthand the type of stellar care that your veterinary practice provides!

In a particular case study, be sure to provide your audience with:

- Why the pet came in to receive veterinary care

- What you did to provide care for the pet

- How the pet is doing today

- A photo, or quick video of the pet!

By providing this level of in-depth information on a pet, you tell the story of your patient and ensure that you can deliver the same quality of care to any prospective pet owner who needs it. You'll be able to forge an immediate bond with pet owners who appreciate your attention to detail, and the accountability needed to provide optimal care for a pet.

People want to hear of your successes, which will brighten their day and instill them with confidence about your veterinary practice. In exceptional circumstances, news coverage has even come about after particularly sincere and uplifting pet stories. This results in absolutely

tremendous publicity, and simultaneously helps you to market your services to a wider audience. This wider audience can soon grow and enhance your veterinary practice online, and in the local community.

Case studies are also a great opportunity to educate your clients. By highlighting a particular toxicity (like xylitol, grapes, or lily toxicity in a cat) you can spread the important information in a success story that will resonate with pet owners. These posts can be timed for specific times of year (the "chocolate holidays", the start of flea season, holiday dangers) to help your clients stay aware of how to best care for their pet, and to keep your practice at top of mind.

GET PERMISSION

Yes, you should receive permission from a pet owner to share their story, pictures or a video of their pet on Social Media or elsewhere. This is an important thing to note and emphasize, as some members of your staff may be appointed to collect signed **Photo/Video Release Forms**, to ensure that you're permitted explicitly to share various types of media.

Most pet owners don't hesitate at the opportunity to share the joy of their pet with the world and online, but **receiving permission firsthand is definitely a must.**

Sample topics for case studies can include:

- **Dermatology:** Before and After Skin Cases

- **Dental:** Before and After Dental Care (Photo)

- **Surgical Case Examples**

- **Laser Therapy Cases** (Pets can often improve a limp in a matter of weeks after laser therapy)

By using Facebook, photos, and videos to create and communicate compelling stories, you can enhance your marketing efforts, stay on the cutting edge, and attract more pet owners to your veterinary practice.

MAKE MARKETING A GROUP EFFORT

Who says that you should only appoint a single point person as your lead marketer? After all, your veterinary practice is a team of unique individuals; creating a unified experience by combining the many talents of a diverse team. If you utilize this same diversity for your marketing, you may be able to generate far more content, that's actually far more compelling.

Read on to see how the team-oriented approach to marketing can actually help you generate content more consistently, and enforce more accountability in the process.

ENHANCE CONTENT AND CAMARADERIE IN ONE FELL SWOOP

Yes, there is some peace of mind that comes with appointing a head of marketing, or other point position, but let's look at what can happen when instead, the effort is spread across a team.

- Rather than making a new hire solely for marketing, spread responsibility among a diverse staff that is interested in participating!

- A team can create a rotation of marketing and social media related tasks, easing the responsibility on everyone.

- Sharing responsibility can create accountability and a sense of true shared-identity related to your veterinary practice! When members of your staff get to share a piece of their experience on social media or otherwise, they are supporting the practice and giving their unique point of view in the process. This can be very empowering for your staff!

- Appropriate rotations can involve each member of staff taking photos to share via social media, and creating a schedule of posts for your website, Facebook, Twitter, etc.

- Content can be diverse and customized to fit the tone and feeling of your veterinary practice! You may decide to include staff photos, quotes, pictures of happy pets, stories, health tips, or more. All of these things help to convey a true sense of identity for your veterinary practice, and exhibit your practice as a place of both work and community.

- Consider designating tasks based on skillset; maybe a staff member that studied photography touches up shots before they are posted to Facebook. Perhaps an English minor helps to proofread content before it goes live. Get creative!

- By working as a team, you can share responsibility, save money on a new hire, and teach your entire veterinary staff the various skillsets that it takes to remain competitive in an evolving veterinary industry!

- Many veterinary practices learn to enjoy the shared approach, creating a sense of camaraderie amongst staff, while enhancing their veterinary practice's marketing efforts in the process.

THE ROUND TABLE APPROACH

There are many ways to facilitate this group effort. The "Round Table" approach means that at least once a week, your staff gets together to discuss the strengths of your approach to marketing, what can be improved, and any other topics that may be deemed appropriate. You can facilitate this approach through a group email where everybody responds, or a lunch where everybody gathers around, yes, a round table! The details are up to you, but your staff will love a chance to be heard and enhance your veterinary practice.

Some ideas for sharing ideas amongst your staff include:

- A weekly lunch where some of the top social media posts are shared to the staff or read aloud.
- A schedule or white-board that helps to delegate social media or marketing tasks amongst a team
- An email thread that encourages everybody to participate and chime in with tips for improvement, or accomplishments experienced over the course of the week.
- Anything else that may excite or motivate your staff to share the stories and perspective that are a unique piece of your veterinary practice. When marketing becomes a group effort, everybody wins!

How to Implement a Social Media Calendar

"Engage, Enlighten, Encourage and especially…just be yourself! Social media is a community effort; everyone is an asset." - Susan Cooper

The trend alone says it all. When you look at Social Media use amongst adults, you'll see an upward, zigzagging line that's rising far steadier than the stock market. In fact, as of January 2014, it's estimated that 74% of all adults using the Internet are also using Social Networking sites as they navigate the web. What's more?

These numbers are still on the rise.

Whether you've already read my recommended article on the Rise of Social Media Marketing, or you're just now diving into the world of online media for the very first time, there's plenty that you need to know to be well-equipped, and I'm here to help you get started.

I'm going to show you how to use a Social Media calendar; a truly integral piece of the digital puzzle. It's not enough to simply log-on to a popular Social Media platform and start shooting off posts left and right via Facebook and Twitter. You've got to be methodical, organized and thoughtful to truly make the impact you're looking for. I can't stress this part enough; it's highly important to remain organized and deliberate when it comes to your overall marketing efforts. The Internet is flush with tweets, fun-facts, blog posts and millions of tidbits. Your posts must be well-crafted and inviting. For veterinary practices, this is a must.

There's another reality to consider. Veterinary practices are so busy focusing on patients and clients (rightfully so), that they often don't know what to post on Social Media, and what sort of content they wish to deliver in the first place! My recommendation? Don't make rapid-fire posts across a wide-range of topics in a short span of time. Instead, use a Social Media calendar to organize specific campaigns and focus on particular topics for longer periods of time. This has the added benefits of letting you explore your topic in-depth, allowing you to get your whole veterinary team on the same page and sparking more client engagement, which is a major goal behind utilizing Social Media in the first place.

Using a Social Media calendar is also a great chance for your management team, doctors, and staff to discuss, engage and truly understand what will be shared on a particular Social Media platform before it's actually posted publicly. It's important to remain unified as a veterinary practice, so members of your team should have a comprehensive understanding of which Social Media campaign is currently active and what topics are being shared with your target-audience. There's a pretty good chance you'll get follow up calls and engaged clients inquiring about major posts and presenting their own scenarios in kind. It's much better to have a receptionist respond with, "I'm so glad you loved the post! Yes, we're focusing on Dental Care and preventative medicine this month", as opposed to a scrambling, "I'm going to have to get back to you on that…"

Ready to get started with your own Social Media calendar? Good. Here are my top three tips to help you implement an effective Social Media calendar today:

Top 3 Tips for Implementing Your Social Media Calendar

(1) Share your Social Media calendar with your entire team. Before you begin presenting content to your community of followers, review content with your team to ensure everyone is well-versed on the topic at hand, and answer any questions that may pop up. Take this as an opportunity to poll team members about potential topics to focus on both now and into the future and refresh knowledge on existing protocols to get everyone on the same page. Your veterinary practice will benefit from this step and so will your audience.

(2) Gather statistics related to the topic at hand, and begin to craft well articulated, 2-3 sentence postings. Share direct case examples from within your practice, but don't forget to have client consent forms signed before posting specific case examples. I recommend case examples from your practice in order to help paint a vivid picture and tell your audience a story that they can related to. You'll be amazed at how many comments can come as a result of an effective, polished post.

(3) As mentioned previously, don't just fire off posts at a whim. When inspiration strikes, that's fantastic, but use it as an opportunity to bring your veterinary practice together and discuss. Use the native scheduling feature within Facebook to methodically schedule postings to go out automatically. If you are posting content to Twitter or other Social Media channels, tools like Hootsuite are free and immensely helpful for scheduling automatic postings. During slow months, use your Social Media calendar to write out content in advance and plan for future months. Even

if the content is scheduled to go out well in advance, be sure to monitor activity and engage with posts when necessary. Scheduling posts helps you to bring consistency to each campaign.

It's simple. Social Media is the way of the future, and the more you put into it, the more you'll get out of it. By using a calendar, you give your team an opportunity to get on the same page, and get ahead of the game when it comes to engaging your audience. A veterinary practice is only as strong as the staff and veterinarians behind the scenes. Use all of the tools at your disposal and you'll start to notice the difference.

Your audience is just waiting to engage with your veterinary practice. The only question left is, what will you share first?

WSV18-0327

SVA INFECTIOUS DISEASES

MANAGEMENT OF PARVOVIRUS INFECTIONS IN DOGS AND CATS

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Objectives. The primary objectives of this session are to learn updates on the diagnosis, treatment, and prevention of parvovirus infections in dogs and cats.

Canine and feline parvoviruses are non-enveloped DNA viruses which require rapidly divided cells to reproduce. Infections in dogs came from feline panleukopenia virus and emerged in the late 1970s. Currently, most worldwide dog cases with clinical diseases are infected with CPV-2b or CPV-2c. The small animal parvoviruses are quite resistant to environmental destruction but are susceptible to bleach. The primary means of transmission is horizontal transmission via oronasal – fecal transmission. Vertical transmission via in utero infection can occur and can leads to myocarditits. CPV-2b and CPV2c can also infect cats.

CPV-2 first enters the oronasal cavity and infects lymphoid tissue followed by viremia for at least 1-5 days. Rapidly dividing cells of the gastrointestinal tract, myocardium, CNS, skin, kidney and other organs are targeted. Most notably, CPV-2 infects the crypt epithelial cells causing villus blunting. Decreased absorption (manifested as diarrhea), necrosis (sloughing of blood) and inflammation result. Lack of gastrointestinal integrity allows normal GI flora to penetrate into the blood stream and can lead to bacteremia with or without sepsis. Canine parvoviruses are shed primarily in feces for 3 to 14 days post infection, often starting before clinical signs appear. Clinical signs usually develop starting 5 to 12 days after exposure. Dogs with maternal or vaccinal antibodies can usually limit viremia and fully immunized dogs have sterilizing immunity.

Any dog or cat can be infected, but disease is thought to be more severe in some breeds like the American Pit Bull Terrier and Rottweilers. Severity of disease depends on virulence of the strain, size of inoculum, age, breed, and host's defenses. Clinical signs of CPV or FPV infection are most severe in puppies or kittens less than 12 weeks that do not have prior immunity. Most dogs have enteritis characterized by foul smelling bloody diarrhea and vomiting. Leukopenia and fever are also common. Cats may have vomiting without diarrhea and sudden death. Both dogs and cats may also have signs of sepsis like red mucous membranes and can develop disseminated intravascular coagulation. CPV-2 can infect the primary CNS with resultant hemorrhage into brain or spinal cord. In utero infection or infection in pups

less than 8 weeks can lead to myocarditis and result in sudden death or congestive heart failure. Depending on the presence of prior immunity, some dogs or cats may have subclinical infections.

Dogs or cats under two years of age with acute bloody diarrhea should be considered at high risk for parvoviruses, particularly if the vaccine history is incomplete. Another differential diagnosis with similar clinical signs is salmonellosis; this should be considered in dogs or cats that look clinically like parvovirus, but are well vaccinated. The clinical diagnosis is usually supported by documenting parvovirus antigen in feces by ELISA or PCR assays which are commonly part of diagnostic PCR panels in the United States.

Treatments. Greater than 90% of dogs with CPV-2 enteritis will survive if administered supportive care shortly after clinical signs develop. Feline panleukopenia often has a higher fatality rate. Fluid replacement, electrolyte balance (particularly potassium), control of hypoglycemia, control of oncotic pressure (hypoalbuminemia can develop), treatment of bacteremia and sepsis (antibiotics), control of nausea and vomiting, and "feeding the gut" as early as possible are paramount to success.

Fluid therapy should be designed to correct losses, hyponatremia and hypokalemia. Oncotic pressure should be maintained with plasma transfusions, hetastarch, or related compounds. Broad spectrum antibiotics with like a first generation cephalosporins are often used in routine cases with therapy escalated to include drugs with a better gram negative spectrum in pets showing signs of sepsis. Injectable enrofloxacin or amikacin can be added to the protocol to enhance the gram negative spectrum. Many clinics use second generation cephalosporins like cefoxitin as their primary antibiotic as this drug has an enhanced gram negative spectrum compared to first generation cephalosporin. Recently it has been shown that maropitant can be used successfully as an antiemetic agent, but also lessens abdominal pain. It is important to "feed the gut" early in cases with enteritis and so at Colorado State University, nasoesophageal or nasogastric tubes are often used to start to deliver elemental diets as soon as possible. Highly digestible diets with or without probiotics are often used in the recovery phase.

A new gastrointestinal recuperation diet, Rebound Recuperation (Virbac) was found to be palatable, as determined by acceptance and preference testing, in healthy dogs during the preoperative and postoperative phases of routine sterilization (Forbes et al, 2015). In a followup study, Rebound Recuperation.was used successfully in a CPV clinical trial performed at Colorado State University (Tenne et al, 2016).

Many different adjunctive therapies like passive immune therapy (hyperimmune serum infections), granulocyte colony stimulating factors, oseltamivir (Tamiflu) are used to attempt to improve survival but has not been shown to be effective in controlled studies. Interferon omega has been beneficial in some puppies. Prognosis is variable. Intussusception may occur as a sequel to severe enteritis and so all dogs or cats should be palpated daily. Not all clients can afford hospitalization and intensive care. Thus, researchers at Colorado State University evaluated an out-patient protocol in dogs with CPV that had an 80% success rate (Venn et al, 2017).

Prevention and public health considerations. Extreme care should be taken to prevent spread to other animals by disinfection with bleach, separation from other hospitalized animals, and vaccination of other dogs in the household. No zoonotic potential is recognized; the parvoviruses of humans are species specific. Vaccination is very effective for both CPV and FPV. Modified live parenteral products are likely to break through maternal antibody interference and should be used for the puppy and kitten series. Vaccines should be administered until at least 16 weeks of age and one more booster after that time could be considered in high risk animals. After a 1 year booster, 3 year intervals for CPV and FPV vaccination is adequate for most animals. Use of serologic test results can be used after the 1 year booster to determine CPV and FPV vaccine need; positive animals do not need to be vaccinated if a validated test is used.

Suggested readings

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ONCOLOGY

SOFT TISSUE SARCOMAS IN DOGS AND CATS

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SOFT TISSUE SARCOMAS IN DOGS AND CATS

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Learning objective: Develop an approach to diagnosis of suspected soft tissue sarcomas in dogs and cats, including interpretation of histopathology reports. Understand the different possible surgical approaches, appropriate staging tests and indications for adjunctive treatments.

Soft tissue sarcoma (STS) is a generic term, encompassing a number of different mesenchymal tumour types. Generally, this term encompasses fibrosarcoma, peripheral nerve sheath tumour, myxosarcoma, perivascular wall tumour and liposarcoma. Tumours which are specifically excluded because they have different presentations and/or behaviour are muscle origin sarcomas, synovial sarcomas, haemangiosarcoma and lymphangiosarcoma.

Feline injection site sarcomas (FISS) are a specific subtype of soft tissue sarcoma characterised by history and location (growth at a site of previous injection), aggressive histologic features and often associated inflammation. Non-injection site related STS in cats should be managed as for canine STS, though evidence for prognostic factors such as grade and mitotic index is lacking.

Diagnosis for all soft tissue sarcomas typically requires histopathology. Cytology may be diagnostic or at least suggestive, but especially for well-differentiated sarcomas, cannot reliably distinguish between neoplastic and reactive mesenchymal tissue. Cytology is recommended as a first step to rule out other possibilities e.g. mast cell tumour. Histopathology may be obtained by incisional or excisional biopsy. In general, unless excision is achievable with wide margins, incisional biopsy for suspected soft tissue sarcomas should be performed to confirm the diagnosis and grade to plan the most appropriate surgery. Revision surgeries for incomplete margins following excisional biopsy are typically more extensive than what would have been required had an appropriate first surgery

been performed. For suspected FISS, referral should always be recommended, as radical excision is typically required and outcomes are improved if the first surgery is aggressive. Incisional biopsy should be considered to confirm the diagnosis, but excisional biopsy is NOT recommended for FISS.

The important features on histopathology report for canine STS (apart from diagnosis of STS) are:

1. Grade: A 3 tier scheme is typically used, based on degree of differentiation, necrosis and mitotic rate. However, the exact methodology for how these factors are assigned is not standardised and so may vary between pathologists. Grade appears to impact risk of metastasis and local recurrence, though confirming suspected metastasis or recurrence is inconsistently done in the published literature. Based on available studies, the metastatic potential is low (< 15-20%) for grade 1 and 2 STS, and up to 40-50% for grade 3 STS. Histologic grade is variably prognostic for survival in the published literature (1)

2. Mitotic index: What is typically reported in the veterinary literature as mitotic index should be more correctly referred to as mitotic count. However, for consistency, we will continue to use the term mitotic index (MI). MI is predictive for survival with < 9/10 high power fields generally being best and > 20 being worse.

3. Margins: Standardised reporting of margins as 'complete/clean' versus 'narrow/close' is also lacking. Generally, any complete margin is likely sufficient for grade 1 or 2 STS - though recurrence can occur, it is very rare. Recurrence even with complete histologic margins is somewhat more common in grade 3 STS. As for mast cell tumours, the deep margin is qualitative as well as quantitative - even if narrow, if the deep margin includes fascia or muscle then this should be an effective barrier to tumour cell invasion. With narrow histologic margins (defined as < 1mm or within the tumour pseudocapsule), < 10% of grade 1 tumours, approximately ⅓ of grade 2 tumours, and ¾ of grade 3 tumours recurrred in one study (2), and it appears likely that recurrence rates are similar for incomplete versus close margins. However, neither margin status nor tumour recurrence have shown a definitive impact on survival time.

Within those tumours typically classified as STS, the exact histologic subtype does not seem to significantly affect prognosis, though there is some association between subtype and grade or mitotic rate. At this stage, without evidence to suggest differences in behaviour, extensive investigation to assign a specific subtype is not generally recommended once a diagnosis of STS has been confirmed.

For FISS, completeness of margins is important, with incomplete margins associated with recurrence (approximately 10x more frequently than complete margins) and with disease free interval. Because of the invasive nature of these tumours, incomplete margins are common without radical surgery.

Surgery

Whilst STSs may appear to be well encapsulated, it is the cells that are at the pseudocapsule margin that are most active. Surgery that 'shells out' the STS is an intralesional excision and is associated with a high local recurrence rate. STSs have microscopic projections at their periphery invading surrounding normal tissues.

Wide surgical excision is the treatment of choice for STSs. The recommended surgical margins for STSs are proportional to grade. For FISS cases, radical margins of 5cm are recommended. It is important to obtain appropriate deep margins as well as lateral margins. The deep margin is considered a qualitative margin rather than a quantitative margin and should consist of fascia or muscle rather than adipose tissue, which is a poor anatomic barrier to tumour extension.

Low grade STSs on the extremities present a particular situation and have been reported to be able to be managed with more marginal resections. If there is insufficient tissue for primary closure or if tension is an issue, then open wound management is reported to be successful.

Adjunctive therapy

- Local treatment: In cases of incomplete or narrow histological margins (i.e. risk of local recurrence), additional local therapy with revision surgery or radiation therapy is recommended. However, since many incompletely or narrowly excised grade 1 or 2 STS do not recur, if clients do not wish to pursue additional therapy then ongoing monitoring is reasonable. For grade 3 tumours, the risk of local recurrence is much higher and adjunct therapy is more strongly recommended. Local chemotherapy with platinum-impregnated beads or intra-incisional 5-fluorouracil has been reported, but only in small numbers of dogs. Although recurrence rates are low, the majority are grade 1 or 2 STS and so the efficacy of these local therapies is as yet unknown, and there is some risk of toxicity.

Chemotherapy:

- Metronomic chemotherapy with low dose daily oral cyclophosphamide along with daily piroxicam has been shown to significantly improve disease free interval compared to surgery alone in dogs with incompletely excised STS, however all of the tumours in the control group in this study recurred (median disease free interval of approximately 7 months), which is inconsistent with other studies (3).

- Adjuvant chemotherapy with doxorubicin is often recommended following surgery for grade 3 STS due to the risk of metastasis and is effective in improving disease-free survival in humans, however this approach has not been proven to improve survival in canine STS.

- FISS: The combination of surgery and radiation therapy is generally accepted to be the most effective treatment. However, for tumours excised with radical surgery with wide histologic margins, adjuvant radiation may not be necessary. A recombinant canarypox virus expressing feline Il-2 (Oncept IL-2) improved tumour control in cats treated with surgery and radiation therapy in one study (4). The addition of chemotherapy (doxorubicin or epirubicin) may improve outcomes, but the true benefit is not clear.

Options for non-resectable tumours

- Canine STS

- Radiation therapy (RT) can be used with a definitive or palliative intent. With definitive type protocols, control rates are approximately 50% at 1 year and 33% at 2 years. Generally, radiation is used in macroscopic STS with a palliative intent, and approximately 50% respond with a median time to progression of approximately 5-10 months. One study in dogs treated with palliative RT +/- metronomic chemotherapy found that the addition of metronomic chemotherapy improved survival time but not progression free interval, so the true impact is hard to determine (5)

- Chemotherapy: Doxorubicin in macroscopic STS is associated with an overall response rate of 20-30%. Mitoxantrone and ifosfamide may be effective in some dogs. Metronomic chemotherapy with chlorambucil may shrink STS or maintain stable disease in some dogs (6)

- FISS

- Palliative RT is often effective at shrinking FISS in the short term, though there is often progression within a few months. Adding chemotherapy (doxorubicin) may improve response rates and duration. Doxorubicin alone will also be effective in some cases, with response rates of 40-50%, though again these do not tend to be durable. Metronomic chemotherapy has not been. IL-2 vaccine may slow progression in unresectable feline STS (unclear if FISS were included in this abstract) (7).

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ONCOLOGY

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- FISS: The combination of surgery and radiation therapy is generally accepted to be the most effective treatment. However, for tumours excised with radical surgery with wide histologic margins,

adjuvant radiation may not be necessary. A recombinant canarypox virus expressing feline Il-2 (Oncept IL-2) improved tumour control in cats treated with surgery and radiation therapy in one study (4). The addition of chemotherapy (doxorubicin or epirubicin) may improve outcomes, but the true benefit is not clear.

Options for non-resectable tumours

- Canine STS

  - Radiation therapy (RT) can be used with a definitive or palliative intent. With definitive type protocols, control rates are approximately 50% at 1 year and 33% at 2 years. Generally, radiation is used in macroscopic STS with a palliative intent, and approximately 50% respond with a median time to progression of approximately 5-10 months. One study in dogs treated with palliative RT +/- metronomic chemotherapy found that the addition of metronomic chemotherapy improved survival time but not progression free interval, so the true impact is hard to determine (5)

  - Chemotherapy: Doxorubicin in macroscopic STS is associated with an overall response rate of 20-30%. Mitoxantrone and ifosfamide may be effective in some dogs. Metronomic chemotherapy with chlorambucil may shrink STS or maintain stable disease in some dogs (6)

- FISS

  - Palliative RT is often effective at shrinking FISS in the short term, though there is often progression within a few months. Adding chemotherapy (doxorubicin) may improve response rates and duration. Doxorubicin alone will also be effective in some cases, with response rates of 40-50%, though again these do not tend to be durable. Metronomic chemotherapy has not been. IL-2 vaccine may slow progression in unresectable feline STS (unclear if FISS were included in this abstract) (7).

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ENDOCRINOLOGY

THE DOUBLE CHALLENGE OF THE DIABETIC CUSHINGOID

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ENDOCRINOLOGY: THE DOUBLE CHALLENGE OF DIABETIC CUSHINGOID

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Hyperadrencorticism and diabetes mellitus often occur concurrently and these patients can be a challenge to diagnose and manage.[1] They have similar clinical signs and the diagnostic tests are affected by the clinical diseases. Additionally, treatment of one disease will affect the treatment of the other. So, how to proceed with such challenging cases. Diabetes mellitus is usually diagnosed first as it is the easiest to recognize. Clinical signs include polyuria/polydipsia, weight loss, persistent or recurrent urinary tract infections, weakness and muscle wasting, cataracts (usually dogs), and peripheral neuropathies (usually cats). Diagnosis can be made by recognition of appropriate clinical signs, and demonstration of persistent hyperglycemia and glucosuria. One confounding factor to this diagnosis is stress. Stress, alone, can cause hyperglycemia, that can be high enough to be cause spill over into the urine and glucosuria. Should the clinician have any doubt of whether hyperglycemia and glucosuria are due to diabetes mellitus, s/he should check a serum fructosamine level. This value gives the average of the blood glucose over the preceding 2-3 weeks. If elevated, then diabetes mellitus can be diagnosed. After diagnosis, stable diabetics may be sent home with insulin to begin therapy. Optimal insulins for dogs include NPH, Lente (Vetsulin[R]), Detemir (Levemir[R]). Of these, Vetsulin[R] is the only one approved for veterinary use by the FDA. NPH insulin is an inexpensive option for owners, although the duration of action may not be optimal in some dogs. Vetsulin[R] and Detemir are more appropriate choices if insulin can only be given once per day. The optimal diet for diabetic dogs is one high in insoluble fiber. This diet slows glucose absorption from the gut and postprandial hyperglycemia. The patient should be re-examined in 7-10 days to determine insulin effect. Optimal monitoring includes evaluation of a glucose curve.[2-4]

Concurrent HAC in the diabetic patient is usually suspected if the diabetes mellitus is not controlled with insulin doses climbing over 1.5 U/kg (insulin resistance)

or if clinical signs consistent with HAC persist with insulin therapy.[5] Other reasons for insulin resistance should be considered to include improper insulin injection technique, outdated insulin, or incorrect syringes. The practitioner should also check for other concurrent diseases such as urinary tract infections. If HAC is still suspected, adrenal function testing should be initiated. Do not test for HAC if the dog is clinically unstable. There should be loose control of diabetes mellitus with the blood sugar ideally ranging from 100-350 mg/dl for most of the day. Do not test for HAC if the dog is ketotic, hypoglycemic, or hyperosmolar. Three screening tests FOR HAC are available.[6] The urine cortisol:creatinine ratio is very sensitive (100%) although not specific. A second screening test for HAC is the low dose dexamethasone suppression test. This should be to go to test unless there is significant concurrent disease such as diabetes mellitus. A third screening test for HAC is the ACTH Stimulation Test. It is more specific in animals with concurrent disease and recommended for use in dogs with concurrent diabetes mellitus to decrease the possibility of a false positive response. The practitioner measures cortisol before and 60 min after injection of 5 ug/kg synthetic ACTH (cortrosyn). In diabetic patients, the owner should feed and administer insulin as usual. A simultaneous glucose curve should be performed in conjuction with the ACTH-stimulation test to ensure that the animal has blood glucose within the optimal range during testing. If ACTH-stimulation testing supports HAC, then treatment should be initiated. If HAC is still suspected but is not supported by the ACTH-stimulation test, then the test can be repeated 2-4 weeks later.

Medical treatment of HAC includes trilostane (Vetoryl[R]) and mitotane (Lysodren[R]). Ketoconazole and deprenyl (Anipril[R]) have also been advocated, but are substantially less effective than trilostane or lysodren. Surgical options include adrenalectomy and hypophysectomy. Trilostane is a competitive 3B-OH steroid dehydrogenase inhibitor that inhibits the conversion of pregnenolone to progesterone in the adrenal gland. This blocks the formation of the end products of progesterone including cortisol and aldosterone. Trilostane can be compounded but a manufactured product is available (Vetoryl [R], Dechra Pharmaceuticals). Vetoryl[R] is FDA approved for veterinary use so that the veterinarian is legally protected. Vetroryl[R] is much more consistent in active ingredients than compounded trilostane and should be used when possible. The drug must be given daily twice daily. The recommended dose is 1.5-3 mg/kg, with the lower dose given bid considered safer and more efficacious.[7]Trilostane therapy should be monitored 10-14 days after initiation. An ACTH-stimulation test measuring cortisol is performed 4-6 hours after the morning dose of trilostane is given.. A sodium/potassium level should be measured as well. This often decreases with trilostane treatment because of increasing K+ due to loss of aldosterone function.[8]

Mitotane is a DDT derivative. It kills adrenocortical cells with a preference for the cortisol producing ones (zonae fasciculata and reticularis). The induction phase consists of administering 35 (big dog)-50 (little dog) mg/kg PO q.d. x 5-7 d. The maintenance phase is 35-50 mg/kg PO divided twice per week. Which therapy should the practitioner choose to treat HAC. In comparing trilostane and mitotane, both are as efficacious in controlling clinical signs and in the longevity of the treated animals.[9,10] We recommend starting with trilostane therapy as it is easier for owners to manage while simultaneously giving insulin injections. If the response to trilostane is not optimal, then lysodren therapy can be initiated.

Treatment for HAC in the patient with concurrent diabetes mellitus should be approached cautiously.[11] Removal of excess glucocorticoids will lessen the concurrent insulin resistance and result in more profound glucose-lowering activity by the usual insulin dose. The current insulin dose should be decreased by 25% upon initiation of treatment of HAC. Owners should be sent home with ketodiastix and instructed to measure urine at least once per day approximately 6 hours after insulin is given (if given twice per day). If ketones are detected or the absence of urine glucose is detected more than twice in a row, the owner should be instructed to inform the veterinarian. Lack of urine glucose may indicate hypoglycemia that may necessitate therapy. Alternately, if owners are able to collect blood glucoses at home, they should perform spot checks 4-8 hours after the insulin is given. Severe hyperglycemia or hypoglycemia should be reported to the veterinarian.

A glucose curve should be performed at every recheck to monitor HAC treatment. After the HAC is controlled, tight control of glucose with insulin can be attempted

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WAVMA ORNAMENTAL FISH DISEASES
CUTANEOUS LESIONS IN KOI

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CUTANEOUS LESIONS IN KOI

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Some of the most common complaints for which the pet fish practitioner is called upon to examine and diagnose are cutaneous lesions in koi. The majority of the time these lesions will be cutaneous ulcers. Along with "mouth rot' and "fin rot", these form the complex described as koi ulcer disease. Treatment protocols will vary depending on whether the lesions are of the trunk, the mouth or the fins or in combination. It will also be dependent on the extent of the lesion(s), the stage of degeneration/regeneration and the treatment options available. It is important to determine the stage in the development and healing as local debridement will be useful in the degenerative phase, but counterproductive during healing. This would limit the use of topical treatment to only the first few days after ulceration. Determination of stressors in the aquasystem leading up to the outbreak should be identified where present. Systemic antibiotics are often indicated, especially with multiple and/or extensive lesions. Additional considerations are maintenance of ideal water quality, especially if treating in a quarantine system and stabilized water temperature at 75 °F (24°C), the preferred optimum temperature for koi. Other conditions that may be seen on the integument are saprolegniasis, koi herpesvirus (CyHV-3), edema ("Pine cone disease"), macroscopic parasites (lernea, argulus, ich), neoplasia (carp pox, papilloma, squamous cell carcinoma), trauma (heron attack), special conditions of butterfly koi fins, special conditions of doitsu koi, and finally, clinically significant non-lesions (narial folds of showa, narial folds of butterfly koi, shimmies).

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ZOETIS (SIMULTANEOUS TRANSLATION INTO MANDARIN CHINESE)

WHAT'S NEW IN THE MANAGEMENT OF FLEAS AND TICKS IN DOGS

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New Strategies for Canine Tick and Flea Control

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Insects and acari have long presented a common and difficult challenge to companion animal veterinarians worldwide. Although effective insecticides and acaricides have been widely available for several decades, safety concerns and compliance issues have sometimes led to less than satisfying results even when best practices were followed. These arthropods cause a health concern not only because of infestation risk but also due to their ability to serve as vectors of serious, sometimes fatal, disease agents. Many flea- and tick-borne agents also infect people, creating a zoonotic disease threat.

Fleas remain the most common and important ectoparasite of pets worldwide. Reasons for our failure to eliminate the high risk of flea infestation for dogs include environmental burdens of immature stages that create an ongoing source for reinfestation, external sources of additional fleas that have not been identified, and owner behaviors including inadequate treatment[1]. These ongoing infestations present potentially serious health consequences for both dogs and people.

Although most dog owners are familiar with fleas, misconceptions abound about this ectoparasite and the best way to limit infestation risk. Some dog owners consider a few fleas to be normal on a pet and may not attempt to intervene until the home environment has been heavily contaminated with immature stages, while others may believe that since they do not see any fleas on their dog, treatment is unnecessary. Unfortunately, a few fleas are easily overlooked even by the most dedicated owner. Other mistakes made by pet owners include only treating once when a flea infestation is present, attempting to treat seasonally, or only treating the dog and neglecting to treat other pets in the family that may harbor active flea infestations[1]. All of these approaches can result in a failure to eliminate flea infestation and an ongoing canine and human health threat in the home.

Ticks have become a more urgent issue for companion animal health in many areas of the world due, in part, to wide scale geographic expansion of several tick species into new regions. As tick populations spread to new areas, the risk of disease transmission increases. Newly introduced ticks harboring tick-borne infections often catch the local community by surprise, resulting in widespread infection and disease even when vaccines and highly effective tick control products are available. Combining vaccination with tick control has been shown to dramatically reduce the risk of canine Lyme disease in endemic areas. Several acaricides, including systemic isoxazolines, have been shown to reduce or block transmission of tick-borne infections in experimental models.

One common misunderstanding among pet owners is that ticks can only be acquired from natural areas outdoors. While ticks are very common in both grassy and wooded areas surrounding homes as well as in urban and suburban parks and more natural, wild areas, one species in particular – the brown dog tick, **Rhipicephalus sanguineus** – thrives inside homes, kennels, and anywhere frequented by dogs. Due to the unique ability of brown dog ticks to survive long-term in the low humidity indoor environment, even dogs that spend no time in nature are at risk of acquiring severe, sometimes fatal tick infestations. All three stages feed on dogs and, when tick numbers are high, anemia and even exsanguination may result[2]. Premise infestations with brown dog ticks are extremely difficult to eradicate, resulting in a long-lived nuisance for owners and pets alike[3].

Controlling ectoparasites in dogs can be difficult. Although many options for flea control are available, until recently, persistent tick control options for dogs were limited to topical products, such as amitraz, fipronil, and pyrethroids. The advent of systemic isoxazolines, which provide safe and long-lived efficacy against fleas, ticks, and mites, offers new opportunities for protecting pets from the blood loss and dermatitis associated with infestation as well as reduced risk of disease transmission.

Isoxazolines have been shown capable of blocking infection with several tick-borne disease agents. Although ticks must attach to the host and begin to feed in order to acquire these systemic acaricides, some of these compounds act quickly enough to reduce or interrupt transmission[4,5]. Isoxazolines have also proven effective at eliminating mite infestations. Published studies to date support the use of these compounds for the treatment of demodectic mange, ear mites, and sarcoptic mange[6,7]. In addition, the isoxazolines provide excellent flea control, reducing flea populations and

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evidence of flea allergy dermatitis in naturally infested dogs.

The trend of increasing flea and tick populations and a greater risk of infections will likely continue for the next several decades. Mite infestations will similarly remain a common issue for pets. Because fleas, ticks, and some mites also create a zoonotic health risk, controlling these ectoparasites effectively has important One Health implications. Incorporating isoxazolines into routine parasite control programs for dogs brings with it the benefits of long-lived flea control, effective tick control, reduced transmission of some tick-borne infections, and the added benefit of treating or preventing mite infestations. Limiting these infestations improves the lives of animals and of people, protecting the human–animal bond and ensuring that pets remain close members of their human families.

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ACUTE ABDOMEN - CRITICAL CARE AND SURGICAL CONSIDERATIONS

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Acute gastric distension is a common cause of abdominal pain in emergency medicine, though it can sometimes be difficult to appreciate on physical examination. Findings on abdominal radiographs may also, at times, be ambiguous. This talk will cover the approach to common causes of acute gastric distension; including gaseous gastric dilation (GD), gastric dilation and volvulus (GDV), food engorgement and fluid distension.

Gaseous distension

Gas dilation of the stomach typically falls into two different categories; gaseous dilation related to the syndrome of GDV and gas distension secondary to aerophagia. Aerophagia, secondary to respiratory distress, usually only causes mild distension. However, sometimes the gaseous distension can impede caudal displacement of the diaphragm and reduce tidal volume, contributing to respiratory distress. In these patients, decompressing the stomach may relieve some of the respiratory distress. Often these patients have such severe respiratory distress that sedation and intubation is imminent. If the patient is already anaesthetized, then it is prudent to decompress the stomach via orogastric intubation (OGI) if distension is evident. If the patient is conscious, then careful nasogastric intubation (NGI) can be attempted, however the procedure must be aborted if it starts to compromise the patient. Gastric trocarisation is not usually indicated in these patients as the distension is not typically severe or compromising perfusion, and it carries the risk of trauma to abdominal organs.

Gaseous gastric dilation, or dilatation, in a large breed, deep chested dog is a problem that is inevitably situated on the continuum to GDV. Typical history is similar to GDV and includes acute retching, hypersalivation, a distended abdomen and agitation. These signs often occur in the early morning or evening, related to meal times. Physical examination findings are often similar to that of a GDV in the early stages, which are predominantly tachycardia, abdominal pain and tympanic abdominal distension. Some dogs do not have obvious tympanic abdominal distension due to their stomach being mostly under the rib cage. Any signs of vasoconstrictive shock (pale mucous membranes, decreased pulse amplitude, delayed capillary refill time, cool extremities) usually indicate that volvulus is present, but can still occur with severe gaseous dilation. Some

dogs with early GD or GDV can present with signs of vasodilation (injected or red mucous membranes, fast capillary refill time). Regardless, any compromise to perfusion prompts swift intervention.

The four essential steps for a dog with GD or GDV are opioid analgesia, blood volume expansion, abdominal radiographs and gastric decompression. Pre-treatment electrolyte and blood gas analysis is also useful. Acid base abnormalities usually parallel a lactic acidosis, however, some dogs display high lactate with a standardised base excess that does not reflect the degree of lactic acidosis.(1, 2) This may be due to pyloric outflow obstruction soon before the GDV occurred, causing preceding metabolic alkalosis, and some dogs are known to have either over-eating or a gastric foreign body as a feature of their GDV presentation.

Electrocardiographic monitoring is advised if a pulse irregularity is detected; pre-surgical ventricular arrhythmias have been associated with a higher incidence of gastric necrosis and a higher mortality rate. (3, 4) However, these arrhythmias usually involve isolated ventricular premature complexes and usually do not require treatment. An intravenous catheter should be placed promptly and any signs of shock treated with fluid therapy. Gastric dilation, with or without volvulus, causes obstructive shock by compressing the major veins in the cranial abdomen, and reducing venous return to the heart. Expanding blood volume increases perfusion pressure, and therefore venous return. This helps to address hypoperfusion quickly while giving more time to address the cause of obstruction. Crystalloid fluid therapy, such as Lactated ringer's solution, is often the most appropriate choice as these dogs can have some degree of dehydration. The use of synthetic colloid fluids in the pre-surgical patient or a patient at risk of Systemic Inflammatory Response Syndrome, without a strong indication, is controversial due to possible adverse effects and should not be routinely administered.

Blood volume expansion and radiography can sometimes be done concurrently if the treatment area is alongside diagnostic imaging facilities. It is ideal to perform radiographs before decompression in order to make the diagnosis clear. However, if the dog is in moderate to severe shock, decompression should be performed before taking radiographs. Decompression may result in resolution of the volvulus,(5, 6) or make it more difficult to appreciate volvulus on radiographs, but should confirm that it was indeed gaseous gastric dilation causing the problem, even if somewhat deflated. Some would debate that if there were no evidence of volvulus on radiographs, then emergency surgery is not indicated. However, if a dog presents with signs of shock on physical examination then chances are that there was some degree of volvulus present. Delaying surgery puts the dog at risk of complications from gastric necrosis or

gastric rupture, ongoing hemorrhage from any ruptured blood vessels or repeat volvulus soon thereafter.(5, 7)

One of the more difficult decisions is surgical planning for large-breed dogs that have simple gastric gaseous dilation on radiographs and **no evidence of shock**. It is still advisable to perform a gastropexy in this patient to prevent future GDV, however, it may not be urgently required. Sometimes these dogs can wait until the next day to either have a laparotomy or laparoscopy performed. The decision to delay surgery needs to be weighed up carefully in terms of how closely the dog can be monitored until the procedure is performed and whether or not the dog shows any signs of repeat gastric dilation, after the initial decompression, or abdominal pain.

There are two main methods for stomach decompression for gas; gastric trocarisation and orogastric intubation. There are positives and negatives to each procedure and no one method has been shown to be superior over the other.(8) Orogastric intubation (OGI) should be attempted first as it is less traumatic to the stomach. However, if it is difficult to decompress the stomach by OGI or the patient requires general anaesthesia to perform OGI due to non-compliance or the distension is severe accompanied by severe shock, then trocarisation is preferred. Sometimes all it takes is a little decompression via trocarisation to facilitate moving the OGI tube through the fundus. Trocarisation is a procedure that should be done carefully, with appreciation for the location of the spleen and being sure not to leave the stylette in place while the gas is being evacuated. Laceration of the spleen or liver is possible with this procedure.

As emergency laparotomy is usually a large expense, and these dogs typically require a high level of care post-operatively, many owners wish to know the pre-surgical risk of their dog dying before committing to the investment. Researchers have attempted to aid this process by assessing the ability of many pre-surgical factors to predict outcome. Clinical signs for >5-6 hours, hypothermia at admission and the presence of gastric necrosis combined with the need for splenectomy have been associated with a increased mortality. Pre-surgical factors that have shown some association with gastric necrosis or a higher rate of complications include high lactate on admission,(1, 9) high lactate post-fluids,(10) and ventricular arrhythmias(3, 4). However, care must be taken in applying results of these studies to individual dogs; the published mortality rates are often from small studies and some are older studies not reflecting today's standard of care. Also, each individual is unique for it's own risk, which can't be well predicted. Overall, for dogs that are taken to surgery, the discharge rate is usually above 90% if appropriate supportive post-operative care is given.

If owners decline surgery, then there are only two options; euthanasia and conservative management. Conservative management is not an appealing option. If decompression successfully repositions the stomach, gastric necrosis and perforation, and repeat GDV can occur within the next 24 hours, causing the patient to suffer. For those dogs that survive the initial GDV, most studies support that repeat GDV will occur in nearly all dogs within a year and, most, sooner.(5, 6)

Food engorgement

Food engorgement, or food bloat, can present with similar history and clinical signs to that of GDV.(2) On physical examination, there may be tachycardia, a distended painful abdomen that can be tympanic and hypersalivation. It is prudent to approach these patients in a similar fashion to a GDV case. Opioid analgesia should be given promptly and if the tachycardia does not resolve in response to analgesia, then a small crystalloid fluid bolus should be given. Gastric distension due to food engorgement can be marked on radiographs, however, if there is no gaseous distension and the stomach is correctly positioned, then there is no indication for surgery. Some clinicians induce emesis, which can be productive and reduce stomach size, however this may also increase abdominal pain due to stomach cramping and places the dog at risk of aspiration.

Fluid therapy, analgesia and time are usually all that is required. It is important to monitor electrolyte and acid-base status, as food engorgement can cause a mild free water deficit (hypernatraemia), and metabolic alkalosis due to third spacing of gastric fluid into the food mass. Close monitoring for any development of signs of GDV also is important. Most dogs improve after 12 hours of hospitalisation, with a decrease in abdominal distension, and can go home with instructions for rest and small meals. Mild diarrhoea or soft stool in the 3 days after engorgement is common.

Fluid distension

Acute fluid distension of the stomach is usually either due to gastric stasis or pyloric outflow obstruction. The degree of gastric distension does not usually cause obvious abdominal distension. Gastric stasis is usually associated with moderate fluid distension whereas pyloric outflow obstruction usually only causes distension if there is a component of decreased gastric motility. Gastric stasis is common in critically ill patients, especially in those suffering from Systemic Inflammatory Response Syndrome or recovering from abdominal surgery. Regional peritonitis, for example, due to pancreatitis, is also a common cause. Gastric stasis should be suspected in any critically ill patients that continue to vomit or regurgitate despite antiemetic and prokinetic drugs. Abdominal ultrasonography is useful to confirm suspicions, whereby a large fluid filled hypomotile stomach can be identified.

Fluid distension of the stomach can contribute to dehydration and electrolyte abnormalities via loss through vomiting or regurgitation. The nature of this loss will depend on whether there is duodenal reflux into the stomach and administration of antacid therapy. If there is no pyloric outflow obstruction, often the vomitus has a neutral to mildly acidic pH, as it is a mixture of stomach and duodenal fluid. If there is either a functional or mechanical pyloric outflow obstruction, or gastric hypersecretory disorder, then the majority of the loss will be hydrochloric acid, promoting a metabolic alkalosis in the patient. Potassium will also be lost in vomiting and regurgitation. As the effects on acid-base and electrolytes varies with the type of loss, it is important to monitor these parameters.

Gastric distension secondary to gastric stasis can be uncomfortable for the patient, and promote vomiting or regurgitation. If there is no response to prokinetic therapy, such as metoclopramide or erythromycin, it may be useful to place an NGT and remove the majority of fluid from the stomach. This helps to relieve some of the discomfort and often helps to control the vomiting or regurgitation. It also allows for gastric pH monitoring in order to assess efficacy of any antacids administered and allows the administration of enteral nutrition. Small-volume microenteral nutrition stimulates gastric motility, can improve lower esophageal sphincter tone and supplies essential amino acids to the gut, helping it to repair.

Acute fluid distension may also be due to ingestion of large volumes of water, such as in near-drowning cases. This usually causes vomiting and reduction in stomach size prior to presentation. However, if a patient presents with gastric distension due to water ingestion and requires a general anesthetic, it would be best to decompress the stomach via OGI soon after induction, while keeping the patient in sternal recumbency. This may avoid regurgitation and aspiration while providing mechanical ventilation.

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SVA INTEGRATIVE MEDICINE & ACUPUNCTURE

HOW I TREAT SEIZURES WITH ACUPUNCTURE AND HERBAL MEDICINE

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I. Introduction

In Traditional Chinese Medicine (TCM), a seizure is called Choufeng and epilepsy is called Xian Zheng. There are Yin and Yang seizures. Yin seizures are rarely connected with epilepsy. Yang seizures are clenched and spastic. The earliest literature on seizure and epilepsy can be found in Su Wen published during the 3rd Century BC. Both seizures and epilepsy belong to "Internal Wind Syndromes". The metaphor implies the movements one sees when wind rattles leaves on trees, causing them to shake erratically and involuntarily. These motions exhibited by leaves in a strong breeze resemble the people experiencing seizures. Traditional Chinese Veterinary Medicine (TCVM) shares the similar philosophy and theory of epilepsy in human TCVM. From the TCVM Medical perspective, etiologies of internal wind invasion involve six patterns (3 Excess and 3 Deficiency) that can result in seizures in both animals and man. The 3 Excess patterns include, Obstruction by WindPhlegm, Internal Profusion of PhlegmFire, and Blood Stagnation. The 3 Deficiency patterns are Liver Blood Deficiency, Kidney/Liver Yin Deficiency, and Kidney Jing Deficiency. Although they can be some overlap and combination of patterns, generally a patient will have a dominant pattern.

II. General TCVM Treatment For Seizures

a) General acupoints for seizures and its functions:

- Extinguish Wind: GB-20, Da-feng-men, CV-15, PC-5
- Liver points: BL-18/19, LIV-3
- Nourish Blood: BL-17, SP-10
- Transform Phlegm: ST-40
- Calm the Shen: GV-17/20/21, PC-6, HT-7, An shen, Nao-shu
- Special points: GV-1
- During seizures: GV-26, Nao-shu, HT-7

b) Basic Chinese herbs for seizures:

- Gastrodia (Tian Ma), Uncaria (Gou Teng), Concha Ostrea (Mu Li), Magarita (Zhen Zhu), Cornu Antelopis (Ling Yang Jiao), Lumbricus (Di Long), Buthus Martenzi (Quan Xie), Acorus (Shi Chang Pu), Bombyx (Jiang Can), Cicada (Chan Tui), Typhonium (Bai Fu Zi)

c) Classic Chinese herbal formula for seizures:

- Di Tan Tang (aka herbal 'Phenobarbital') from Ji Sheng Fang by Yan Yong-He, 1253
- It is functioning to transform phlegm, clear Internal Wind and stop seizure
- I usually always start with this formula and then add others if needed. Give 0.5 gram every 10 pounds of body weight, two to three times a day

III. Pattern Differentiation & Treatment

1) Obstruction by WindPhlegm

a) Signs: Sudden onset of seizures, loss of conscious, foaming at the mouth, possible U/D incontinence, depression or change of behavior right before seizures, often due to vascular event. Tongue is pale or purple with a white greasy. Pulses are wiry and slippery

b) Treatment principles: Expel Phlegm, extinguish Wind, open the orifices, calm the Shen

c) Acupoints: General acupoint above; add ST40, BL12, ST6 to clear Phlegm and Wind

d) Herbal formula: Ding Xian Wan, 0.5 g per 1020 lb body weight BID-TID

2) Internal Profusion of PhlegmFire

a) Signs: Sudden onset of seizure without warning, Wood personalities prone, sudden loss of conscious, foaming at the mouth and sometimes screaming, easily agitated and irritability, constipation, coughing, insomnia/barking or other abnormal behavior, clinical signs worse at night, increased panting, coolseeking. Tongue is red or purple, with a yellow greasy coating. Pulses are rapid, wiry and slippery

b) Treatment principles: Clear the Liver, drain fire, transform phlegm, open the orifices

c) Acupoints: General acupoint above; ST44, LI-4, LI-11, GV-14, Er-jian, Wei-jian to clear Heat/Fire

d) Herbal formula: Long Dan Xie Gan Tang + Di Tan Tang, 0.5 g per 1020 lb body weight of each BID-TID

3) Blood Stagnation

a) Signs: Usually trauma associated (i.e. history of injuries to the head), sudden onset of seizure without warning, loss of consciousness, foaming at the mouth and screaming, loss of continence of bowels and/or urine, temporary disorder of consciousness with or without seizure. Tongue is pale or purple, often with a white greasy coating. Pulses are wiry and slippery

b) Treatment principles: Expel Phlegm, extinguish Wind, and invigorate Blood

c) Acupoints: General acupoints above; add LI-4, ST-30/36, BL-21 to clear stagnation

d) Herbal formula: Ding Xian Wan + Tao Hong Si Wu San, 0.5 g per 1020 lb body weight of each BID-TID

4) Liver Blood Deficiency:

a) Signs: Chronic seizures (epileptics), anemia, weight loss, dry or brittle hair, cracked nails, muscle rigidity, especially of the neck and jaw, generalized weakness, cool ears and nose, warm seeking. Tongue is pale and dry. Pulses are weak, deep and thready

b) Treatment principles: Nourish Blood, expel Wind and Phlegm

c) Acupoints: General acupoints above; add: LIV-8, ST-36, SP-6, CV-12, BL-20, ST44 to support Qi and Blood

d) Herbal formula: Bu Xue Xi Feng San or Tian Ma Gou Teng Yin, 0.5 g per 1020 lb body weight BID-TID. Add Di Tan Tang if there is also Phlegm pattern

5) Liver/Kidney Yin Deficiency

a) Signs: Chronic seizures (epileptics), dry nose and mouth, seizure occurred at late afternoon or night, increased panting, coolseeking, nose and body feel warm to the touch. Tongue is red and dry with thin or no coating. Pulses are deep, fast and thready

b) Treatment principles: Nourish Yin, extinguish Wind, harmonize KID and LIV=

c) Acupoints: General acupoints above; add BL23, KID3/7, SP6/9, LI11, Er-jian to nourish Yin and clear Heat

d) Herbal formula: Yang Yin Xi Feng San or Tian Ma Gou Teng Yin, 0.5 g per 1020 lb body weight BID-TID. Add Di Tan Tang if there is also Phlegm pattern

6) Kidney Jing Deficiency

a) Signs: Multiple seizures at a very young age (1 year old or younger), often in combination with other congenital problems, inactive, dry nose and mouth. Tongue is pale or red but can be normal. Pulses are weak and thready but can be normal.

b) Treatment principles: Nourish Kidney Jing, expel Wind, strengthen Spleen

c) Acupoints: General acupoints above; add KID3/7, BL20/23, ST36, SP6, Bai-hui, GV-4 to support Qi, Yin and Yang

d) Herbal formula: Epimedium Powder + Di Tan Tang, 0.5 g per 1020 lb body weight of each BID-TID

IV. Other Considerations

a) General guidelines:

1) Acupuncture, once every 2 to 4 weeks for 5-8 sessions initially, then every 3-6 months for maintenance.

2) Herbal formula based on the TCVM pattern/differentiation, 3 to 6 months. General doge is 0.5 gram per 10 pounds BID-TID. If you don't know the patterns, you may always start with Di Tan Tang.

3) If it is a cluster of seizure, or starting a case already on Western medication, combine use of anticonvulsants: Phenobarbital (2 mg/Kg, PO BID) + Potassium Bromide (33 mg/kg, PO q24h) with the herbal formulations. Herbal medicine can be safely taken with these drugs. When the seizures are controlled (seizures free for 2-3 months with both TCVM treatments and medications), gradually reduce phenobarbital or KBr to lowest effective dose.

4) I usually start by reducing one medication by 1/4 and gradually decrease by 1/4 every couple weeks as long as there are no seizures, but I would continue herbs for about 6 months.

b) Avoid the Yang (warm) proteins (chicken/ lamb/ venison, etc.)

c) Avoid chemicals and drugs, which could make them more susceptible to seizures including Heartgard, Program, Advantage or Frontline (may lower the seizure threshold.)

a. Interceptor and Filaribits appears to be safe for dogs with seizures

b. Revolution may be safe to control heartworms

d) Avoid stress and exercise regularly

V. Scientific Evidence

- In neurobiological terms, the metaphors of "eliminating wind in the head" or "dispersing heat" translate into vagal nerve stimulation and reduction in sympathetic tone.
- In a rat model study, electroacupuncture at ear-point reduced epileptiform discharges in the cortex as well as epileptiform behaviors.5 Electroacupuncture suppressed levels of excitatory neurotransmitters in the hippocampus, whereas levels of the inhibitory neurotransmitters glycine, taurine, and GABA increased.
- Electroacupuncture stimulation in rats with pilocarpine-induced epilepsy improved cognitive deficits and prevented shrinkage of areas within the limbic system of the brain.6
- Electroacupuncture at either 1 mA or 3 mA significantly inhibit the pentylenetetrazole-induced cortical epileptiform activities in rats, and higher stimulation (3 mA) was not associated with a greater inhibition.7
- In a preliminary report, exogenously supplemented taurine improves the ability of electroacupuncture to protect against seizures in rats with penicillin-induced epilepsy; certain Chinese herbs prescribed for epileptic patients contain high amounts of taurine.

VI. Summary

TCVM can be an excellent adjuvant to conventional therapy in epileptic animals, especially those with poorly controlled seizures. In mild cases, or after an initial seizure, TCVM can be used alone to help prevent and minimize the occurrence of further seizures.

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Common TCVM Pattern Differentiation of Seizures and Its Treatment Plan

	Main Acupoints	Additional Acupoints	Main Chinese Herbal Formula	Additional Chinese Herbal Formula
Obstruction by Wind-Phlegm	• Soothe the Liver: BL-18, BL-19, LIV-3	ST-44, Er-jian	Ding Xian Wan	
Internal Profusion of Phlegm-Fire	• Clear Heat/Fire: LI-4, LI-11, GV-14			Long Dan Xie Gan Tang
Blood Stagnation	• Extinguish Wind: GB-20, CV-15, BL-17, SP-10, Da-feng-men	ST-36, TH-6, CV-4, CV-6	Di Tan Tang, and then add whatever else based on pattern differentiations.	Tao Hong Si Wu San
Liver Blood Deficiency	• Transform Phlegm: ST-40	LIV-8, SP-6, CV-12, BL-20		Bu Xue Xi Feng or Tian Ma Gou Teng Yin
Liver and Kidney Yin Deficiency	• Calm the Shen: GV-20, HT-7, An shen, Nao-shu	BL-23, KID-3, SP-6, SP-9		Yang Yin Xi Feng, Tian Ma Gou Teng Yin, or Zuo Gui Wan
Spleen Qi Deficiency	• Special points: GV-1, GV-26 (during seizure)	ST-36, SP-6, CV-6, BL-20, BL-21		Tian Ma Bai Zhu or Liu Jun Zi Tang
Kidney Jing Deficiency		KID-3, KID-7, BL-23, Bai-hui, GV-4		Epimedium Powder or Tian Ma Gou Teng Yin

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TIPS FROM THE EXPERTS FOR THE APPROACH OF...

ORTHOPEDIC EXAM

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Learning Objectives

At the end of this session, which will focus on canine lameness, you will be able to:

• recognise a shift in stance and how this identifies the site of lameness

• identify a positive sit test and how this helps localise disease

• identify elbow effusion and elbow pain as indicators of elbow dysplasia

• palpate medial buttress and stifle joint effusion as indicators of cruciate disease

• perform an Ortolani test for identifying hip laxity as an indicator of hip dysplasia

• identify typical signs of panosteitis on orthopaedic examination

• perform a test for biceps tendon laxity

An orthopedic examination, like a neurological examination, is a detailed systems examination that is performed in addition to a general physical examination. So you will need to allow **additional** time if you are going to perform a complete orthopedic examination.

The amount and quality of information derived from an orthopedic examination varies from clinician to clinician.

Much of an orthopedic exam relies on subjective assessment of the dog compared to a normal dog of that breed or type or compared to the joint / segment / limb on the contralateral side. Where possible try and rely on objective changes - for example where a joint is clearly enlarged and unstable in comparison to the contralateral joint. Unfortunately, identification of clearly objective abnormalities is not always possible – early disease with subtle changes and bilateral disease are examples.

To improve identification of subjective or subtle abnormalities requires a thorough, methodical examination based on a sound understanding of **normal** anatomy, stance and movement. Using a consistent method of performing orthopaedic examination will improve your ability to detect subtle changes.

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> A consistent and methodical approach to orthopedic examination is essential to maximise success

Signs of an orthopedic problem may include some or all of the following:

- stiffness on rising after rest
- change in exercise capacity or exercise behaviour
- change in stance (altered distribution of weight)
- lameness / change in gait
- pain – pain is the most common cause of lameness. Lameness can also result less commonly from mechanical causes (such as patella luxation), neurologic disease, vascular disease, and systemic disease.
- change in normal shape
- asymmetry – for example joint enlargement or muscle wasting. Reduction in muscle mass is typically seen in chronic lameness.
- change in normal alignment
- change in stability or range of motion

It is important to recognise that quadrupeds are very good at "masking" lameness or not showing obvious limping until their pain is quite marked. This has both evolutionary and practical benefits. Close observation of how an animal stands, moves, and gets up and down from a recumbent position will often indicate lameness in earlier or less severe cases.

What is lameness? Lameness is an abnormal gait or stance resulting from some abnormality in the locomotor system.

What are you trying to achieve in an orthopedic examination?

1. **localize the limb(s) affected** is the animal lame and if so in which limb(s)?

2. **localize the site(s) of the lameness** where is the source of the lameness?

3. **identify the cause(s) of the lameness** this usually involves radiography, advanced imaging or other diagnostics

4. **determine the appropriate treatment and prognosis**

Some definitions that are useful when talking about lameness:

- Stride - the full cycle of limb advancement
- Contact phase - the part of the stride when the foot is in contact with the ground
- Swing phase - the part of the stride when the foot is in the air and not in contact with the ground

There are 4 steps in an orthopedic examination

1. **History**
2. **Observation of stance and observation of gait**
3. **Standing symmetrical examination followed by recumbent examination**
4. **Further diagnostics** – as indicated

1. History

The history should be taken from the owner while the dog is allowed to stand or walk around in the consulting room or outside rather than placing the dog on the examination table. Having the dog walk or stand lets them become familiar and "more trusting" in unusual surroundings and allows you to observe them during this time.

In many cases simply by the way they stand or in some cases sit you will be able to identify the lame limb(s).

2. Observation of stance and observation of gait
Observation of stance: simpler than gait observation
Observing an animal stand often is sufficient to identify lameness and, in many instances, gives more useful information than gait exam.
Quadrupeds have the ability to shift their centre of mass both to one side, as with humans, and also cranially or caudally. This re-distribution of their mass reduces the load going through the lame / sore limb(s) thereby reducing the pain and very often minimises the observable lameness.
Visual observation of canine lameness is relatively insensitive. In one study 75% of dogs with no observable lameness were actually lame when measured objectively on a force plate[1]. In other words, even in dogs with significant unilateral lameness (and remember that bilateral lameness is even **more** difficult to detect than unilateral lameness) only 25% showed apparent lameness on visual examination of their gait.

However even though quadrupeds shift their centre of mass to minimise observable lameness, this shift is usually readily apparent provided that you are familiar with normal stance of dogs – and that you actively **look** for this change.

To expend the least amount of energy when standing a normal dog will stand with the foot directly beneath the dependent joint – the shoulder or hip joint. When dogs have a unilateral limb lameness they bend their spine laterally towards the lame side and bring their sound leg closer to the midline. The lame leg is laterally abducted. This creates a "tripod" stance where the lame limb is used for balance but is not taking full weight.

In some cases when you look at their feet you will see that in the sound limb that is taking most of the load the metacarpal / metatarsal pad is fully compressed and the nail tips consequently sit up off the ground. On the contralateral lame limb the pad is not fully compressed and so the nails are usually in contact with the ground.

When dogs have a bilateral forelimb lameness they often

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shift their mass more to the pelvic limbs by bringing the feet of the pelvic limbs cranially. If you look at them stand from the side you will see that their feet are cranial to a line drawn perpendicularly from the hip to the ground.

When dogs have a bilateral hindlimb lameness they often shift their mass more to the thoracic limbs by bringing their elbows caudally so they are positioned more caudally underneath their thorax than normal. If you look at them stand from the side you will often see that the feet of the thoracic limbs are caudal to a line drawn perpendicularly from the shoulder to the ground. They will often also lower their head to further shift their weight from the hindlimbs.

Not that too many dogs do "handstands" but if you think of the action they would need to make if they were going to perform a handstand this will explain the way they change their stance in bilateral hindlimb lameness.

Observation of gait: what sort of gait abnormalities could you observe? This depends whether the cause of the lameness is unilateral or bilateral.

If lameness is **unilateral**, or if one leg is significantly worse than the other, then these gait changes are typically observed:

- **the classic "head-bobbing" lameness**. Remember the head drops on the good limb not on the lame limb. "Down on the sound" is the phrase that is often used. This not infrequently is misinterpreted by owners and they may wrongly advise you that they think the dog is lame on what is actually the sound limb. The classic head-bobbing lameness is most obvious in unilateral forelimb lameness. Landing more heavily on the sound limb is also seen in the hind limbs though is less obvious than in the forelimbs.

- **shortened contact length and duration in the affected limb and increased contact length and duration on the sound limb**. The animal wants to spend as little time with the sore leg bearing weight as possible. The contact phase is shorter in the lame leg and longer in the sound leg. The swing phase is quicker in the sound leg as the dog is "rushing" the good leg through to take weight to minimise the time that the sore leg needs to bear weight. This faster swing phase in the sound leg is often misinterpreted as indicating this is the lame leg.

- **circumduction** – the animal with a painful joint or a decreased range of motion in a joint will often circumduct the limb in preference to flexing the joint.

Bilateral lameness, particularly when it is symmetric, is particularly hard to detect. Beware the bilaterally lame animal as these commonly go undetected as the gait abnormalities are more subtle. In these cases, the dog has "lost the luxury of limping" as the limb on one side is just as sore as the other. Their main compensation in these cases is to walk with their centre of mass set in the position described above under changes in stance.

Typically dogs with bilateral joint-related lameness will walk with a "stiff" or "stilted" gait as they limit the range of motion of their painful joint.

What about grading the severity of the lameness?

This is subjective and varies with the individual doing the grading. There are a number of "systems" for grading lameness, including a descriptive scale, a scale of 1 to 10, a scale of 1 to 5 etc but none is widely accepted. None have been validated for repeatability. The key is to be consistent with whatever system you choose and ideally use the one simple system throughout all clinicians in your hospital.

3. Standing symmetrical examination followed by recumbent examination

Standing symmetric examination

The aim of this part of the exam is to compare each side simultaneously for evidence of difference in size, either muscle wasting or joint / segment / limb enlargement. Also, to palpate the musculature of the spine. This is most easily done standing behind and over the dog.

The forelimbs are easier to detect relative muscle wasting than the hindlimbs by comparing the prominence of the spine of the scapula. In the hindlimbs muscle wasting is identified by symmetric palpation of the main muscle masses and is more affected by the amount of load the dog is taking on the limb at the time.

Relative hindlimb muscle mass is most reliably compared visually when the dog is anesthetised in dorsal recumbency during the subsequent diagnostic investigation. Relative joint enlargement, which can be from a variety of causes most commonly effusion and periarticular fibrosis, can be palpated in the elbow and stifle and joints distal to these. It is uncommon to be able to palpate joint enlargement in the shoulder or hip joints.

Conscious proprioception should be assessed as part of the standing symmetry exam. The spine should be assessed by gentle palpation of the epaxial muscles and vertebrae for pain and assessment of free range of motion of the cervical and lumbosacral spine.

Recumbent examination

This obviously relies on having a cooperative dog. Most dogs will allow a calm recumbent exam. Owners are most often not useful in helping quietly restrain the dog.

Start at the foot pads and work proximally. Palpate every structure progressively as you move up. Think of the underlying anatomy as you do so.

Palpate each joint and assess whether it is enlarged, either through effusion or periarticular fibrosis, has normal stability and a normal range of motion, whether there is crepitus or palpable osteophytes present and in particular whether there is consistent localisation of pain.

Localising a focus of pain is of course challenging. It

is subjective and relies on a cooperative and trusting patient. However consistent localisation of a focus of pain is one of the most useful findings on orthopaedic exam to identify the site of the problem.

If you have identified possible limitations in joint range of motion during the recumbent exam you should confirm this if the dog is having further diagnostics performed under sedation or general anaesthesia by repeating the recumbent examination. This is very useful as it allows easy immediate comparison of range of motion and stability of joints although of course removes identification of pain as a localising factor.

Palpate the soft tissue structures. Palpate the muscle groups for defects, abnormal texture, masses and pain. Palpate and stress accessible tendons and ligaments. Be familiar with the location of regional lymph nodes and assess for evidence of enlargement.

Firmly palpate the bone where it is superficial enough to do so. It is abnormal for bone to be painful on palpation.

> **Successful detection of the abnormal requires a sound familiarity with what is normal**

Repeating the orthopedic examination while the animal is under general anesthesia is often very beneficial as it allows more detailed examination, particularly of joint structures, than is often possible while the animal is conscious. This is usually done if further diagnostics such as radiography are to be performed. In animals with a poor temperament examination under sedation or anesthesia may be the only way to perform a physical examination. It is important to remember that reliable pain localisation is lost under heavy sedation or general anesthesia.

4. Further diagnostics.

History, observation and physical examination generally allow localisation of the site of the lameness. In some cases it may also determine the cause of the problem and the appropriate treatment and prognosis. More usually however further diagnostics, most commonly radiographic examination of the affected area, are necessary before an accurate diagnosis and appropriate treatment may be determined.

Other diagnostic aids commonly used in orthopedics include arthrocentesis (joint tap and synovial fluid examination), arthroscopy, arthrotomy, biopsy (of bone, muscle, joint capsule), haematology and biochemistry panel.

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SOCIAL MEDIA FOR VETERINARIANS

TOP MARKETING & COMMUNICATION MISTAKES & HOW YOU CAN FIX THEM

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THE KEYS TO MARKETING SUCCESS

Making sure your veterinary practice is running smoothly day-to-day is your priority, as it should be. But what about expansion, marketing, return on investment and all of the elements needed to truly ensure that your veterinary practice isn't just open for business, but is also **thriving like never before**?

A surprising amount of veterinary practices are operating with old software/infrastructure in place and are even avoiding the huge array of tools that can help them to truly maximize their marketing efforts across the board. On the other hand, some veterinary practices are achieving unprecedented growth and success by utilizing both new marketing techniques and software, while continuing to dedicate their efforts to delivering remarkable service, day in and day out.

The difference is in the effort put forth and the dedication it takes up to keep up with evolving tools and trends when it comes to social media, marketing and digital infrastructure.

While some of the advances and changes can be daunting at first, all it takes is a slight adjustment in thinking to realize that these new tools, whether we're talking about **Survey Responses** or **Social Media**, can help us to get our veterinary practices running in top-gear with a full roster of very happy clients.

Fortunately, for veterinary practices that may have yet to make the transition toward a new world of marketing, there are tried and true techniques that can enhance marketing efforts for all types of practices; whether emergency care, specialty, general practice or other.

When things are moving quickly within your veterinary practice, it's especially important to make sure that there are techniques already in place to ensure that your clients are satisfied, feeling appreciated and consistently leaving your veterinary practice in full confidence that both their time and money have been well spent. You might be surprised at how many veterinary practices handle a full client load, but forget to capitalize on strategies that could be enhancing their business from the inside out. This might mean that they are missing opportunities to gain new referrals and gain crucial feedback on what may or may not be working when it comes to their veterinary practice. This could also mean

that they are being out-paced by their local competitors, without even knowing how to keep up...

Here are a few proven techniques that can help you to successfully implement a marketing strategy that's built for the twenty-first century and beyond. Using these techniques, while making adjustments that allow you to customize the strategies to benefit your specific veterinary practice, will allow you to retain your current clients, expand your outreach in order to gain new ones, spend your marketing dollars far more effectively and enhance the way that your veterinary practice operates each day. By following and implementing these simple rules, you can set your veterinary practice up for total success, with the tools needed to keep improving each day:

MEASURE CLIENT SATISFACTION

1.) The importance of measuring client satisfaction cannot be overemphasized. **Every client that leaves your practice without giving feedback on services and their overall experience is a lost opportunity to improve and gain invaluable information from the most important person to your veterinary practice: your client.**

In order to measure client satisfaction successfully, you may want to focus most intently on clients that have recently visited your practice, preferably within the last one or two days. Their visit with you is still in recent memory, and any feedback and/or critique can easily be garnered at this stage. You can complete this step by sending out surveys via email, or even handing out a final form once the client is making payment and scheduling a future appointment.

Techniques for the client survey can vary, but should effectively monitor satisfaction and ensure that you've provided good service that will render future visits and references. You may try asking questions with a "**One to Ten**" level of response to most effectively gauge the services that can be improved by your veterinary practice. Use this strategy to measure the promptness of the visit, friendliness of the staff, knowledge of the primary veterinarian, etc. You can design surveys to be anonymous or to even enter the client into a contest as incentive for completion. For example, "Tell us what you think and you can win a $100.00 gift card!" Different clients have different ways of rendering their experience in total candor, so you may want to experiment with different incentives and techniques here.

You can also measure which techniques of giving a survey yields the most results, I.E. sending out an online survey the same day or the next day after a client appointment and/or giving out a physical survey which can elicit an on-the-spot response.

The technique you choose to use is of course up to

your veterinary practice, but should allow for honest feedback to be gauged, analyzed and recorded by your veterinary practice, resulting in room to improve service and consequently ratings, for future appointments. Even the most successful veterinary practices have room to improve and measuring client satisfaction is one of the best possible ways to do this.

TRACK YOUR RETURN ON INVESTMENT

2.) It can be tempting to start plugging money into marketing techniques that are polished, newly released and seem exciting. Why not spend $100.00 to promote a cute Facebook post or $50.00 on a new Facebook advertisement? Well, the truth of the matter is that **you should not spend money on marketing without putting the necessary tools in place to track your return.**

ROI is a term that you should learn inside and out. Simply put, it means **Return On Investment**. Unless you know that a specific advertisement is bringing new patients directly through your front door, there is no way to ensure that you're effectively advertising. Ineffective advertisements can actually do more harm than good, causing your veterinary practice to spend money and direct attention to a specific medium, without actually garnering results. Practices that do not implement a proper infrastructure to measure their return can blindly spend money, without ever achieving the results that they desire. This can actually be trickier than you think. For example, if you do spend $100.00 on Facebook and get plenty of replies and shares, this may seem like a successful campaign! The post may even lead more people to visit your website or to follow your Facebook account. Still, if the visitor is not contacting your veterinary practice directly to schedule an appointment, you may be spending more money on web-traffic and your online promotion, than you are actually securing a new client! There are however, ways to distill your marketing efforts and ensure that dollars spent, result in dollars earned:

One great place to start is with **CallRail.com**; a tool that records incoming calls and determines where the client came from before calling you directly. For example, if a client finds you on Facebook (a very common example) and proceeds to your website before calling you, CallRail will allow you to gain valuable insight on the process, noting which lead resulted in the call (in this case, Facebook) and other important data about their process. You can then distinguish if the call came from a targeted Google search, Google AdWords, Facebook, Twitter, etc.

One of the largest advantages of our digital age, is the ability to leverage new tools and analytics to derive more data than ever. While the process of caring for your clients and delivering impeccable service may stay more or less the same, the process of tracking leads, traffic and growth has changed more than ever before.

By making sure that your veterinary practice is adjusting to these changes and implementing the right software, you'll be poised to compliment your great service with great tools and marketing as well. You'd be amazed at what can be achieved when great veterinarians and staff deliver outstanding care, while implementing the right tools to measure their success.

APPRECIATE YOUR LOYAL CLIENTS

3.) While showing appreciation to your local clients may seem like it goes without saying, it's actually something that a surprising amount of veterinary practices forget to do on a daily basis. When you've got a packed calendar, it can be easier to focus on the next client coming in, than it is to thank the one that's walking out the door after spending money on your services. Instead, show your appreciation by saying thank you, leaving a note, calling the next day to follow-up, or building in a client loyalty program to your practice. One way to do this is to **create a referral program**, which is an all-in-one way to reward existing clients, attract new ones and spread a feeling of generosity to your existing clientele. This can be small but significant, allowing the person who refers the practice to receive $50.00 (or another appropriate amount) toward a future service or an additional purchase.

Many times, a client has a built-in network of friends and family that they could easily recommend to your veterinary practice, but this additional step is often overlooked. Simply by appreciating your loyal clients and adding in this extra layer of incentive, you can extend your outreach to a new level and bring in new clients who already may be right around the corner. If your client has had a positive experience, the best thing you can do is parlay this positivity outward. By showing your appreciation for the client and offering a small monetary incentive, you can do just that!

Companies like Uber do this same thing, utilizing referral codes directly through their app. This occurs to the tune of tremendous success, as those who are already utilizing the app can actually offset the cost of the service by recommending it to another. For a rider who is in transit, this may just be the most effective use of their time. Similarly, if you offer these incentive based strategies while your client is in the waiting room or while they are making a final payment, you may just find that they are eager to use the time to offset existing costs. Little things like gift cards, discounted purchases or free pet treats can make the difference between a new referral and a shrug.

CUSTOMIZE FOR YOUR PRACTICE

These strategies allow room for you to make changes and implement the techniques that will work best for you and your veterinary practice. This can also read: there is no single way to go about any one of these specific marketing techniques and the strategies will require adjustments and fine-tuning to fully optimize them toward your individual practice. A large part of successful implementation will rely on your understanding of your individual veterinary practice and your individual clients, including how to customize your client survey/satisfaction techniques, marketing strategies and reward/incentive programs directly toward your clients. You may find that certain strategies work with incredible consistency, while others need to be scrapped or fine-tuned along the way. The key is to take the larger themes, and work within them to enhance what's already working and fill in the voids where you may be lacking a strategy at all.

While technology as a whole continues to accelerate, it's more important than ever to tune into the increasing needs, wants and expectations of clients and consumers alike. From text-message updates to newsletters and online pharmacy prescription refills, we rely on the digital world more than ever to get things done every day. Your clients expect the same, wanting to experience an exceptional veterinary experience while ensuring the health of their pet is totally optimized. Sure, expectations are high. But so are the opportunities to deliver and amaze.

With all of the tools at our disposal and new strategies that can be used to enhance marketing and customer experience, the only thing left to do is implement accordingly and reap the rewards.

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TICKS AND TICK-BORNE DISEASES UPDATED: WHAT VETS NEED TO KNOW

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Introduction

Tick infestation and tick-borne diseases are one of the most important factors hindering pet owners to do outdoor activities with their dogs in many countries. Ticks are basically blood-sucking arthropods that cannot survive or lay eggs without blood meal, and they are present outdoors and in the wild where blooded animals are present. Tick infestation is zoonotic and so are many tick-borne diseases. Understanding the biology of tick and their life cycle are as critical as chemotherapy in controlling ticks and tick-borne diseases in both animals and humans.

Tick biology

Although many insects consume blood meal from animals, ticks and mites are not insects. They are closer in taxonomy to spiders and scorpions than to insects. Therefore, nymph and adult stages have four pairs of legs while larval stage has three pairs of legs. Most insects do not take blood meal from host animals unless they become adult stage, while ticks have to take blood meal even from the juvenile stages. Therefore, while hard ticks can go for several months without feeding, all three stages have piercing mouth parts. Since ticks have to take blood meal even from the larval stages, they try to get close to where blooded animals wander around. Although both male and female ticks take blood meal, females have expandable abdomen to take enormous amount of blood meal from the host to lay eggs. Those engorged ticks on the dog skin, therefore, are females [1].

Most ticks that get onto humans and companion animals are hard ticks and are three-host ticks. It means all developmental stages, larvae, nymph and adult, will fall off to the ground after blood meal from the animal host either to molt or to lay eggs. It also means that it is impossible to eradicate ticks among the wildlife animals and will keep coming up onto dogs wandering in the woods if not on preventitives. They seldom climb up onto the tree tops and fall down to animals and humans. In general, ticks that are questing on the tips of grasses and shrubs are found up to 50 cm from the ground. Unlike fleas, ticks cannot jump or fly. Completion of the entire life cycle may take from less than a year in tropical regions to over three years in cold climates, where certain stages may enter diapause until hosts are again available. Although ticks have preferred animals to take blood meal, they have low host specificity and will therefore get onto any blooded animals including humans [2].

Ticks spread diseases

Like mosquitoes, ticks transmit pathogens that cause disease through the process of feeding, not via the feces. When a tick finds a feeding spot after a searching period of 10 minutes to 2 hours on host, it inserts its feeding tube into the skin. After secreting a cement-like substance that keeps them firmly attached during the blood meal, the tick secrets small amounts of saliva with anesthetic properties so that the animal cannot feel pain. The saliva also contains anticoagulants to keep the blood from hardening, and exhibits cytolytic, vasodilator, anticoagulant, anti-inflammatory, and immunosuppressive activity [3].

Ticks are major vectors of pathogens affecting both humans and animals worldwide and transmit a variety of microorganisms including viruses, bacteria, and parasites (protozoa and helminths). Each year, more than 30,000 cases of Lyme disease in humans are reported in the U.S., while studies suggest the actual number of people diagnosed with Lyme disease is more likely about 300,000. Other less known, but serious tickborne diseases include Rocky Mountain spotted fever, anaplasmosis, ehrlichiosis, Powassan virus, and babesiosis. In dogs, ehrlichiosis, rickettsioses, hepatozoonosis, hemoplasmosis, tick-borne encephalitis, and tick paralysis can occur as well as anaplasmosis and babesiosis [4].

The most important tick-transmitted infectious diseases causing severe clinical illness in dogs are babesiosis, anaplasmosis, ehrlichiosis and, in the USA, RMSF and hepatozoonosis. However, although Borrelia burgdorferi and Rickettsia conorii infections commonly produce subclinical infection, their association with a clinical disease in dogs is more difficult to evaluate. Infection with tick-borne pathogens can also be complicated by other arthropod-borne diseases that possess overlapping distribution with the different tick species, such as leishmaniosis with sand flies as the transmitting vector. In dogs, several different co-infections of Anaplasma, Ehrlichia, Bartonella, Babesia, Hepatozoon, Leishmania and Rickettsia species occur frequently in endemic areas [5].

Several of these tick-borne infections can also cause serious diseases in humans, and dogs may play an important role in the transmission of such pathogens because dogs can act as a domestic reservoir for certain nidicolous ticks such as Rhipicephalus sanguineus and I. canisuga if they are natural hosts. Dogs significantly increase the contact between these species and humans, thereby increasing the risk of transmission. Dogs may carry ticks of all life stages that are not

attached to the host or may be interrupted during feeding. These ticks occasionally leave the canine host and are able to find another host, infest and finally transmit pathogens. This is rather important for companion animals living in close contact with humans.

Epidemiological tick control and preventative measures

Although strategies to reduce populations of vector ticks through area-wide application of acaricides (chemicals that will kill ticks and mites) and control of tick habitats (e.g., leaf litter and brush) have been effective in small-scale trials, chemical removal of ticks from wildlife habitats are impossible. Community-based, integrated, tick-management strategies may prove to be an effective public health response to reduce the incidence of tick-borne infections. This includes general preventative measures of dog owners. However, limiting exposure to ticks is currently the most effective method of prevention.

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ONCOLOGY

DIAGNOSIS AND TREATMENT OF ENDOCRINE NEOPLASIA IN DOGS AND CATS

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DIAGNOSIS AND TREATMENT OF ENDOCRINE NEOPLASIA IN DOGS AND CATS

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Learning objective: Develop an algorithm for diagnosis and staging of thyroid tumours in dogs and adrenal tumours in dogs and cats. Understand rational application of different treatment modalities in different situations.

Thyroid tumours:

Cats with thyroid tumours typically present for clinical signs of hyperthyroidism, dogs for a palpable mass. Although dogs with thyroid tumours are rarely hyperthyroid, elevated T4 in a dog is almost certainly due to a thyroid tumour (rather than hyperplasia). Thyroid carcinoma represents the least common cause of feline hyperthyroidism. Once a thyroid tumour in a dog is palpable, it is almost certainly malignant. Because size and mobility is an important prognostic factor in canine thyroid tumours, early detection via routine neck palpation as part of regular physical examination should be emphasised in dogs. Carcinoma should be considered in hyperthyroid cats with extremely elevated T4, very large or fixed goitre. Screening for metastasis is warranted in cats with suspected thyroid carcinoma, and treatment with surgery or I 131 may be more effective than medical management, however the true best approach is not defined.

In evaluation of a dog with a suspected thyroid tumour, our general approach is as follows:

1. Confirm cervical mass is of thyroid origin and assess resectability - thyroid origin can be confirmed with imaging (ultrasound or CT scan) and cytology, though cytology is not specific for malignancy (often thyroid carcinomas have few cytologic criteria of malignancy). Mobility is best assessed under heavy sedation or general anesthesia. If a thyroid tumour appears to be relatively fixed/immobile, contrast CT scan should be avoided if I 131 treatment is a possibility. Total T4 should be evaluated in every case (+/- TSH, free T4 if TT4 is low)

2. Staging for metastasis - evaluation of local lymph nodes (LN) - generally submandibular and prescapular - is recommended. Cytology should be performed in all local LN, with excision for histopathology in any concerning for metastasis. Imaging (ultrasound or CT) may increase suspicion of LN metastasis but should not be considered a substitute for cytology. Thoracic imaging (CT is more sensitive but radiographs reasonable) +/- abdominal imaging (ultrasound or CT) should also be performed.

Then:
- If tumour is mobile and there is no gross metastasis, surgery is recommended
- If tumour is fixed/non-resectable and no gross metastasis, definitive radiation therapy (RT) is preferred, with I 131 considered next best option if external beam RT is not available. Chemotherapy (see below) may also be an option of I 131 is not available.
- If tumour is metastatic - I 131 can still be effective (risk of radiation pneumonitis or liver injury with diffuse pulmonary or hepatic metastasis), palliative external beam RT can be considered if significant clinical signs from primary tumour, or chemotherapy.

Surgery:
- Surgical excision of the affected thyroid gland is approached via a ventral midline cervical approach. The sternohyoid and sternothyroid muscles are bluntly separated on the midline and retracted laterally to expose the thyroid gland on either side of the trachea
- The extracapsular technique is used for removal of thyroid tumours.The blood supply to the thyroid gland is via the cranial and caudal thyroid arteries. The cranial and caudal thyroid artery and vein, and any other large vessels associated with the tumor are ligated. Canine thyroid carcinomas can invade into or be adhered to adjacent tissues. The recurrent laryngeal nerve and vagosympathetic trunk (within the carotid sheath) are identified and spared if possible. The jugular vein, carotid artery, vagosympathetic trunk and laryngeal recurrent nerve can be sacrificed unilaterally with acceptable morbidity if required.
  - The contralateral thyroid gland should be visualised at the time of surgery.
  - Potential complications after thyroidectomyectomy include hemorrhage and anemia, hypothyroidism, laryngeal paralysis and megaesophagus.

Radiation therapy (RT):
- For non-resectable primary tumours, definitive RT results in median survival times of several years, though maximal tumour shrinkage can take months to > 1 year. Adjuvant RT following surgery is commonly recommended to prevent local regrowth for incompletely excised tumours but there is limited information on effectiveness.
- Palliative RT for the primary is a reasonable option for dogs with metastatic disease or owners for whom definitive RT is not feasible. Many tumours will shrink, at least partially.

- I 131, where available, may be a more attractive option in dogs with metastatic disease because it has the potential to be effective against both the primary tumour and metastases. In dogs without metastasis, median survival time after I 131 is approximately 2 years, and with metastasis approximately 1 year. Use of I 131 as an adjuvant treatment following surgery is commonly done in humans but its true utility is not fully established in dogs. Scintigraphy is required prior to I 131 treatment to assess for uptake. If iodinated contrast has been performed, scintigraphy and I 131 treatment must be delayed for a minimum of 4 weeks (preferably 6-8 weeks).
- Hypothyroidism and myelosuppression can occur following I 131 treatment, and hypothyroidism can occur after external beam RT also.

Chemotherapy:
- In dogs with unresectable or metastatic disease, responses to carboplatin, doxorubicin, and toceranib (Palladia) are reported. In my practice, toceranib is generally my first choice because it can be given long term if effective and well-tolerated. Use of adjuvant chemotherapy following surgery for high-risk tumours is often recommended but has not been thoroughly evaluated for effects on survival. Tumours considered high-risk are large tumours (> 4-5 cm diameter likely to have higher metastatic risk), or those with lymph node metastasis.
- **Adrenal tumours** are more common in dogs than in cats, and may be of cortical (adenoma/carcinoma) or medullary (phaeochromocytoma) origin. Animals may present for effects of products of functional adrenal tumours (hyperadrenocorticism, intermittent weakness or other signs due to catecholamine release, rarely weakness due to hypokalaemia from a mineralocorticoid secreting tumour), but non-functional adrenal tumours may have no clinical signs and be found incidentally on abdominal imaging for other concerns, or may present with abdominal pain or haemorrhage directly related to the tumour. Pre-operative diagnosis of adrenal tumours can be challenging, except in the case of obvious adrenal dependent hyperadrenocorticism. In small (< 2cm), incidentally diagnosed adrenal tumours, 'watchful waiting' is likely reasonable. Consideration should be given to screening for a primary tumour elsewhere, as metastasis to the adrenal glands can also occur. In larger adrenal tumours or those with vascular invasion, malignancy is more likely and further investigation is warranted. If possible, identification of functional tumours prior to surgery is desirable for appropriate planning for perioperative management. Staging with thoracic and abdominal imaging (CT preferred, otherwise thoracic radiographs and abdominal imaging) is indicated in any concerning adrenal mass to evaluate for vascular invasion and screen for metastasis. Testing for functional status should include assessment for hyperadrenocorticism and evaluation of urine catecholamine/metanephrines. Fine needle aspiration and cytology of adrenal masses in many cases can distinguish between the two main tumour types. There are potential risks with this procedure, however in the little published data available it overall seems safe.

The primary treatment for adrenal tumours is surgical

excision. This can be acheived by open ventral midline laparaotomy approach or via a retroperitoneal approach. For smaller adrenal masses without caval invasion, laparoscopic adrenalectomy is possible. Tumour invasion into the phrenicoabdominal vein and caudal vena cava require venotomy to remove the tumour emboli. In the case of phaeochromocytoma, pre-operative use of phenoxybenzamine decreases the perioperative mortality rate. After the perioperative period, outcomes for both adrenal carcinomas and phaeochromocytomas can be very good in dogs. Vascular invasion, emergency surgery for haemorrhage, and large tumour size increases the perioperative risk but do not necessarily impact on long term outcome.

In non-resectable adrenal tumours, stereotactic radiation therapy has been recently reported with good outcomes (several year survival time). Adjuvant therapy following surgery has not been established in companion animals. For adrenal dependent hyperadrenocorticism, if surgery is not a good option medical therapy can be attempted. Mltotane can be used as a cytotoxic agent (higher doses than required for pituitary dependent hyperadrenocorticism are typically required, and approaches to ablate the adrenal tissue are described), or trilostane to control clinical signs can also be considered. Other chemotherapy agents have not been evaluated in companion animals with adrenal gland tumours.

ONCOLOGY

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ENDOCRINOLOGY

INSULIN THERAPY - SO MANY CHOICES AND SO LITTLE TIME

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ENDOCRINOLOGY: INSULIN THERAPY – SO MANY CHOICES AND SO LITTLE TIME

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Insulin is a small peptide hormone that has a highly conserved amino acid sequence throughout different mammalian species. This allows for the use of human-based insulins in veterinary species. Insulin is secreted in a stable hexameric form stabilized by a zinc molecule in the middle. The hexamer needs to be broken down to a monomer before it can bind to the insulin receptor and activate cells. In considering insulin therapy, the practitioner should be aware of the source of the insulin (animal, human recombinant DNA, human sequence mutated), the type of the insulin or how it is made into a repository form, and the concentration (U-40, U-100, U-300).

Regular insulin is available as a human recombinant insulin in 100 U/ml (U-100) form. It is the stabilized hexameric form of insulin, and therefore, not precipitated or mutated. It may be used intravenously, intramuscularly, and subcutaneously. Regular insulin is used to treat unstable or dehydrated diabetics. Effective protocols for IM intermittent therapy or continuous rate IV infusion are available in any emergency medicine handbook.

Protamine Zinc Insulin (ProZinc[R]) is human recombinant insulin. It is precipitated with protamine zinc and is stable in suspension. It should be rolled gently to mix. ProZinc[R] is formulated at a concentration of 40 U/ml; therefore dosing can be easier in smaller animals like cats. The 0.3 ml syringes can be used so that half units can be easily measured. ProZinc[R] is approved by the FDA for veterinary use; therefore, company support of the product is readily available for veterinarians. This is a great insulin in cats and also is effective in dogs.

Vetsulin[R] is a purified porcine insulin that has an identical animo acid sequence to canine insulin. It is a mixture of ultralente and semilente insulins and is precipitated with zinc to form a suspension. To keep the ratio of semilente to ultralente consistent, it must be shaken vigorously before use. VetPens[R] containing Vetsulin[R] are available for convenient dosing by owners. These pens may be measured for accurate ½ unit dosing. Vetsulin[R]

is approved for veterinary use by the FDA and company support is readily available for veterinarians. This should be a first pick for dogs and is also effective in cats.

NPH (neutral protamine hagedorn) is a recombinant human insulin. It is distributed by several manufacturers under names such as Humulin N® (Eli Lilly) and Novolin N® (Novo Nordisk). A generic version is also available. It is a crystalline suspension of human recombinant insulin with protamine and zinc added. The concentration of NPH is 100 U/ml. This is the most inexpensive insulin on the market and may work in some dogs. It has a short duration of action in cats that can remain hyperglycemic for significant portions of the day. Thus, NPH is not recommended as a long term insulin in cats (unless the owners want to give 3-4 injections daily to their cat)

Glargine (Lantus [R]) is a long-acting human mutated insulin available as a U-100 and U-300. It is stable at pH 4.0 but forms crystals at pH 7.0 when injected under the skin. Insulin adsorbs off the crystals and is released into the blood streallt is marketed to give 24 hour basal control of insulin circulation. In humans, glargine is often used in a basal-bolus pattern with injection of another insulin preparation at meal times. Lantus[R] pens are available but are only adjustable in 1U increments and have relatively short injection needles. This is a great insulin in cats and will also work in dogs.

Detemir (Levemir [R]) is a similar insulin to glargine in its use in human diabetes mellitus. It is a mutated human insulin with fatty acid side chains added so that it can bind to albumin after being injected under the skin. It is also available as a prefilled pen with the caveats of the Lantus[R] pen. This is a fabulous insulin in dogs especially as a "rescue" insulin. It may also be used in cats.

Lispro (Novalog[R]) and Aspart (Humalog[R]) are two mutated human recombinant insulins that are used to manage human diabetes mellitus along with the longer acting glargine and detemir insulins. They are available as U-100 insulins. These insulins work extremely quickly because they are already in monomeric form and do not need to be broken down from the normal insulin hexamers. Their onset of action is rapid (5-15 minutes) and their duration of action is approximately 1 hour. Their use has been limited in veterinary medicine.

The starting dose of insulin should be: 0.25 U/kg for cats and 0.5 U/kg for dogs, except for detemir in dogs that should be started at a lower dose of 0.25 U/kg. Most dogs and cats will need insulin twice per day. If owners are only able to give insulin once per day, consider Vetsulin[R] or Levemir[R] in dogs and ProZinc[R] or Lantus[R] in cats. Dogs and cats should be fed twice a day when insulin is given. A small amount of food should be presented, and the animal's appetite noted. Insulin should then be given. If the pet does not eat normally, half the dose of insulin should be given. Many owners give insulin while the animal is eating. This makes the insulin injection a pleasant experience for the pets and easier for owners to treat the animal. Some cats prefer to nibble food throughout the day. These grazers can often be well managed by allowing them free choice eating with insulin injections twice per day. Care should be taken to ensure that the cats are not receiving more than their caloric needs since extra weight should be avoided.

Exercise is beneficial to diabetics and serves to lower insulin requirements and provide better glycemic control. Daily walking for dogs and cat play can be effective ancillary treatments for diabetes mellitus. Average time for initial diabetic control is 4-6 weeks.

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WAVMA ORNAMENTAL FISH DISEASES

ANESTHESIA IN AQUATIC VET MEDICINE

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ANAESTHESIA IN AQUATIC VET MEDICINE

Abstract

In fish, anaesthetic agents are used for handling, restraining, and for prevention of pain during painful procedures. The presentation will give practical advice depending on the situation, factoring water temperature, body mass, species and physiological state. Additionally, some common surgical procedures and their techniques will be discussed.

WSV18-0281

SVA INTEGRATIVE MEDICINE & ACUPUNCTURE

HOW TO USE ACUPUNCTURE AND HERBAL MEDICINE FOR TREATMENT OF CHRONIC RENAL FAILURE

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I. Introduction

Chronic renal failure (CRF) is a common renal disorder in both dogs and cats. There is little treatment available from a Western perspective, other than fluid therapy and a protein-restricted diet, for the end stages of CRF. Other recommended dietary changes include reducing quantities of phosphorus and sodium, and increasing caloric density, potassium, dietary fiber, B-vitamin content, and magnesium.1,2 Recent studies have shown that antioxidants and Ω-3 polyunsaturated fatty acids (PUFAs) may benefit patients with renal failure. Antioxidants relax smooth muscle and increase glomerular filtration rate.8 Fish oil reduces plasma lipids and intraglomerular pressure.9 Ω-3 fatty acids heal glomerular and tubulointerstitial lesions which may offer some protection of glomerular function, thereby minimizing renal disease progression.10 Other mechanisms of Ω-3 fatty acids include anti-inflammatory, anti-coagulant, and anti oxidant effects, as well as reducing intrarenal calcification. 3,4

Traditional Chinese Veterinary Medicine (TCVM) has more to offer patients with CRF, as it has been used in animals for thousands of years in China.5 Clinical anecdotal evidence indicates acupuncture and Chinese herbals may greatly benefit patients with renal failure and slow disease the progression.6,7 Acupuncture has shown to significantly improve the renal functions on the recovery from ethylene glycol- induced acute renal injury in dogs.11 These improvements in renal functions are likely due to acupuncture's neuromodulatory influence on autonomic tone.12 Many Chinese herbs or herbal mixture (formula) have been shown to lower the serum creatinine level, increases inulin clearance, decreases urinary protein excretion, and attenuates lipid derangements in human or animal models with CRF.13

II. General TCVM Treatment

In the theory of TCVM, the notion of the 'Kidney' is not the same as that of the kidney in Western medical science. Kidneyt in the TCVM perspective is 'the place where the true Yin and true Yang hibernate; it is the base of hiding and the place for storing the refined energy and essence, its quintessence appears on the hair, and its function is to enrich the marrow of bone, and

associates with water.14 Thus, abnormalities of 'Kidney' are believed to cause multiple disturbance of the body.14 Several clinical signs related to CRF were recorded, such as edema, 'guan-ge' (anuria with vomiting), 'ni-du' (stranguria), and 'long-bi'.14

The general treatment principle for CRF is to:
- Drain Dampness to regulate immune system and promote urination
- Activate and nourish Blood
- Resolve Stasis
- Replenish vital energy
- Coordinate Yin and Yang

a) Common acupoints for seizures and its functions:
- Strengthen Kidney Qi: BL-23/52, BL-26, KID-3, Shen-shu
- Warm Kidney Yang: GV-3/4, BL-24, CV-6, Bai-hui, Shen-shu/peng/jiao
- Nourish Yin: BL-23, KID-1/3/7, SP-6/9, Shen-shu
- Benefit urination: BL-22/28, SP-9/15, GB-25, ST-28, CV-9

b) Additional acupoints for clinical signs:
- Poor appetite: Shan-gen, Jian-wei, BL-20/21, ST-36
- Poor digestion: BL-20/21, ST-25/36
- Nausea/vomiting: PC-6, BL-20/21, ST-36, GB-34
- Diarrhea: ST-36, SP-6, BL-25, GV-1
- Edema: GV-4, SP-6
- Stranguria: CV-3, BL-39
- Heat: LI-4, LI-11, GV-14, Er-jian, Wei-jian
- Abdominal pain: SP-6
- Restlessness/abnormal behavior: PC-6, HT-7
- Panting/coughing: CV-17, BL-43, BL-13
- Hearing loss: SI-9
- Anemia: BL-17, BL-20, BL-23, SP-10
- Hypertensive: PC-6, LI-4, KID-1, LIV-3, GB-20, GB-34
- Itchy skin: LI 11, St 36, GB-20, SP-10

c) Methods of Stimulation:
- Dry needle for 20-30 minutes
- Electroacupuncture, 20Hz for 10-15 minutes followed by 80-120Hz for 10-15 minutes
- Aquapuncture, vitamin B12 injected into 5-10 acu-points, 0.1-0.3 cc per acupoint
- Moxibustion at GV-4, BL-23/24, CV-6, KID-3, SP6 for patient with Kidney Yang deficiency or edema
- Acupressure or laser acupuncture

d) Tui-na massage:
- Mo-fa (Touching Skin and Muscle) or Ca-fa (Rubbing) along the back-shu Bladder meridian (BL28 to BL11) and along the Conception Vessel meridian (CV8 to CV2) for 3-5 minutes
- Rou-Fa (Rotary-Kneading) along the back-shu Bladder meridian (BL28 to BL11) from caudal to rostral and along the Governing Vessel meridian (GV-2 to GV-14) back and forth 12 times

- Rou-fa along the Conception Vessel meridian (CV8 to CV2) back and forth 12 times
- Clockwise thumb Rou-fa to stimulate GV-4, GV-14 and GV-20 for 1-3 minutes
- Ca-fa and Rou-fa along the medial thigh over the 3 Yin channels (Kidney, Spleen and Liver) of the hind limbs, following the flow of the meridians (from foot to upper limb) for 3-5 min
- Clockwise thumb Rou-fa to stimulate KID-3 and KID-7 for 1-3 minutes
- Single-finger Ji-dian-fa (Dotting) to stimulate ST-36 for 1-3 minutes

e) 10 Common Chinese herbs for CRF:
- Huang-qi (Astragalus membranaces or Astragalus mongholicu), Chuan-xiong (Ligusticum chuanxiong), Dang-gui (Angelica sinensis), Da-huang (Rheum pal-matum), Dan-shen (Radix salvia miltiorrhizae), Dong-cong-xia-xao (Cordyceps sinensis), Di-huang (Salvia Miltiorrhiaei), Fang-ji (Stephania tetrandra), Fu-ling (Poria cocos), Lei-gong-teng (Tripterygium wilfordii)

There are hundreds of Chinese herbs used to treat chronic kidney diseases. Many of them have been shown to improve renal function in patients with CRF in a number of studies. Their mechanisms of action are mainly related to antioxidation, anti-fibrosis, and improvement of metabolic disturbance in CRF. Unfortunately, the effective components or chemical compounds in most of these herbs remain unknown due to the difficulty of pharmacodynamic studies of herbs and the isolation of active ingredients from herbs or herbal mixtures.

Huang-qi (Astragalus membranaces or Astragalus mongholicu) has multiple beneficial effects on stimulation of the immune system, promotion of diuretic activity, antioxidation, anti-inflammation, and renoprotection. It decreases glomerular hyperperfusion and proteinuria, and improving the plasma levels of total cholesterol and albumin.

Da-huang (Rheum palmatum L) plant have been studied extensively in China.15 It was found that rhubarb lowers the serum creatinine level, increases inulin clearance, decreases urinary protein excretion, and attenuates lipid derangements.15 Some studies reported that rhubarb not only has an renoprotective effect by itself but also may have an additive beneficial effect with ACE inhibitors.15 In a systemic review of 18 randomized or quasi-randomized trials from 15 Chinese journals, rhubarb showed a positive effect on relieving uremic symptoms, lowering serum creatinine, improving hemoglobin levels, and adjusting disturbance of lipid metabolism in 1,322 human patients with CRF.16

Among other kinds of Chinese herbs used to treat CRF, Dan-shen (Radix salvia miltiorrhizae) relaxes vessels by enhancing microvascular protein synthesis of endothelial nitric oxide synthase, leading to an increase in nitric oxide production.17 Danshen, in combination with

seven other herbs including Chinese rhubarb, confers nephroprotection in chemical-induced acute and chronic renal failure in rats.18 Animal studies have shown that Dong-cong-xia-xao (Cordyceps sinensis) delays progression of worsening kidney function in 5 of 6 nephrectomized rats, through the inhibition of glomerular hypertrophy, reduction of proteinuria, and reversal of metabolic abnormalities of protein and the lipid profile.19 Fang-ji (Stephania tetrandra) decreases the accumulation of extracellular matrix and reduces glomerulosclerosis in adriamycin-induced nephrotic rats.20 Lei-gong-teng (Tripterygium wilfordii) has been known to protein excretion in many types of CRF.21

III. Pattern Differentiation & Treatment

1)Kidney Qi Deficiency

- Signs: Dysuria, polyuria, stranguria, lower back pain, hindlimb weakness, exercise intolerance, tired easily, urinary incontinence, uremia, prefer warm area. Tongue is pale and wet. Pulses are deep and weak (right pulse is weaker)

- Acupuncture treatment: Strengthen Kidney Qi (BL-23/52, BL-26, KID-3, Shen-shu); Benefit urination (BL-22/28, SP-9/15, GB-25, ST-28, CV-9); Add additional points above to manage clinical signs if needed

- Herbal formula:

  - 1) Suo Quan Wan, 0.5 g per 1020 lb body weight BID-TID, to treat renal failure with mild urinary incontinence

  - 2) Jin Suo Gu Jing, 0.5 g per 1020 lb body weight BID-TID, to treat renal failure with chronic urinary incontinence and weakness

2) Kidney Yang Deficiency

- Signs: often older animals or end-stage CRF, low body temperature, cold ears, back and extremities, aversion to cold, warm-seeking behavior, subdued manner, lower back or lumbar region soreness, hearing loss, copious/long clear urine, urinary incontinence, general debility/ weakness, morning diarrhea, edema in limbs or ventral abdomen. Tongue is pale, wet and swollen with teeth marks. Pulses are weak, deep and slow (right pulse is weaker).

- Acupuncture treatment: Warm Kidney Yang (GV-3/4, BL-24, CV-6, Bai-hui, Shen-shu/peng/jiao); Benefit urination (BL-22/28, SP-9/15, GB-25, ST-28, CV-9); Add additional points above to manage clinical signs if needed; Use less acupoints (typically 5-10 needles in total) for debilitated patients

- Herbal formula:

- 1. Jin Gui Shen Qi Wan or Rehmannia 11, 0.5 g per 1020 lb body weight BID-TID, to tonify Kidney Qi and Yang, nourish Yin, Blood and Jing, drain Damp and strengthen the Spleen. Jin Gui Shen Qi Wan is slightly warmer than Rehmannia 11

- 2. Zhen Wu Tang, 0.5 g per 1020 lb body weight BID-TID, if edema is noted. It tonifies Kidney Yang, drain Damp and promote urination

- 3. Caution should be taken when using either formula in cats or geriatrics as they both contain Fu Zi (Aconite)

3) Kidney Yin Deficiency

- Signs: dysuria, stranguria, polyuria, easily dehydrated, dry coat with dandruff, warm to the touch, cool-seeking behavior, excessive panting, generalized erythema, restlessness or abnormal behavior at night, signs often worse at night. Tongue is red and dry with cracks and little to no coating. Pulses are weak, deep, thready and fast (left pulse is weaker)

- Acupuncture treatment: Nourish Yin (BL-23, KID-1/3/7, SP-6/9, Shen-shu); Benefit urination (BL-22/28, SP-9/15, GB-25, ST-28, CV-9); clear Heat is needed (LI-4, LI-11, GV-14, Er-jian, Wei-jian); Add additional points above to manage clinical signs if needed

- Herbal formula:

  - 1. Liu Wei Di Huang Wan, 0.5 g per 1020 lb body weight BID-TID, to nourish Yin and Jing, tonify Kidney Qi and drain excess Damp

  - 2. Zhi Bai Di Huang, 0.5 g per 1020 lb body weight BID-TID, to nourish Yin and Jing, tonify Kidney Qi, drain excess Damp, and clear Heat; it is used when apparent Heat signs are noted

  - 3. In CRF patients with both Kidney Qi and Yin deficiency, use Jin Gui Shen Qi Wan or You Gui Wan in the morning to tonify Kidney Qi-Yang; in the afternoon, use Liu Wei Di Huang Wan or Zuo Gui Wan to nourish Yin and Jing. The dosage is 0.5 g per 1020 lb body weight of each formula

4) Kidney Jing Deficiency

- Signs: renal failure at a young age, other congenital problems from an early age, congenital defects, poor neonatal growth and development, developmental bone diseases, premature aging, poor dentition, failure to thrive, often also show signs of Kidney Yin or Yang deficiency. Tongue is pale or red but can be normal. Pulses are usually weak but can be normal

- Acupuncture treatment: Benefit urination (BL-22/28, SP-9/15, GB-25, ST-28, CV-9); For Qi deficiency, use BL-23/52, BL-26, KID-3, Shen-shu; For Yang deficiency, use GV-3/4, BL-24, CV-6, Bai-hui, Shen-shu/peng/jiao; For Yin deficiency, use BL-23, KID-1/3/7, SP-6/9, Shen-shu; Add additional points above to manage clinical signs if needed

- Herbal formula: Sheng Jing San or Epimedium Powder, 0.5 g per 1020 lb body weight BID-TID, to tonify Qi, nourish Kidney Yin and Yang, Jing and Blood. Because this formula is warm, if the patient shows more signs of Yin deficiency with Heat, treat it as a case of Kidney Yin deficiency initially

IV. Summary

There is little treatment available for chronic renal failure from a Western perspective. In contrast, Traditional Chinese Veterinary Medicine has treatment options for patients with CRF, which may minimize the progression of disease and may help maintain quality of life. The combination of herbal medicine, acupuncture, and dietary management, in addition to Western treatments, can be a very effective therapeutic approach to treat chronic renal failure in small animals.

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ENDOCRINOLOGY

MONITORING DOGS WITH DIABETES MELLITUS

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MONITORING DOGS WITH DIABETES MELLITUS

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Aims of Management

Treatment of diabetes mellitus (DM) involves insulin along with dietary modification. Goals for treatment include eliminating clinical signs by controlling blood glucose levels below the renal threshold for as much of 24-hour period as possible and avoiding clinically significant hypoglycaemia.[1] This is facilitated through determination of the optimal insulin type and dose, maintenance of appropriate body weight and appetite, as well as manage or treatment of comorbidities.

Monitoring Tools

These include performance of blood glucose curves (BGC), monitoring of glucosuria, measuring fructosamine, clinical signs and weight. Results from these modalities may also yield conflicting information.[2]

  i)   Clinical signs and weight

An owner log would be useful to keep track of appetite, water consumption and urination habits. Clinical hypoglycemia should also be recorded. A physical examination including measurement of body weight should be performed at each visit.

  ii)   Urine glucose

Urine glucose measurements can be helpful but remain only an indication of the BG over the time interval that the bladder was filling and relying solely on urine glucose measurements are not encouraged. Persistence of trends may also be more significant than single measures unless ketonuria is detected.

  iii)   Glycosylated proteins

Use of glycosylated proteins for monitoring diabetic patients includes serum fructosamine and glycosylated haemoglobin. Fructosamine forms from the Irreversible binding of glucose to serum proteins, mainly albumin. Fructosamine concentrations are proportional to the blood glucose levels as well as affected by the half-

life of albumin. It reflects glucose levels over the 1-2 weeks before sampling. However, several factors, other than plasma glucose concentration, affect fructosamine concentration including hypoproteinemia, hyperlipidemia, and azotemia.[3]

Glycosylated haemoglobin (HbA1c) is a haemoglobin product with glucose attached to its N-terminal amino acid valine. Glycosylation is irreversible, and concentration of HbA1c within the circulation is approximately 2–3 months, the lifespan of the red blood cell. Therefore, it may be a consideration for long-term control of stable diabetics, especially when the patient has comorbidities that affect turnover of fructosamine. Renewed interest has been shown in monitoring HbA1c, as unlike fructosamine, is associated with outcomes and therapeutic targets in humans. [4,5]

1. iv) Blood glucose curves

A BGC should be performed on the introduction of a new type of insulin; when deciding on a dose change; one to two weeks after a dose change; three monthly in stable diabetics; when clinical signs of DM recurs; or when hypoglycaemia is suspected. The goals of a BGC are to determine the duration of action of the insulin, the glucose nadir, and the range of BG throughout the day. Ideally, we aim for the duration of insulin effect to be 10–12 h, BG nadir 100–150 mg/dl for the long-term diabetic pet, and average BG less than 250 mg/dl over that 10–12 h.[1,5] However, that while these parameters are ideal, AAHA defines goals of diabetic monitoring to be control of clinical signs while avoiding hypoglycaemia.[1] Also, there appears to be a considerable variation in serial BGCs in diabetic dogs, making curve interpretation and decision making difficult.[6] Therefore, glycaemic control should not be based solely on these numbers. The AAHA recommends home BGCs where possible, as they are expected to be representative of the patient's activity, and eliminates stress and associated influences.

BG monitoring should be based on methods validated for dogs. The AlphaTrak 2 is the glucometer recommended for use in veterinary patients as it has been calibrated in dogs and cats.[1,7] The preference is to use whole blood with this glucometer to improve accuracy, rather than plasma or serum.[8] The use of continuous and flash glucose monitoring systems have also been described.[9,10] These devices allow measurement of interstitial glucose and have shown good correlation to blood glucose within specific ranges. [8,9] These systems provide advantages of allowing more frequent measurements, not requiring patient restraint and phlebotomy, and decreased cost to the client. It also improves the ease at which home glucose curves to be performed.

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ZOETIS (SIMULTANEOUS TRANSLATION INTO MANDARIN CHINESE)

WHAT'S NEW IN THE MANAGEMENT OF FLEAS AND TICKS IN CATS

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The primary objective of this session is to learn the clinical signs of vector-borne diseases in cats so that appropriate diagnostic tests, treatments, and preventions can be used.

Multiple vector-borne diseases can affect cats; those transmitted by ticks (multiple agents), fleas (multiple agents), mosquitoes (**Dirofilaria immitis**), and sandflies (**Leishmania** spp) are among the most common. The Companion Animal Parasite Council (www.capcvet.org), European Scientific Counsel Companion Animal Parasites (www.esccap.org/guidelines/), and Companion Vector-Borne Diseases (www.cvbd.com) are excellent sources of information about vector-borne diseases in cats.

As high as 80% of fleas collected from cats contain at least one organism that could induce illness in cats or people. **Bartonella** spp, the haemoplasmas, and **Rickettsia felis** are most common. Fever is one of the most common manifestations of both bartonellosis and haemoplasmosis in cats. **Bartonella** spp and **R. felis** both can cause clinical diseases in people. Haemoplasmosis is one of the most common causes of hemolytic anemia in cats. Other notable flea-borne parasites in cats include **Dipylidium caninum** (the flea is ingested to induce infection) and **Yersinia pestis** (rodent fleas). Whether fleas are common vectors for **Coxiella burnetii** is currently unclear.

Most of the tick-borne diseases diagnosed in dogs have now been identified in cats. Many of these tick-borne agents have been grown or amplified from blood or have induced serum antibodies in the serum of normal cats or those with clinical signs such as fever. In some countries, however, thorough evaluation of cats for tick-borne disease agents has not been completed. In those situations, dog results can be used as evidence for the presence of individual agents in the region that could potentially infect cats. Most cats exposed to these agents do not develop clinical illness. But when illness occurs, most of the clinical and laboratory findings of tick borne diseases in dogs have been identified in cats. Thus, if you have a suspected agent in dogs in your area, you should also consider it as a differential for cats with similar clinical findings. Results of studies from regional ticks can also be used as evidence for risk in cats.

The most common tick borne diseases in cats are **Anaplasma phagocytophilum** (**Ixodes** spp.), **A. platys** (**Rhipicephalus sanguineus**), **Borrelia burgdorferi** (**Ixodes** spp.), and **Ehrlichia** spp. (**Rhipicephalus sanguineus**). Some **Cytauxzoon** spp. are pathogenic, in particular, those in the United States. **Hepatozoon** spp. infections are fairly uncommon in cats and it is unclear how often **Francisiella tularensis** infections are transmitted to cats by ticks. It is also not clear what role that the Spotted fever group rickettsia play in cats but fever is a likely clinical finding if fever exists. **Rickettsia rickettsii** would be the most common agent infecting cats in the United States; In Spain, **R. conorii** and **R. massiliae** antibodies were found in cat serum and DNA amplified from cat blood (Segura et al, 2014).

Antibodies can be detected against some of the agents using IFA or other serological tests (in particular, **Anaplasma phagocytophilum**, **Bartotella** spp., **Borrelia burgdorferi**, **Ehrlichia canis**, and **Rickettsia** spp.). However, serologic tests have not be optimized for use with cat sera for most of the agents and serological test results can be falsely negative in the acute stages of illness. Thus, currently amplification of agent specific DNA by PCR assay is the best way to prove infection. For the haemoplasmas, there is no currently available serological test and so cytology and PCR are the only way to prove infection.

When clinical disease associated with a flea or tick borne agent is suspected, particularly **Anaplasma** spp., **Bartonella** spp., **B. burgdorferi**, **Ehrlichia** spp., and **Rickettsia** spp., administration of doxycycline at 5 mg/kg, PO, twice daily or 10 mg/kg, PO, once daily generally is effective. It is possible that minocycline will also prove to be effective for some agents. Care should be taken to avoid esophageal strictures depending on the tetracycline compound being used. **Cytauxzoon felis** is treated with the combination of atovaquone and azithromycin (Cohn et al, 2011). Optimal duration of therapy is unknown for most of the agents, but clinical signs of fever and cytopenias generally resolve quickly.

Other than the Spotted fever group agents, permanent immunity is not induced by flea or tick borne agents and so reinfection can occur. Thus, since vaccines are not currently available for any of the agents for cats, it is imperative that flea and tick control be maintained on all cats. There is no danger for direct zoonotic infection of owners, but infected fleas or ticks harbored by infected cats could be brought into the home.

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SVA INTEGRATIVE MEDICINE & ACUPUNCTURE

APPLICATION & TECHNIQUES OF QING MING XUE AND QIU HOU XUE

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APPLICATION & TECHNIQUES OF "Qing Ming Xue" AND "Qiu Hou Xue"

Introduction

Animals commonly suffer from eye diseases such as conjunctivitis, corneal edema, corneal ulcer, cataracts, dry eyes and glaucoma. These can lead to damage to the ocular surface and are commonly associated with symptoms of ocular discomfort.

Objective

The objective is to relieve ocular discomfort.

Methods

Acupuncture at the "Qing Ming Xue" and "Qiu Hou Xue" is applied with the aim of achieving maximum therapeutic effect.

Results and Conclusion

There is evidence that this method can improve blood circulation, relax the tense muscles around the eyes, and promote part of the complex regulatory mechanisms.

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FELINE TRANSFUSION MEDICINE

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Feline Transfusion Therapy

Transfusion support is also critical for the feline patient, most commonly to correct anemia and less often bleeding. Nevertheless, blood transfusions are overall still less frequently administered to cats than dogs for a variety of reasons. The peculiarities of feline blood types, blood collection and transfusion will be presented.

Compared to canine transfusion medicine, cats can tolerate anemia better, they still get somewhat less medical attention, except for rodenticide toxicity and hepatopathies they bleed less severely, recruiting healthy donors is more difficult (occult heart disease, viral infections), blood collection requires sedation and special small bag collection systems, component therapy is less commonly practiced in clinics, cats have important naturally occurring alloantibodies and may experience life-threatening complications with a first transfusion, and the anemic cat is more sensitive to volume overload. There is no specific trigger PCV, but rather the overall clinical picture with a PCV of <20% is used.

Blood Typing: The major feline blood group system is known as the feline AB blood group system and contains 3 alleles: type A, type B, and the extremely rare type AB (except Ragdolls). Type A is dominant over B. Thus, cats with type A blood have the genotype a/a or a/b, and only homozygous b/b cats express the type B antigen on their erythrocytes. In the extremely rare AB cat, a third allele recessive to the a allele and/or codominant to b allele leads to the expression of both A and B substances. AB cats are not produced by mating of a type A to a type B cat unless the A cat carries the rare AB allele. Cats with type AB blood have been seen in many breeds and domestic shorthair cats.

Most domestic shorthair cats have type A blood, but the proportion of type B cats can be substantial in certain geographical areas. The frequency of A and B blood types varies greatly between different breeds, but likely not much geographically in purebred cats. Kitten losses due to A-B incompatibility and changes in breeding practices influence the frequency of A and B in various breeds. Most blood donors have type A blood, but some places also keep cats with the rare type B and type AB as donors. All blood donors must be typed. Naturally-occurring alloantibodies have been well documented in type A and type B cats and require that blood typing be performed prior to both blood transfusion and breeding to assure appropriate blood compatibility.

Cats have naturally-occurring alloantibodies. All type B cats have very strong naturally-occurring anti-A alloantibodies, which can be detected by hemolysis and hemagglutination assays. Kittens receive alloantibodies through the colostrum from type B queens and develop high alloantibody titers (>1:32) after a few weeks of age. These alloantibodies are strong hemolysins and hemagglutinins, and are of the IgM and, to a lesser extent, IgG classes. They are responsible for serious transfusion reactions and neonatal isoerythrolysis in type A or AB kittens born to type B queens. Type A cats have weak anti-B alloantibodies, and their alloantibody titer is usually very low (1:2), nevertheless they can also cause hemolytic transfusion reactions, but have not been associated with NI. Type AB cats have no alloantibodies. Furthermore additional blood group systems have been identified such as the common Mik red cell antigen in domestic shorthair cats and Mik-negative cats may also produce naturally occurring alloantibodies.

Serological testing relies on identification of surface antigens, leading to agglutination and hence can distinguish A, AB or B phenotypes. Several different reagents may be used but monoclonal antibodies against the type A and type B antigen are currently used in typing kits. A genetic test is also available for identification of the b allele, allowing identification of type B cats and carriers of this allele, but not distinguishing A or AB phenotypes.

Table. Blood type A and B frequency in cats in certain countries and breeds*

Domestic shorth. cats		Percentage (%) Type A	Type B	Purebred cats	Percentage (%) Type A	Type B
USA	Northeast	99.7	0.3	Abyssinian	84	16
	North Central	99.6	0.4	Am. shorthair	100	0
	Southeast	98.5	1.5	Birman	82	18
	Southwest	97.5	2.5	British shorthair	64	36
	West Coast	95.3	4.7	Burmese	100	0
Argentina		97.0	3.0	Cornish rex	67	33
Australia		73.7	26.3	Devon rex	59	41
India (Bombay)		88.0	12.0	Exotic shorthair	73	27
Europe				Himalayan	94	76
	Austria	97	3	Japanese Bobtail	84	16
	England	97	3	Maine Coon	97	3
	Finland	100	0	Norwegian Forest	93	7
	France	85	14	Oriental shorthair	100	0
	Germany	94	6	Persian	86	14
	Hungary	100	0	Scottish Fold	81	19
	Italy	89	11	Siamese	100	0
	Netherlands	96	4	Somali	82	18
	Scotland	97	3	Sphinx	83	17
	Switzerland	100	0	Tonkinese	100	0
	Turkey	7	25	Turkish Angora/Van	50	50

*Ignoring the rare AB cats in many breeds with type B cats

There are no universal donor cats. Donor and patient need to be typed, even if it is "only" a domestic shorthair cat. Simple AB blood typing cards (DMS Laboratories, 2 Darts Mill Road, Flemington, NJ) and chromatographic strip cartridges (Alvedia DME, Lyon, France and recently DMS) are available for in practice use.

Blood crossmatching tests: Blood incompatibilities have been recognized related to the AB blood group system and following blood transfusion through crossmatching cats or as a result of acute hemolytic

transfusion reactions. Standard laboratory tube and gel column crossmatching techniques, but also in-clinic gel tube (DMS) and strip kits are now available. Screening feline blood donors and patients for the presence of naturally occurring alloantibodies (AB and Mik systems) prove necessary in clinical practice. The presence of autoagglutination or severe hemolysis may preclude the crossmatch testing.

The major crossmatch tests for alloantibodies in the recipient's plasma against donor cells, whereas the minor crossmatch test looks for alloantibodies in the donor's plasma against the recipient's red blood cells. Mixing a drop of donor/recipient blood with donor/recipient plasma will detect A-B incompatibilities if typing is not available. However, proper techniques for crossmatching and experience are required to detect other less severe incompatibilities. A major crossmatch incompatibility is of greatest importance because it predicts that the transfused donor cells will be attacked by the patient's plasma, thereby causing a potentially life-threatening acute hemolytic transfusion reaction. As fatal reactions may occur with <1ml of incompatible blood, compatibility testing by administering a small amount of blood is not appropriate. This has been shown in experimental studies to result in fatal reactions. The major and minor crossmatch can show incompatibility prior to any transfusion due to the presence of naturally occurring alloantibodies in cats, not only for the AB but also the Mik and possibly other blood group systems.

Previously transfused cats should always be crossmatched, even when receiving blood from the same donor. The time span between the initial transfusion and incompatibility reactions may be as short as 4 days and lasts for many years (i.e., years after the last transfusion alloantibodies may be present). Obviously, a blood donor should never have received a blood transfusion to avoid sensitization.

Xenotransfusion: Occasionally anemic cats are given canine blood because either no feline blood is available or the feline blood is incompatible (AB, mik and other mismatch). In a recent study by the author's laboratory (Euler et al 2016) we determined that canine blood is incompatible and very short-lived (<4 days) in cats. Therefore, we do not recommend such xenotransfusions (Euler et al 2016). Apparently, Oxyglobin, a highly purified bovine hemoglobin solution should be again shortly available in the USA as it has been in Europe.

Feline blood donors: There are few commercial blood bank that offer feline blood products. Many hospitals have a small in-house colony and/or involve the staff's or client's cats. Healthy, young adult (1-8 years), good tempered cats of at least 4 kg lean body weight can be recruited. Due to the infectious disease risks indoor cats free of fleas and intestinal parasites are selected. A freely roaming cat in a veterinary hospital would not be a good donor candidate because of the potential of having acquired some infections from patients.

Blood donors must have no history of prior transfusion; have been regularly vaccinated and are healthy as determined by history, physical examination, and laboratory tests (complete blood cell count, chemistry screen, and fecal parasite examination every 6-12 months) as well as free of infectious diseases (testing depends on geographic area but may include regular FeLV, FIV, FIP, Hemomycoplasma, Cytauzoon testing. Donors should receive a well-balanced, high performance diet, and may be supplemented twice weekly with ferrous sulfate (Feosal, 10 mg/kg), if bled several times a year. Packed cell volume (PCV) or hemoglobin (Hb) should be >35% and >12 g/dl in cats.

Blood collection: Cats are regularly sedated e.g. with a combination of ketamine (10 mg), diazepam (0.5 mg), and atropine (0.04 mg) by intravenous injection. Some sedatives, such as acepromazine, interfere with platelet function and induce hypotension, hence they should not be used. Blood is collected aseptically by gravity or blood bank vacuum pump from the jugular vein over 5 to 10 minute period. Large plastic syringe containing 1 ml CPD-A or 3.8% citrate per 9 ml blood and connected to a 19 gauge butterfly needle is commonly used for cats. This represents an open collection system in which connections allow exposure of blood to the environment; because of the potential risk for bacterial contamination, blood collected via an open system should not be stored for more than 48 hours. The maximal blood volume to be donated is 40-50 ml blood (one typical feline unit) per ≥ 5 kg cat. We have developed a closed blood collection system that permits component preparation into packed red blood cells and fresh frozen plasma as well as storage (28 days of red cells, 1 year FFP). Blood components are prepared from a single donation of blood by simple physical separation methods such as centrifugation generally within 4 hours from collection.

Blood administration: The regular principles used in transfusing dogs are applied in cats. No food is given during the transfusion, and blood is administered separately without any drugs or other fluids. Because of the small volumes shorter tubing with a small filter are used instead of the large infusion sets. Despite assuring blood compatibility particular attention is given to the first few milliliters infused. Monitoring is done like in dogs. Transfusion reactions may be related to blood type incompatibilities but also allergic reactions, physical hemolysis, hypocalcemia, and infection.

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SOCIAL MEDIA FOR VETERINARIANS

AUDIT OF YOUR WEBSITE & SOCIAL MEDIA

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" Google only loves you when everyone else loves you first. " - **Wendy Piersall**

The World Wide Web can be a mysterious and confusing place. Is Google just a website or is it an all-seeing Internet-empire? Does my veterinary practice need active Social Media accounts to help me reach a wider audience and succeed online? And how important is it for me to optimize my visibility online anyway?

Luckily, I'm here here to answer all of your questions and help you navigate the myriad of digital elements that come with running a successful veterinary practice in the 21st century. To answer briefly; Google is both a website and an empire, Social Media is an imperative medium for most if not all businesses, and Search Engine Optimization is crucial to your success. I'll be focusing on the later in this month's blog and will provide you with 3 simple and proven steps to improve your **S**earch **E**ngine **O**ptimization (SEO) strategy.

Simply put, SEO means page visibility, and your natural page rank across online search engines. SEO can result in an immediate boon to your practice, as a surprisingly large percentage of business starts online with a simple Google search. If you've heard me speak before then you already know Google is the #1 search engine (by far). **In fact, in 2014 alone we collectively searched Google trillions of times**. We searched so much that it prompted Google to release a "Year In Search" compilation, which as a side note, we recommend watching since it just might make your day.

Why focus on Search Engine Optimization and make sure that this element is as fine-tuned as the rest of your business? Well, if you remember our "Safeguarding Your Online Reputation" article, you remember that Google has "Crawlers" which comb through billions of web pages from all across the Internet, algorithmically analyzing, assessing, ranking, and indexing web pages. They rank pages based on a multitude of factors (known as an algorithm), but the key to a successful page is to **leverage the factors that you can control**. A better page rank means a stronger reputation and more visits to your website, which ultimately will convert to more pet owners walking in the door.

That last line is the crux, so hang on to the takeaway: **a successful SEO strategy can convert for your business and thus provide more money in your pocket**. Great! I understand. Now, how do I actually implement this and apply it in real-time?

Top 3 Tips To Improve Your SEO

(1) Secure as many positive Google Reviews as possible for your business. Again, we encourage you to read our previously published "Safeguarding Your Online Reputation" for a full analysis of Google Reviews, but simply put, this is a crucial element to enhanced SEO. Reviews instantly provide credibility to your business, while bumping you up in Page Rank.

(2) Head over to the 'Moz Local' website and register your business. This simple tool will help local search engines and new pet owners find your practice online, all while enhancing your SEO efforts. While you're at it, go ahead and register your business on Yelp, Google+ and Bing. All of these tools help to enhance your Page Rank, and will allow your business to be recognized easily across each search engine.

(3) Make sure your content has a naturally included list of keywords that people will use when they search for you. These keywords shouldn't be forced or crammed excessively into your website. They should however, be clear, confident and relevant. A few solid keywords might be "**animal hospital**", "**veterinarian**" "**animal clinic**", and the city or region you're doing business in. All of these words help Google pinpoint who you are, what you do, where you're located, and effectively deliver results to users with more accuracy.

This is only a small fraction of what is involved in Search Engine Algorithms (and yes, they are constantly evolving). However, these steps should empower you to claim your listings, dive into basic SEO and even understand a bit of what works behind the scenes at Google.

Keeping Up With Google: How to Leverage Recent Changes to Enhance Your Web-Ranking

" If it isn't on Google, it doesn't exist. " – Jimmy Wales

When you work in Information Technology, keeping up with Google is a lot like checking the weather. You can avoid keeping up with what's coming if you want, but by doing so you risk being caught off-guard in a downpour. Simply by thumbing through the most prominent tech blogs on a routine basis (in conjunction with setting up a few Google Alerts) it's easy for me to keep track of what major changes are coming about in IT, and on the web as a whole. This month, I want to share with you a major change that's recently occurred to the Internet-giant Google, and their approach to web-ranking. We'll explore what you can do to leverage their most recent changes, and explore how to utilize these changes to enhance your veterinary practice and garner more business than ever.

The announcement that Google would be factoring mobile compatibility into their comprehensive web-rankings beginning April 2015, caused immediate shockwaves across the web. No modern day business is truly exempt from these changes, that is unless of course they don't have a website, or use the Internet at all (which is an entirely different issue). Modern, competitive veterinary practices rely more than ever on the web, and consequently, they rely on Google too. That's because Google sets the tone for what exists on the Internet as a whole, controlling, monitoring and distributing an unthinkably large amount of data each day. In fact, Google possesses over 65% of all search market share (yes, feel free to confirm this statistic with a Google search), effectively making or breaking what exists on the web in any capacity. While it may be easier than ever to start a blog or launch a website, it's much harder to truly thrive on the web. This is why my goal is to help you adapt and leverage the absolute latest technological developments to enhance your veterinary practice.

As of recent, Google has overhauled their web-ranking system, and is now incorporating the functionality of websites on mobile devices as a major component of Google Rankings. I know that pet owners and veterinarians alike use their smartphones more than ever, and Google has inevitably caught onto this trend. If a website is not optimized for mobile, it will now fall dramatically in search rankings conducted on mobile devices to reflect this shortcoming. In other words, if your veterinary practice website is not comprehensively optimized for both mobile and desktop, your business is at risk of losing traction and falling behind the competition. On the other hand, practices that can rapidly adjust may be able to outpace competition that has faltered behind such adjustments. If practices choose not to adapt to the new formula, they risk losing out on a significant amount of business.

The trend of accessible data on the web is now skewed toward mobile, and Google has fully adjusted to this reality. Comprehensive Simply Done Tech Solutions analytics finds that on average, 33% of all organic (non-paid) traffic generated to a veterinarian's website is from mobile. It's important to note that this percentage is increasing over time, and in some cases, veterinary practices actually secure more mobile traffic than they do via desktop. So what makes a good mobile website, and how can you ensure that you're well equipped moving forward? Look below for some examples of a successful mobile build, and read on for a step-by-step guide on how to optimize your veterinary practice's website.

A good mobile website strips down the best of a desktop site, and makes it easily available for the user to access on their mobile device. Mobile devices are smaller and more compact, and thus the aesthetics and build of a proper mobile website reflects this. A good mobile site will emphasize big buttons that are accessible on the go. This might mean a tap-to-call button that lets the user call your primary business line with a single finger-tap. The same functionality can be lent to locators and GPS features that can ostensibly bring a new client right to your door. A proper mobile build will show off the best of your brand with a custom color pallet and seamless links to Social Media. Well-conceived and implemented mobile builds will also boast simple bits of information that any prospective or current customer might need to know (think location, phone number, slogan). Now that we've got the basics down, let's look at a simple step by step process to adapt your website to mobile, today.

3 Simple Steps for Adapting to Mobile

(1) Check your current websites mobile compatibility with the Google's Mobile-Friendly Test Tool. Simply copy and paste your primary web URL into the tool to begin.

(2) Analyze your results, which is made easy by Google. You might be in good shape with your existing website, or you may receive a "Not mobile-friendly" rating. If the later rating is given, Google will provide you with several options to help make your website more mobile-friendly than before.

(3) Based on your results, you may wish to contact your current Web Developer, or explore an array of other options. There are many website development companies in the veterinary industry that have an easy approach to mobile-optimization.

In an age of rapid digital transformation, keeping up with the breakneck speed of change can be difficult. By staying tuned to SimplyDoneTechSolutions.com, you'll know what changes are coming your way, and how to adapt accordingly. This time it's mobile-compatibility, but who knows what lies around the bend. I know that your veterinary practice deserves to thrive now, and into the future.

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SVA INFECTIOUS DISEASES

FLEAS AND FLEA-BORNE DISEASES UPDATED

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Introduction

Carpets and companion animals are frequently seen in modern living rooms. However, the combination of the two gives a perfect condition for flea infestation in dogs and cats in a residential house. It is important to control flea infestation in companion animals and larval stages on the ground to prevent severe allergic dermatitis and the transmission of flea-borne pathogens that can cause serious disease in animals and/or in humans. Understanding the biology and ecology of flea is critical, as it affects what strategies will be effective to control flea infestation. A wide range of flea control products is available in the form of sprays, spot-ons, collars, oral tablets and injections. Used correctly and understanding the mechanism of kill will ensure safe and highly effective flea control.

Flea biology

Adult fleas live on animals. Unless they're dislodged through grooming, fleas become permanent residents of their acquired host. Out of over 2500 distinct species in Siphonaptera, the two species of most importance for veterinary practitioners are the cat flea **Ctenocephalides felis** and the dog flea **C. canis**. Fleas have a shiny, glossy appearance because they have strongly sclerotized and chitinized bodies. Adults are laterally flattened, appearing as if they've been compressed side-to-side. The flattened body is an adaption which helps them move forward through host fur. Fleas, unlike most other insects, don't have wings [1].

Ctenocephalides felis felis is the most common species associated with domestic dogs and cats in the USA and most parts of the world. The term "cat flea" is the common name of **C. felis** and doesn't refer to all fleas recovered from cats. The species **C. felis felis** is characterised by an acutely angled frons and head appears 'pointier' than **C. canis** [2].

Unlike larval stages on the ground, both male and female adult fleas rely exclusively on host blood for living, so the mouth parts of a flea are specialized for blood feeding. While imbibing blood, salivary ducts open to introduce anticoagulant saliva to the wound. Flea legs are long and well-adapted for jumping. The hind legs are much longer than the others, and are connected to specialized internal structures for leaping. A highly elastic protein called resilin enables their incredible jumps, not leg muscles [3].

Adult fleas imbibe much more blood than they can use. As a result, fleas produce large amounts of feces, consisting largely of undigested blood. Feces from adult fleas which is flea dirt, is the primary food source of flea larvae[4]. A bit weird, but somewhere in nature close to our living, mother's poop is baby's food, and is bloody red instead of milky white. Fleas excrete feces in two different forms: Spherules and coils. Spherules are round and 0.07-0.25 mm in size. They can also be diarrheic. Coils are long with an average length of 0.84 mm. Newly emerged adults produce spherules. By the tenth day of feeding, their poop is mostly in the form of coils. Finding flea dirt on a dog or cat is one of the best ways to diagnose an infestation. If it's truly flea feces, the black speck will smear red or dark yellow when rubbed on a wet paper towel. Flea dirt dries in irregular shapes and gets embedded into pet fur. The dry blood dislodges when animals scratch and groom themselves. As a result, it's most common to find feces (and eggs) in areas where pets commonly rest and groom. Coils contain 33% more protein than spherules. The spherules are thought to be preferred by young larvae, while the protein-rich coils are more suitable for 3rd instars.

Adult fleas always stay on their host and females lay eggs directly on their host after mating with male fleas. Roughly one flea egg is produced per hour. The eggs are initially wet and sticky, but they dry quickly and become non-adherent. Around 60% of eggs fall from the host within two hours of being laid. Flea eggs measure 0.5 mm in length, and 0.3 mm in width. To survive, flea eggs must fall onto substrates with a warm, humid microclimate, such as carpeting. If the microenvironment is too dry, eggs willshrink and die. Flea eggs develop rapidly in warm, humid environments. They'll hatch within one and half days when conditions are optimal. Ideal conditions occur at temperatures near 89.6°F (32°C), and humidity between 75-92%. A relative humidity below 50% is often lethal while 80% of flea eggs survive when relative humidity exceeds 50%. In homes, it takes 2 to 3 days before flea eggs hatch. Most eggs and larvae live in carpeting because the temperature and humidity are well secured. The microclimate within the carpet fibers is near ideal for developing fleas. A sharp spine projects from the front of the head. It helps a larva rupture its egg shell and hatch. The egg burster spine is only found on first instars. It's lost in the first molt. Newly hatched flea larvae are 2 mm long, growing to a final length of 5 mm. The larvae resemble worms or maggots. Dog or cat flea larvae develop through three stages, taking the form of 1st, 2nd, and 3rd instars. They molt once between each stage. Dried blood feces from adult fleas is the primary food source of larvae. As a larva feeds on flea dirt, its gut turns a dark red to purple color.

A mature larva will move to an undisturbed location to spin a cocoon. The cocoon's sticky silk collects debris from the environment, such as carpet fibers, and becomes camouflaged. Mature flea larvae are 4-5 mm long, and 0.5 mm wide. While forming cocoons, the larvae fold themselves in half. Flea cocoons are envelopes of thin, white, silk-like material. The fibers are soft, moist, and sticky. They're wrapped loosely around the larvae. As a result, environmental debris easily adheres to cocoons. This detritus may consist of sand, dust, soil, carpet fibers, or any other small fragments of dry material. The larvae purposefully collect and integrate these particles into their cocoons with a few silk threads. Flea cocoons are difficult to detect. Adhering debris originates from the same environment where the cocoons rest, giving them a near perfect camouflage. Within homes, larvae pupate at the base of carpeting. Once adulthood is reached, fleas can remain in a motionless, dormant state inside their cocoons. This quiescent period lasts up to 5 months, but it ends when a nearby host triggers their emergence. The presence of heat or pressure causes them to exit the cocoons within 5 seconds. After emerging, a flea will climb atop nearby objects, such as carpet fibers, where it's able to jump onto a passing host. There, the flea orients itself towards sources of light. As a result, fleas tend to gather near openings where light enters, such as vents, crawl spaces, window sills, and other entrances.

The complex flea life cycle means that there are a number of challenges for veterinarians who help clients control fleas in their homes: the physical resilience and rapid multiplication in flea numbers; their ability to find alternative hosts; and the presence of environmental conditions in the home that are conducive to flea survival. In addition, there is a need to select an appropriate form of treatment from among the many options available and to manage the client's expectations of what is possible to achieve. Clients need to be aware which strategies will and will not be effective: e.g. neither removing pets from a flea-infested house nor leaving the house vacant (to starve fleas) is likely to work, since immature stages live for a year or more, can survive winter temperatures and will only hatch when they feel vibrations (from a passing human, animal or vacuum cleaner), or sense the CO2 given off by a potential host [5].

Flea-borne diseases

Fleas are important clinically as causes of pruritus, flea bite dermatitis, and in young animals with severe infestations anemia. Fleas are intermediate hosts for filarial nematodes and the tapeworm **Dipylidium caninum** and vectors for various pathogens, including **Bartonella henselae, Rickettsia felis, Haemoplasma** species and **Yersinia pestis**. Cat fleas can also transmit **Rickettsia typhi**, a causative agent for murine typhus - normally transmitted by the rat flea **Xenopsylla cheopis** [6].

The cat flea, **C. felis**, is the most frequently encountered parasite on both cats (98%) and dogs (93%), and is also the flea species with the most potential for transmitting zoonotic diseases. **Yersinia pestis** causes both bubonic (abscesses and lymphadenitis) and pneumonic forms of plague, with clinical signs of fever, inappetence, lymphadenopathy and subcutaneous abscesses. Cats seem to be more clinically susceptible than many species. In feline patients the abscesses can be difficult to distinguish from cat bite abscesses. In around 10% of human cases the disease is transmitted by cats, through passing on infected fleas, or directly via scratches, bites or air droplets.

Cats are infected with the tapeworm **D. caninum**, which has a worldwide distribution, via **C. felis**. Fleas ingest tapeworm eggs and the tapeworm is transmitted when an infected flea is swallowed by a cat or dog, after which there is a pre-patent period of 2–3 weeks. In most infected pets the condition is asymptomatic, although the proglottids may be detected in the faeces or on the perianal area. Tapeworms may cause anal pruritus, which may lead the affected animal to 'scoot' along the ground, rubbing its anal area, which can embarrass dog owners. Other signs can include diarrhea, weight loss and failure to thrive. Control is achieved through anthelmintic dosing with praziquantel, along with flea treatment to prevent reinfection and hygiene measures to remove the contaminated faeces.

Bartonellosis is the main flea-borne bacterial disease circulating in pets - **Bartonella henselae** or **B. clarridgeiae** are the usual variants in cats while **B. vinsonii** subsp. berkhoffii is the most common in dogs, along with **B. henselae**. The prevalence is much lower in dogs than cats and there is some suggestion that it is an opportunistic pathogen in this host. Bartonellosis is normally transmitted through skin contact with the contaminated flea dirt, and the disease has a worldwide distribution. Testing of healthy animals is currently not recommended. Clinical signs may appear when the bacteria are released from the infected cells into the circulation and will consist of endocarditis in cats and dogs, and other inflammatory diseases such as recurrent pyrexia, uveitis and immunemediated polyarthritis. The bacterium may also cause vascular proliferative disease, particularly in human patients. This pathogen appears to have co-evolved with the feline host, which may show no obvious clinical signs, but due to its regular release from infected cells it may have role in various chronic disease conditions.

Another emerging zoonotic disease is fleaborne spotted fever caused by the **Rickettsia felis** or **R. typhi**. The pathogen has a worldwide distribution and is transmitted via bites from infected fleas, which are capable of vertical transmission through the egg. Cats are rarely symptomatic and are not known to be reservoirs. Dogs

may be a source, based on limited evidence from countries such as Australia. Humans may be infected through bites or contact with infected flea faeces on skin abrasions and will show non-specific signs of fever, rash, headache and myalgia.

Fleas may also be a source of mycoplasmal disease, with three Mycoplasma species (**M. haemofelis, M. haemominutum** and **M. turicensis**) present in cats and two in dogs (**M. haemocanis** and **M. haematoparvum**). Of these, **M. haemofelis** is the most pathogenic, causing severe haemolytic anaemia, especially in young animals. The other species are only likely to produce anaemia in cats with concurrent disease, or in immunocompromised or splenectomised patients. Haemoplasmas are known to be ingested by **C. felis** while feeding and may be detected in flea faeces, which is one possible mode of transmission, and there is recent experimental evidence of **M. haemofelis** transfer by fleas during feeding.

Flea-borne human diseases

Rickettsia typhi and **Rickettsia felis** are two major flea-borne rickettsiae of humans that are distributed throughout the world. **Rickettsia typhi** is acquired by fleas while feeding on rickettsemic rats. The organism infects the midgut epithelium of the flea and is shed in the feces, where it is transmitted to humans by the inoculation of **R. typhi**-laden flea feces onto flea bite wounds or mucous membranes[6]. Murine typhus is characterized by the abrupt onset of fever with accompanying headache, chills, myalgia, and malaise. Rash, which is the sign that often prompts a clinician to consider a rickettsiosis, is absent in 50% and may be present in as few as 20% of those with darkly pigmented skin. Since the mid-1990s, it has been increasingly recognized as a cause of human infection throughout the world. The primary reservoir and vector of **R. felis** is thought to be **C. felis**.

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ONCOLOGY

PALLIATIVE CARE AND NUTRITIONAL SUPPORT IN CANINE AND FELINE CANCER PATIENTS

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PALLIATIVE CARE AND NUTRITIONAL SUPPORT IN CANINE AND FELINE CANCER PATIENTS

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Learning objective: To develop a framework for instituting a palliative care plan including pain relief and nutritional support in dogs and cats with cancer, and determining when surgery and radiation therapy can be applied with a palliative goal.

Definition and general approach to palliative care

Palliative care is aimed at improving quality of life and relief of suffering rather than specifically treating the underlying disease.

In the case of animals with cancer, offering palliative care only as an option is appropriate in cases where the condition is likely to be terminal and owners elect not to pursue treatment, or treatment is not successful. In human studies, early palliative care is associated with not only improved quality of life at the end of life but actually with improved survival time. Early discussion of palliative care also allows carers to prepare, resulting in reduced episodes of prolonged grieving and major depression following the death of the patient.

Many factors go into owners' decisions when choosing treatment for pets with cancer. These include treatment related factors (cost, side effects, time commitment), disease related factors (prognosis with different treatments), and patient factors (comorbidities, amenability to different levels of treatment, age). Age is commonly cited as a factor in owner's decisions though it may not directly affect the suitability of treatment options otherwise.

Suggested steps in developing a palliative care plan

- Client education - discussion of all of the options for diagnostic testing or treatment, and prognosis. Consideration of the bigger picture is necessary - for example, if the owners are not going to pursue specific treatment for a dog with osteosarcoma, will taking chest radiographs change the plan?
- Determination of the owners goals and beliefs. If owners goals are unrealistic, this should be addressed clearly and honestly, with empathy. Determining owners beliefs about euthanasia may be appropriate at this time.
- Developing a personalised treatment plan - patient assessment with regard to hydration, nutrition, pain, mobility, mood and engagement to determine which need to be addressed, client assessment with regard to their willingness and ability to provide care, plan should be agreed upon by vet and owner. Treatment protocols should be simplified as much as possible and instructions should be clear. Owners should feel able to express concerns about their ability to deliver the recommended care.
- Implementation and reassessment: Once a plan has commenced, frequent reassessment is required to ensure that goals of the owner and needs of the patient are being met, and that quality of life is maintained as much as possible. Tumour progression alone does not mean that palliation is not successful, as long as quality of life is maintained. Use of the animal hospice care pyramid and/or the HHHHHMM quality of life scale may assist in assessing for changes in quality of life over time. At the beginning of the process, having owners make a list of the things that most impact quality of life for their pet (positive and negative) and then having them assess their pet regularly for these key behaviours may allow detection of changes in quality of life over time.

Pain management in cancer patients:

Pain is a major concern of human cancer patients and of owners of pets with cancer. Instituting analgesia early in animals with tumours likely to be painful is an important part of palliative care and of cancer treatment in general. Owners may not always recognise pain in their animals and careful questioning and client education is important. In some cases, an analgesic treatment trial may help an owner to recognise that their pet was painful. Drug therapy is the mainstay of pain management in veterinary oncology and the WHO cancer pain ladder is a reasonable approach. For mild pain, an NSAID is recommended +/- adjuvant e.g. gabapentin. For more severe pain, addition of opioids is recommended. Multimodal management is recommended, with combinations of drugs and of modalities (i.e. massage or other physical therapy along with drugs). Using multiple drugs improves pain control and allows lower doses to be used of individual drugs. Adjuvant drugs are generally weak analgesics alone but when used in combination with other drugs can be beneficial. Simplifying timing and frequency of dosing will improve compliance when prescribing multiple medications.

For tumours that are affecting quality of life due to local effects, surgical removal or radiation therapy may improve quality of life even in cases with metastatic disease. For example, in appendicular osteosarcoma improvement in quality of life can be achieved with amputation or palliative radiation therapy due to relief of local pain even though these modalities may not improve overall survival. Intranasal tumours are likely painful due

to their invasive nature and will often respond very well to palliative radiation therapy, which has a low risk of acute side effects. When removing tumours for purely palliative purposes, aggressive resection to achieve margins should not be a major factor in the approach but if margins are achievable without increasing morbidity this may be reasonable.

Nutritional support in cancer patients:

Maintenance of adequate nutrition is a key factor in assessing quality of life in dogs and cats with many illnesses, including cancer. In the specific setting of palliative care in the veterinary oncology patient, both strategies to increase voluntary intake and strategies bypassing the need for voluntary intake (e.g. feeding tubes) may be used, but clear consideration of goals and quality of life is required. Anorexia or hyporexia in dogs and cats with cancer may be caused by inflammatory response to the tumour, pain or nausea, obstruction (oral or other GI tumours), or effects of chemotherapy. Even in patients where caloric intake appears adequate, disordered metabolism can cause loss of condition (cancer cachexia), though this does not seem to be as common in dogs as in people. It may be more common in cats.

The first step in developing a nutritional support plan is a baseline assessment including body weight, body and muscle condition score, and dietary history. Weight alone is not sufficient to fully assess nutritional status as weight gain with significant loss of body and muscle condition may be seen due to e.g. tumour growth or ascites.

If nutritional supplementation is required, resting energy requirement (RER) must be calculated. There are several formulae for this. One approach, used at the University of Melbourne, is:
- RER in kcal/day =
- Body weight (kg) x 30 + 70 for dogs and cats
- 70 x Body weight (kg)^0.75 for dogs > 45kg

An alternative approach for lean/active cats < 5kg cats is: RER = Body weight (kg) x 60

For hospitalised patients, aiming for RER is reasonable. Increased energy requirements are expected for more active animals at home or those in whom extensive tissue repair is taking place and maintenance energy requirements can vary from approximately 1.2-1.8 RER depending on activity level and neuter status. Regardless of which calculation or approach is taken, regular reassessment is required to assess whether current caloric intake is sufficient.

Recommended steps for increasing caloric intake in palliative care:
1. Address pain, nausea or other underlying causes where possible
2. Coaxing (e.g. hand feeding, dietary modification)
- Syringe feeding is usually not practical

1. Pharmacologic approaches (1) including:
   1. Mirtazapine - can cause behavioural changes e.g. vocalisation or agitation. Should not be used concurrently with cyproheptadine. Is absorbed transdermally in healthy cats with effective appetite stimulation, although the appropriate dose has not been determined (2)
   2. Cyproheptadine - may take a few days to be effective, may cause sedation or paradoxical hyperexcitability
   3. Capromorelin - recently approved (in dogs), ghrelin receptor agonist. Can cause diarrhoea, vomiting, and excess salivation (3)
   4. Prednisolone
2. Feeding tubes

In my opinion, feeding tubes should be considered in palliative patients only in select circumstances i.e. where other quality of life factors are considered good (e.g. pain is controlled, activity and mobility are acceptable) but inadequate caloric intake persists. Feeding tubes are not recommended where inadequate intake is due to effects of cancer that are not otherwise being addressed e.g. in oral tumours where the animal is not eating well due to pain. Oesophagostomy and gastrostomy tubes are the most common approaches used. Oesophagostomy tubes typically last for weeks to months. Gastrostomy tubes should be considered when anticipated need is for > 6-8 weeks. Placement of gastrostomy tubes is more challenging than oesophagostomy tubes and typically requires either an endoscopic or surgical approach. Naso-oesophageal or nasogastric tubes are quick and non-invasive to place but are usually short term (days) and can only accommodate liquids. These approaches may be used in cases where a definitive diagnosis is pending or while assessing for rapid response to treatment before making longer term decisions.

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ONCOLOGY

PALLIATIVE CARE AND NUTRITIONAL SUPPORT IN CANINE AND FELINE CANCER PATIENTS

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ENDOCRINOLOGY

GLUCOCORTICOID-DEFICIENT HYPOADRENOCORTICISM IN DOGS

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ENDOCRINOLOGY: ENDOCRINE CALCIUM DISORDERS

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Small animal patients presenting with a primary complaint of hypercalcemia can often be a diagnostic challenge. Typically, the clinical signs are insidious and nonspecific. A thorough work-up, sometimes necessitating repetition in diagnostic steps may be required to determine the etiology of the hypercalcemia. Treatment should be directed toward resolution of the underlying disease.

Serum ionized calcium is regulated within a narrow range that is controlled by two principal hormones: parathyroid hormone (PTH) and the active form of vitamin D, calcitriol. PTH, synthesized by the chief cells of the parathyroid gland, acts on the bone to increase resorption and calcium mobilization. It also works on the kidney to increase calcium reabsorption and decrease phosphorous reabsorption, and to increase the formation of calcitriol. The net result of PTH secretion is to increase calcium and decrease phosphorous in the serum. Vitamin D is formed from cholesterol precursors in the skin by the action of sunlight. The active form, calcitriol, is formed in the kidney. Vitamin D works primarily on the intestine to increase calcium and phosphorous absorption. The net effect of vitamin D is to increase plasma calcium and phosphorous.

Causes of hypercalcemia

The most common cause of hypercalcemia in dogs and cats is malignancy. The neoplastic diseases reported to cause hypercalcemia include lymphoma, anal sac apocrine cell adenocarcinoma, multiple myeloma, mammary carcinoma, thymoma, oral melanoma hepatoblastoma, chronic lymphocytic leukemia, and nasal carcinoma, with lymphoma being the most common. Hypercalcemia seen with malignant disease probably results primarily from enhanced osteoclastic bone resorption mediated by factors systemically released by neoplastic cells. One such factor has been has been identified in the dog as parathyroid hormone related protein (PTHrP) which is identical to PTH at the N-terminal region and can bind with equal affinity to PTH receptors. By activating PTH receptors, PTHrP causes

hypercalcemia by a similar mechanism as that of an excess of PTH. Osteolysis from primary bone tumors are a rare cause of hypercalcemia.

Other common disorders associated with hypercalcemia are hypoadrenocorticism chronic renal failure, and urolithiasis in cats. Although in these retrospective studies, a significant number of Addisonian animals were reported to be hypercalcemic, the pathogenesis of the hypercalcemia is unknown. Dogs and cats with chronic renal failure demonstrate hypercalcemia uncommonly, and it is thought that decreased renal calcium excretion and PTH-mediated osteoclastic activity from renal secondary hyperparathyroidism may be responsible. In the cat, idiopathic hypercalcemia of mild to moderate severity has been described. In some cases it has been associated with calcium oxalate urolithiasis and has resolved with discontinuation of an urinary acidification diet. The pathogenesis of hypercalcemia with this condition is unknown.

Endocrine etiologies of hypercalcemia in the dog and cat include primary hyperparathyroidism and hypervitaminosis D. Primary hyperparathyroidism may result from parathyroid hyperplasia, adenoma or adenocarcinoma, which are all rare in small animals. Hypervitaminosis D, most commonly results from vitamin D-containing rodenticides or medications, but may be seen with over supplementation with vitamin D, especially in small dogs being given human vitamins. Infectious and inflammatory disorders such as blastomycosis, coccidioidomycosis, feline granulomatous disease, endometritis, and schistosomiasis rarely are reported to cause hypercalcemia in dogs and cats. A recent report also details hypercalcemia following renal transplantation in the cat.

Clinical signs

How does one recognize a hypercalcemic patient? The clinical presentation of hypercalcemia can range from the animal with no clinical abnormalities in which hypercalcemia was found on a routine chemistry to a severly weak or even comatose one. Usually, the clinical signs of hypercalcemia are insidious and so mild that many owners fail to recognize that there is anything wrong with their pet. The most common clinical sign of hypercalcemia is polyuria/polydipsia (PU/PD). This is a direct effect of hypercalcemia on the concentrating ability of the kidney; however, hypercalcemia can also cause acute or chronic renal failure, also resulting in PU/PD. Hypercalcemic animals may also present with signs of lower urinary tract disease since they are predisposed to urinary tract infections and the formation of calcium uroliths. Other less commonly recognized clinical signs include: muscle weakness/atrophy, depression, anorexia, vomiting, constipation, bone pain, pathological fractures, and cardiac arrhythmias. Signs related to specific tumors may also be present.

Evaluating the Hypercalcemic Patient

The clinical approach to the diagnosis of the hypercalcemic patient requires patience and thoroughness. A detailed history should be obtained and questions regarding the possible exposure to rodenticides should be included. Owners should also be questioned about any nutritional supplementation they may be using that may contain vitamin D. The first thing that needs to be established is that the patient is truly hypercalcemic. Since approximately ½ of the plasma calcium is complexed or protein-bound, the total calcium level must be evaluated in light of the serum albumin concentration. The most ccurate way to determine serum calcium levels is to measure ionized calcium in the serum. It is this form that the body regulates through PTH and Vitamin D activity.

Once the presence of hypercalcemia has been established, ruling in or out the differential diagnoses listed above should be attempted. Physical examination should be thorough and include palpation of the neck area for parathyroid masses. Although this is a low-yield procedure since the majority of parathyroid masses are too small to be palpated externally. Peripheral lymph nodes should be carefully evaluated for size and firmness, changes in which may indicate the presence of lymphoma. Abdominal palpation should be thorough including evaluation for masses and kidney size. Skeletal pain should be noted that may signal the presence of bone disease. Anal sacs should be thoroughly palpated to check for the presence of anal sac adenocarcinoma.

Biochemical work-up should be initiated by a CBC, chemistry profile, and urinalysis. The leukogram may reveal a leukocytosis that may signal an infectious or inflammatory process. The presence of lymphocytosis or of abnormal lymphocytes may suggest the presence of lymphoma. The hemogram may reveal a nonregenerative anemia that may indicate the presence of chronic disease, renal failure,or bone marrow invasion of neoplastic cells. A chemistry profile will allow evaluation of azotemia that may indicate renal failure. The phosphorous concentration must be carefully evaluated since hypophosphatemia along with hypercalcemia may indicate hyperparathyroidism. Hyponatremia or hyperkalemia may suggest the presence of hypoadrenocorticism, and hyperglobulinemia may signal multiple myeloma or chronic infection. A urinalysis should always be performed in order to fully evaluate the chemistry profile. Unconcentrated samples or isosthenuria may reflect the lack of renal concentrating ability caused by direct effect of hypercalcemia and not necessarily the presence of renal failure. A sediment examination should be performed to determine the presence of crystalluria or urinary tract infection. Urine culture should be performed if indicated.

Since neoplasia is the most common cause of hypercalcemia, this should be looked for. PTHrP is sometimes available for the dog and cat and serum samples looking for the presence of this malignancy-related hormone should be submitted if possible. Imaging studies should be performed to look for metastatic disease in the lungs and radiographs and/or ultrasound to look for evidence of neoplastic disease in the abdomen.

If results of the studies outlined above do not reveal the etiology of the hypercalcemia, parathyroid disease should be suspected. If the serum phosphorous level is low, this is further indication of hyperparathyroidism. Hyperparathyroidism may be diagnosed by measuring PTH hormone levels. An ionized calcium most be run on a sample obtained at the same time in order to evaluate the PTH level since PTH is regulated based on the serum Ca^{2+}. In order to diagnose hyperparathyroidism, an inappropriately elevated PTH level must be detected as compared to serum Ca^{2+}. Serum vitamin D levels can be checked to rule-out unobserved rodenticide ingestion or inadvertent supplementation with vitamins. If hyperparathyroidism is suspected, an ultrasound examination should be performed to try to identify a parathyroid tumor. Scintigraphy using technetium may provide an additional, nonivasive imaging technique if nothing can be seen on ultrasound examination. In some cases, surgical exploration may be the only way to identify parathyroid disease.

During the quest for diagnosis, the hypercalcemia must be addressed since hypercalcemia may result in tubular degeration and necroses of the kidney. Additionally, with elevations in serum phosphorous levels, hypercalcemia may lead to precipitation of calcium/phosphorous crystals in soft tissue structures. Treatment for hypercalcemia includes intravenous saline diuresis and lasix. In refractory cases, calcitonin is very effective in lowering serum calcium levels, although its effects are transitory. Although glucocorticoids also promote calciuresis and thus are effective in treating hypercalcemia, they should not be used until an etiology is determined since their use can confound the diagnosis of lymphoma, a chief rule-out for hypercalcemia. Bisphosphonates are a new class of drugs just starting to be used in veterinary medicine. Pamidronate and zolendronate has been used intravenously in dogs and cats with good results. These compounds are especially helpful in controlling bone pain associated with malignancies. Success has also been reported with oral alendronate (Fosamax); however, care must be taken to avoid esophagitis and stricture especially in cats.

WSV18-0042

WAVMA ORNAMENTAL FISH DISEASES

THERAPEUTICS IN AQUATIC VET MEDICINE

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THERAPEUTICS IN AQUATIC VET MEDICINE

Abstract

In fish, therapeutic agents are used for the treatment of a variety of infectious diseases. The presentation will give practical advice for special considerations when dealing with different pathogens, different fish species and in different environments.

43RD WORLD SMALL ANIMAL VETERINARY ASSOCIATION CONGRESS
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WSV18-0198

SVA INTEGRATIVE MEDICINE & ACUPUNCTURE

A CASE STUDY ON SKIN ALLERGY

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CASE STUDY: APPLICATION OF ACUPUNCTURE FOR SKIN ALLERGIES

Introduction

Bao Bao, a Golden Retriever, was presented with serious pruritus and would constantly scratch at her burning sores and her scaly skin all over her body, even at night. As her allergies became worse, her skin also became more red and inflamed.

Objective

The objective was to stop skin allergies from becoming worse and to improve skin condition.

Methods

Bao Bao's condition and symptoms were understood from a Traditional Chinese Medicine perspective and acupuncture was performed accordingly.

Results

Within 3 months of treatment, most of her skin has recovered and with only a few occasional hotspots.

Conclusion

The method "circle the dragon" can be effective in helping to quickly alleviate serious skin allergies.

WSV18-0080

ENDOCRINOLOGY

ENDOCRINE CALCIUM DISORDERS

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ENDOCRINOLOGY: GLUCOCORTICOID-DEFICIENT HYPOADRENOCORTICISM IN DOGS

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The adrenal cortex consists of distinct zones that secrete different hormones based upon enzymes available in that region. The zonae glomerulosa is responsible for the secretion of mineralocorticoids, and the zonae fasciculate and reticularis secrete glucocorticoids. The primary mineralocorticoid and glucocorticoid secreted in the dog are aldosterone and cortisol, respectively. Aldosterone secretion is primarily stimulated by activation of the renin-angiotensin system and hyperkalemia with only a small role of ACTH during severe volume depletion. Cortisol secretion from the adrenal gland is primarily regulated by the hypothalamic-pituitary-adrenal (HPA) axis with ACTH providing the primary control.

Primary hypoadrenocorticism results from destruction of the adrenal cortex such that cortisol and aldosterone are not able to be synthesized or secreted. This results in the classic presentation of hypoadrenocorticism, with the animal being severely volume depleted with an array of electrolye abnormalities including hyperkalemia, hyponatremia, hypochloridemia, and hypercalcemia. The hallmark of primary hypoadrenocorticism is the low sodium:potassium ratio (less than 25-27). The test used to diagnose primary hypoadrenocorticism is an ACTH-stimulation test in which cortisol is measured pre- and post-synthetic ACTH (Cortrosyn[R]) administration.

Secondary hypoadrenocorticism or atypical hypoadrenocorticism disease or glucocorticoid-deficient hypoadrenocorticism results from a lack of ACTH. In humans mutations resulting in CRH and ACTH deficiencies have been reported; however, in dogs, secondary hypoadrenocorticism is thought to result from a lack of ACTH due to pituitary failure. The failure of ACTH secretion has its primary effect on the zonae fasiculata and reticularis and thus spares the zona glomerulosa and aldosterone secretion. These dogs will maintain mineralocorticoid activity, but will be glucocorticoid deficient. In most dogs, the HPA axis is only evaluated by cortisol (glucocorticoid) measurement. The mineralocorticoid activity is estimated by evaluating Na and K concentrations. It has been shown that some dogs with normal serum Na and K concentrations

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are actually deficient in aldosterone. Conversely some dogs may be hyponatremic with only glucocorticoid deficiency. Therefore, the lines have been blurred between primary and secondary hypoadrenocorticism in the absence of routine mineralocorticoid measurement. In order to fully determine aldosterone activity, it should be measured directly both before and after stimulation by ACTH remembering that pharmacologic doses of ACTH (such as Cortrosyn[R] doses used in a stimulation test) will stimulate aldosterone secretion.

So, what distinguishes full blown hypoadrenocorticism and glucocorticoid-deficient hypoadrenocorticism? In the latter case, the dog will only be cortisol-deficient and will maintain mineralocorticoid activity. Clinical signs relate to glucocorticoid deficiency and will often be centered around gastrointestinal problems to include: vomiting, diarrhea, melena, hematemesis, hematochezia, weight loss, and generalized weakness. As with a full-blown Addisonian, the clinical signs may wax and wane. The dogs are generally not polyuric and polydipsic since aldosterone concentrations are normal. Clinical pathology abnormalities may include a mild normocytic, nonregenerative anemia, reverse stress leukogram (lack of neutrophilia, eosinophilia, lymphocytosis), hypoglycemia. If significant blood loss has occurred, the anemia may be severe. Sodium and potassium concentrations are generally normal.

Glucocorticoid-deficient hypoadrenocorticism should be diagnosed with an ACTH-stimulation test. Cortisol and aldosterone should be measured since Na and K are only rough estimates of mineralocorticoid activity. If aldosterone levels are low or approaching the low end of the normal range, the dog should be monitored closely for emerging mineralocorticoid deficiency. Diagnosis of glucocorticoid deficiency is based on resting cortisol levels lower than the normal range without a significant increase after ACTH-administration. Using resting cortisol levels of greater than 2 mcg/dL to rule out glucocorticoid-deficient hypoadrenocorticism has not been examined experimentally.

Treatment: Since mineralocorticoid activity is not affected, only glucocorticoids need to be supplemented. Dexamethasone-SP may be used in an acute situation at a dose of 0.05-0.1 mg/kg. Dexamethasone has the advantage of being parenteral and also will not be read in the cortisol assay used for the ACTH-stimulation test. After stabilization, the dog may be transitioned to prednisone at 0.2-0.4 mg/kg once per day. Often dogs will need a dose increase during stress. The dose should be titrated to the lowest that will maintain good quality of life for the dog.

Follow-up: Glucocorticoid doses should be adjusted based on clinical signs. Sodium/potassium levels should be monitored so that conversion to mineralocorticoid deficiency will not be missed.

SVA DERMATOLOGY (SIMULTANEOUS TRANSLATION INTO MANDARIN CHINESE)

UPDATE ON ALLERGIC DISEASE: ATOPY VS CONTACT ALLERGY

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CANINE ATOPIC DERMATITIS (CAD)

A definite diagnosis of atopic dermatitis is based on:

- A typical history
- Typical clinical signs
- Rule out of other pruritic dermatoses
- Allergy investigations

HIreed:

There are strong breed and/or familial predispositions for CAD but predisposed breeds vary with geographical regions. Some breeds, such as West Highland White terriers, boxers and bulldogs seem predisposed in all regions; others such as German Shepherds, golden retrievers and Labradors seem to be predisposed for CAD only in some geographic regions. Breed-associated phenotypic variability of CAD has been reported.

Age of onset:

The typical age of onset is reported to be between 6 months and 3 years of age, but occasional dogs develop clinical signs after this age range. Food allergies can commence at any age.

CLINICAL:

The most prominent clinical sign in canine atopic dermatitis is pruritus. In the majority of cases, the pruritus has a characteristic distribution. The pruritus may be seasonal (with CAD) or more typically continuous. Approximately 80% of dogs with seasonal signs are symptomatic in spring or summer while other exhibit signs in autumn or winter.

Distribution

Generalised or limited to the face, ears, feet, and pinna, and axillae, inguinal region

Unilateral or bilateral otitis externa

Lesions

Pruritus must be present. Early in the disease some CAD dogs exhibit pruritus with no evidence of any skin lesions. Other cases may have erythema of the affected areas but no other lesions. In some dogs the initial presentation of CAD is an episode of otitis externa.

In the first stages of the disease, pruritus generally responds well to corticosteroid therapy (0.3mg to 0.5mg/kg prednisolone daily).

CAD is a progressive disease. Most of the clinical signs develop due to self trauma and/or secondary infections. Chronic changes of CAD include alopecia, excoriations, hyperpigmentation, lichenification and/or signs of secondary bacterial (papules, pustules, crusts, erosions, epidermal collarettes) and/or yeast (hyperpigmentation, lichenification) infections. Recurrent ear infections are frequently observed. It is important to recognise these because they may require specific management in addition to control of the allergic dermatitis.

Other historical features help to support the diagnosis of CAD such as sneezing, reverse sneezing and conjunctivitis. A lack of gastrointestinal signs in a dog with pruritus compatible with ACD also helps by decreasing but not eliminating food as a cause.

DIAGNOSIS

The diagnosis of canine atopic dermatitis is based on typical history and clinical presentation and most importantly by exclusion of other pruritic skin diseases.

Pruritus the defining feature of canine atopic dermatitis is a common presenting problem for veterinarians. Pruritus is a sensation in the skin that occurs with a number of skin diseases however the list of differential diagnoses for common causes of pruritus that appear clinically similar to CAD is relatively small. Causes of pruritus are often classified into primary diseases, able to cause pruritus directly, or secondary causes which are diseases (usually infections) that occur as a consequence of the damage to the skin caused by a primary disease. The possibility of several co-existing primary and/or secondary pruritic diseases should be considered. In particular, for the diagnosis of CAD, it is important to determine if the pruritus and erythema are the result of environmental CAD, and/or the presence of hypersensitivity to fleas, and or adverse reaction to food, and/or infections. Often a variety of therapeutic or diagnostic trials are indicated to really determine

the cause of the clinical signs of dogs with pruritus. Allergy testing is not considered to be a major tool in the diagnosis.

KEY POINTS FOR THE DIAGNOSIS OF ATOPIC DERMATITIS

1. ASSESS THE DISTRIBUTION OF THE DISEASE
Paws, especially ventral interdigital
Concave base of pinna
External orifice: ears
Flexor surface metacarpal or metatarsal
Flexure of the elbow
Axilla
Abdomen/inguinal
Periocular
Perioral

2. LOOK FOR SKIN LESIONS
Erythema
Check carefully for **papules** (secondary infection, flea bites, scabies or contact dermatitis)
Check carefully for **bacterial infection** (papules, pustules, crusts, erosions, epidermal collarettes)
Check carefully for **yeast infection** (erythema, scale, (hyperpigmentation, lichenification, greasy exudate, odour)

3. LOOK FOR FLEAS
Acute: macules, papules, crusted papules, hot spots
Chronic: alopecia, lichenification, hyperpigmentation
Flea therapeutic trial: oral daily nitempyram
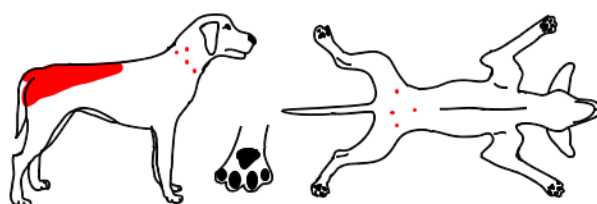
4. EVALUATE FOR FOOD ALLERGY
Age of onset< 12 months
Non seasonal (cf seasonal for CAD/FBH)
Gastrointestinal signs
Elimination diet trial
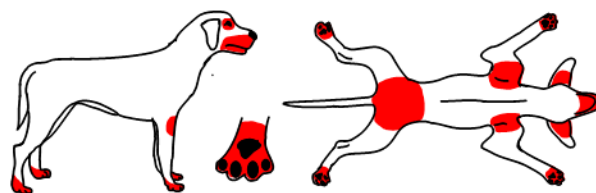
If the dog is still pruritic after the above conditions have been ruled out or controlled and it has compatible clinical signs, then it is usually possible to make a tentative diagnosis of atopic dermatitis.

At this point the clinician has two options: either **symptomatic treatment or allergy testing**. Which route is taken depends in a number of factors including the severity of the pruritus, age of the dog, financial considerations and what the owner wishes for their pet. In some cases, symptomatic treatment may be recommended at the beginning and then allergy testing performed at some point in the future.

Allergy testing

Two methods of allergy testing are routinely available for the further investigation of canine atopic dermatitis: **intradermal allergy testing (IDAT) and serum in vitro testing (SIVT)**. The diagnostic accuracy of either test is low because there are multiple allergens that can give positive results in clinically normal dogs and dogs with other skin diseases. The test result is only meaningful if the dog has clinical signs consistent with atopic dermatitis and all other pruritic diseases have been ruled out. They are useful tests if owners wish to consider immunotherapy as a management strategy for the dog with chronic allergic dermatitis.

In summary, allergy testing is recommended after a clinical diagnosis of CAD is confirmed in patients where ASIT is indicated and reduction in pharmacotherapy is desirable in a dog with a compliant temperament and a motivated client.

Intradermal allergy testing (IDAT)

A variety of companies manufacture allergens for IDAT however most dermatologists currently utilise allergens from Greer Laboratories (Lenoir, NC, USA) and dilute according to the manufacturer's recommendations with a final allergen concentration of between 1000 and 1800 PNU/ml (protein nitrogen units/ml) for dogs. Most veterinary dermatologists test for reactivity against the following antigens: house dust mite and storage mite antigens (**Dermatophagoides farinae, Dermatophagoides pteronyssinus, Acarus siro, Tyrophagus putrescantiae**); insect body parts/faecal elements (cockroach, moth, ant, houseflies); pollens (from trees, weeds and grasses); moulds (from the household or from crops) and **Malassezia**.

The inclusion of regional allergens (pollens) in the testing kit is based on knowledge of the plants in a particular geographical location. The intradermal allergy test is interpreted by correlating the positive reactions with the patient's history. Clinically relevant reactions can then be used to choose allergens for specific immunotherapy.

Intradermal testing is best performed with the dog under sedation in lateral recumbency. Medetomidine (Domitor®) at a dose of 5 to 10mcg/kg IV is the preferred sedative. Acepromazine is not acceptable because it reduces skin test reactivity. A patch of fur is clipped from the lateral thorax (15cm x 10cm) and the injection sites are marked with a black marker pen.

A standard intradermal injection of 0.05mls per allergen is injected intradermally along with the positive (histamine) and negative (saline) control. The reactions are read 10 to 20 minutes later. These appear as wheals. The reactions are subjectively graded based on wheal diameter, height, turgidity and erythema and graded from zero to four.

A score of zero represents a negative reaction (equivalent to the negative saline control) and a score of four represents a positive reaction that is equivalent to the positive histamine control. In some cases, late phase reactions may occur at some sites 24 to 48 hours later. These appear as erythematous, indurated areas that may contain a papular eruption. The full significance of these reactions is currently unknown.

A positive reaction requires functional cutaneous mast cells and the presence of allergen specific, mast cell bound, reaginic (presumed IgE) antibodies. Allergenic epitopes cause dimeric or trimeric cross-linking of IgE with resultant release pharmacologically reactive substances, such as histamine, serotonin, and various leukotrienes. An immediate reaction is mediated by histamine and neurogenic factors while histamine, prostaglandins and other vasoactive amines are involved in late phase reactions.

Intradermal allergy testing
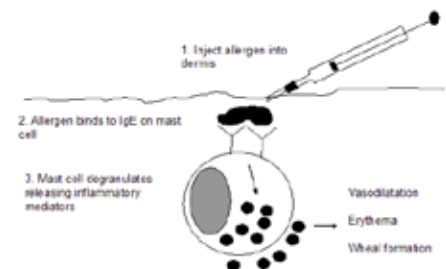
Reproduced with permission from Dr Peter Hill and Zoetis: Australian Veterinary Dermatology Advisory Panel Guidelines for the Diagnosis and Management of Pruritic Dogs.

Allergen solutions are expensive and it would not be cost effective to offer this service unless one or two tests per week were being performed. Practitioners interested in performing this procedure should study for a further qualification in the discipline or undertake residency training

Serum in vitro IgE allergy testing (SIVT)

In-vitro testing simply requires a blood sample to be collected and sent off to an appropriate laboratory. The serum is assayed for allergen-specific IgE and the results are reported as relative units (the higher the score, the higher the level of IgE). The in-vitro test is interpreted by correlating the positive reactions with the patient's history. Clinically relevant reactions can then be used to choose allergens for specific immunotherapy.

The diluted serum sample is added to a plate containing individual wells coated with specific antigens. The types of antigens tested are similar to those used for intradermal allergy testing but there are usually less than in a skin test. If there is any IgE in the serum that is specific for a particular antigen, it binds to it. The bound IgE is then detected by adding an enzyme-linked reagent that can bind to IgE. This is either a monoclonal antibody or a receptor for IgE molecules. A substrate is added that changes colour when it contacts the enzyme attached to the IgE reagent. The degree of colour change is proportional to the amount of IgE that is bound. The colour change is measured by an automated reader and the results are reported as a numerical score. The significance of various scores is indicated by the laboratory.

In-vitro measurement of allergen-specific IgE
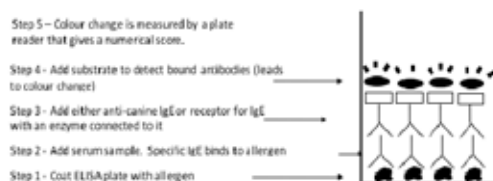
Step 5 – Colour change is measured by a plate reader that gives a numerical score.

Step 4 - Add substrate to detect bound antibodies (leads to colour change)

Step 3 - Add either anti-canine IgE or receptor for IgE with an enzyme connected to it

Step 2 - Add serum sample. Specific IgE binds to allergen

Step 1 - Coat ELISA plate with allergen

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The ELISA (enzyme linked immunosorbent assay) most commonly used in Australia is the Heska ALLERCEPT™ assay. The ALLERCEPT™ ELISA assay uses the alpha chain of the high-affinity FcεRI receptor as its detection reagent to ensure specificity for IgE.

Preparation of animals for allergy testing

Before either of the above tests are performed, it is important that the patient is adequately prepared. Clinicians should ensure that:

- Other pruritic diseases have been ruled out

- Anti-pruritic drugs have been withdrawn for a suitable period of time (Table 1)

Treatment	Intradermal testing	In-vitro testing
Methylprednisolone acetate injection	6-10	3-5
Daily prednisolone	4-6	0
Alternate day prednisolone	4-6	0
Topical steroids (including ear and eye drops)	1	0
Antihistamines	1	0
Cyclosporin	0	0
Essential fatty acids	0	0
Oclacitinib	0	0

NOTE: Treatment with methylprednisolone, daily and every other day prednisolone, or cyclosporin for periods longer than 3 months may require longer withdrawal times

Which test is better for selecting allergens for ASIT?

Veterinary dermatologists are often asked which is the better test. When answering this question, it is important to remember that the tests are not measuring the same thing. **In vitro** tests merely measure the amount of allergen-specific IgE that is present in the blood. Intradermal allergy testing detects the presence of allergen-specific IgE that is bound to mast cells in the skin. However, intradermal allergy tests also measure mast cell releasability (this can be altered in atopic dermatitis) and the response of the skin to inflammatory mediators. Intradermal allergy tests, therefore, provide a complete functional assessment of some of the pathways that are required to initiate an allergic reaction in the skin. In contrast, **in vitro** tests only measure one particular point in the pathway. For this reason, most veterinary dermatologists regard intradermal allergy testing as the superior test

If it is not possible for a dog to undergo intradermal allergy testing (e.g. if the practice doesn't perform it, there is no local referral centre, the owner doesn't want referral), **in vitro** tests can be used as alternative to identify allergens for use in immunotherapy

Despite the above theoretical and practical considerations, it is common for a positive reaction to occur in one test and not the other. Performance of both tests at the same time is more informative, although it may be cost prohibitive. Of note, an increase in the efficacy of the chosen immunotherapy based on the combined test results has not been confirmed in properly controlled studies.

CONTACT ALLERGY

Allergic contact dermatitis is commonly suspected in dogs but establishment of a precise diagnosis can be challenging. The most common plant contact allergens are grasses (**Cyodon and Kikuyu** species); plants (**Tradescantia** spp.) and other members of the **Commelinceae** (succulent ground covers) family. Other causes of contact allergy include topical antibiotics (neomycin), vehicles used for topical preparations (propylene glycol), shampoos (chlorhexidine), flea products, carpet deodorizers and metals

Clinicians should be aware that there is considerable overlap between the clinical appearance of atopic dermatitis, food allergy, staphylococcal pyoderma, **Malassezia** dermatitis and contact dermatitis**.** It can be difficult to diagnose and may be frequently misdiagnosed as atopic dermatitis. In many cases, contact allergy actually co-exists with atopic dermatitis and this makes the diagnosis complex and difficult.

Age of onset:

The typical age of onset is reported to be adult dogs of 6 months and older, although it has been diagnosed in dogs as young as 2 months of age.

CLINICAL:

Distribution

Contact allergies typically affect the sparsely haired regions of the face (muzzle and periocular region) concave pinnae, inguinal area, feet, perineal and genital area (plants or carpets) and scrotum (floor detergents, cement, bleach).

Lesions

Lesions are particularly evident on glabrous areas. Intense pruritus is common and in severe cases can lead to a lack of response to anti-inflammatory dosages of corticosteroids. A primary erythemic maculopapular eruption is visible in affected areas. Self trauma and chronic inflammation may lead to hyperpigmentation and lichenification.

DIAGNOSIS

The diagnosis of contact allergy is usually based on the combination of clinical signs and response to confinement followed up by scratch or patch testing. Interpretation may be complicated by the fact that animals may have more than one condition at the same time, some of which wax and wane. An integrated and sequential investigation is usually required if successful results are to be achieved.

Four diagnostic tests can be used to investigate suspected contact dermatitis

1. **Removal of suspected causes of contact irritation**: collar, plastic food bowl, topical medication, shampoo or floor disinfectant.

2. **Environmental restriction:** bath and isolate dog in a new environment (kennel or hospitalisation) for allergen avoidance. Complete resolution of the lesions within 7 to 10 days following restriction suggests that contact dermatitis may be involved; re-challenge to confirm the diagnosis and lesions should recur within 1 to 4 days.

3. **Scratch or patch testing:** permits identification of the allergen (kikuyu grass, buffalo grass, **Tradescantia** sp etc) and is usually performed after confinement.

Scratch or patch test

A **scratch/patch test** can then be performed to identify specifically what the trigger is. A patch pf skin is clipped over the lateral thorax. The test sites are then outlined with a marker pen. The suspected surfaces are then rubbed onto the skin. A scratch to the skin surface can be made using a 23G needle to ensure penetration of the stratum corneum.

Topical medications and shampoos can be applied in their normal formulations, powders can be mixed with petroleum jelly and floor cleaners and disinfectants can be applied at their normal working oilutions. Plant extracts can be made using a mortar and pestle. The sites are then monitored for signs of erythema, oedema and pruritus. The test should be read at 15 to 20 minutes looking for immediate reaction while the substance is still on the skin. The test solution is not washed off. Owners should be asked only to gently wash or wipe the dried solution off with tap water after 24 hours and then to observe the test site for redness or rash over the next 24 to 48 hours.

TREATMENT

Immunotherapy is not effective. The best approach is avoidance. When avoidance is not feasible, glucocorticoids can be used either topically or systemically to minimise the severity of clinical signs. Pentoxifylline has been reported to be effective for contact allergies but works best as a preventative rather than treatment and should be started 48 hours prior to exposure.

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WSV18-0307

FELINE LOWER URINARY TRACT DISEASE - 2018 UPDATE

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Feline lower urinary tract disease - 2018 update

Introduction:

Diseases of the lower urinary tract of cats are summarized under the term "Feline Lower Urinary Tract Disease" FLUTD. FLUTD describes the common clinical presentation of different diseases with a wide variety of causes. The signs of FLUTD are pollakiuria, stranguria, periuria and hematuria (1). Obstruction of the urethra occurs frequently in this disease complex.Causes:

If the cause of FLUTD cannot be identified the diseased is called idiopathic cystitis (FIC). Between 55% and 63% of the cats with FLUTD are considered to suffer from FIC (2, 3, 4, 5). Common causes of FLUTD are urinary calculi, urethral plugs and urinary tract infections. Further less common causes of FLUTD are neoplasia (e.g. transitional cell carcinoma), acquired or congenital anatomic defects, and central nervous system disease leading to micturition disturbances.Diagnosis:

Because all forms of FLUTD have a very similar clinical presentation, laboratory tests and diagnostic imaging are required in each case to establish a diagnosis. Recurrent episodes in the same cat can have different causes and have to be worked up completely each time (6). Urinalysis is very important and urine should always be collected before any therapy is instituted. Ideally urine should be collected by cystocentesis; however there is some debate about the danger of cystocentesis in obstructed cats. Urinalysis should include measurement of the specific gravity, a dip-stick analysis, analysis of the urine sediment and a urine culture. Serum biochemical analysis can provide information about underlying diseases. Furthermore it is important to identify and quantify metabolic disturbances including hyperkalemia or postrenal azotemia in cats with urinary tract obstruction.

On radiographs radio dense stones can be seen, furthermore size and form of the bladder can be evaluated. It is important to make sure that the distal end of the urethra is on the radiograph. Ultrasound evaluation of the urinary tract provides information about the bladder wall and the content of the bladder. Diseases of the urethra can be seen by contrast urethrography. Urethroscopy and cystoscopy are not routinely performed in cats with FLUTD.FIC:

It is still not known what's causing FIC. Associations with viral diseases or with bacterial DNA in culture negative urine could not be found (7,8). FIC was suggested as model fort interstitial cystitis in people (9). Typical glomerulations (small petechial bleedings) in the submucosa of the bladder wall are part of the human disease and are required for the diagnosis. However cystoscopy is not routinely used for the diagnosis of FIC and the term interstitial cystitis is only applicable for cats in the few cases where cystoscopy was performed.

Risk factors associated with FIC are male gender, being neutered, middle-aged (2-7 y), and overweight. Stress caused by husbandry or environmental factors like indoor housing are also associated with FIC (10).

Cats suffering from FIC show pain, hematuria, pollakiuria, stranguria, periuria or are not able to urinate at all. This picture is not different from other causes of FLUTD. Treatment of urinary tract obstruction/Prognosis:

Cats with urinary tract obstruction are emergency patients. The main goal of the therapy is to re-establish urine flow. Life threatening metabolic derangements like hyperkalemia or severe acidosis have to be corrected immediately. Possibilities for the therapy of hyperkalemia are: -infusion with NaCl; -infusion with glucose; -regular insulin followed by a glucose bolus followed by infusion with glucose; -calcium gluconate, or -sodium bicarbonate.

If urethral patency can't be re-established, urine can be evacuated by cystocentesis. Possible side effects of decompressive cystocentesis are extravasation of urine into the peritoneal cavity and injury to a pre damaged bladder wall. Once the urethra is patent an indwelling catheter is left in place and connected to a closed urine collecting system. Re-obstruction ratios are reported to be between 11% and 36% (11). Duration of catheterization seems to be important, leaving the catheter longer (mean 21.62 h vs 32.1 h) was associated with a lower recurrence rate and only establishing patency without leaving the catheter in place was associated with a higher re-obstruction rate (11,12). The size of the catheters left in place was associated with recurrence rate in one study, with bigger catheters (5 F vs 3.5 F) having a higher re-obstruction rate (13).

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TIPS FROM THE EXPERTS FOR THE MANAGEMENT OF...

PRURITUS - GENERAL APPROACH

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APPROACH TO THE PRURITIC DOG

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Itch is defined as an uncomfortable sensation on the skin that causes the desire to scratch. The desire to scratch however, may be induced from a plethora of causes, from some seemingly innocuous environmental triggers, to even include infectious ones. Thus, a pragmatic and systematic approach to a pruritic canine patient with a final end result/diagnosis of canine atopic dermatitis (CAD) is very important in order that a correct diagnosis may be made consistently and the quality of life quickly restored to the patient. CAD is defined as a genetically predisposed, inflammatory and pruritic, allergic skin disease with characteristic clinical features, most commonly associated with IgE antibodies to environmental allergens.[1] Since the first diagnostic criteria for CAD was published in 1986 by Dr. Willemse, who directly transposed from the human criteria as set forth by Hanifin and Rajka, it set in motion a collective effort by veterinary dermatologist, immunologist and researchers to further define the disease, refine its diagnosis and treatment whilst broadening our understanding of this complex, multifactorial disease.[2, 3] Approximately 10 years later, Dr. Prélaud and colleagues proposed a new set of five criterias, where fulfilment of three criterias yielded a sensitivity of 79% and a specificity of 81% for the diagnosis of CAD.[4] Most recently in 2010, Favrot and colleagues proposed a new criteria where a fulfilment of five criteria from the first set had a sensitivity of 85% and a specificity of 79%.[5] An improvement from the previous. This increase in sensitivity could be explained by the added exclusion criteria of an affected ear margins (suspicious of sarcoptic mange) and also an affected dorso-lumbar area (suspicious of flea allergic dermatitis). Thus setting the tone for our approach in a pruritic canine patient where the diagnostic end point is the diagnosis of CAD, which is a clinical diagnosis based on the exclusion of other pruritic disease.

The approach to a pruritic canine patient begins with the history and clinical signs of the patient. From the age of onset, seasonality, acute/chronic itch, presence of alesional pruritus to response to previous treatment/s are important indispensable clues to aid identification of the aetiological pruritic agent. Puppies are more prone to develop contagious pruritic diseases such as sarcoptic mange, dermatophytosis or demodicosis. Patients with onset of pruritus that begins less than 12 months of age is highly suspicious of cutaneous adverse food reaction (CAFR), whereas CAD patients are typically presented between 12-36 months of age.[5,6] Seasonal pruritus is often associated with flea allergic dermatitis or canine atopic dermatitis. A typical canine atopic patient is often presented with chronic alesional pruritus that respond previously to steroids.[5] From clinical signs, sarcoptic mange infestation often manifests itself on the ear tip margins whereas it is the dorsal lumbar area for flea allergic dermatitis. The typical anatomical site that is involved in canine atopic dermatitis are the forepaws and also the concave ear pinnae.[5] Needless to say, patients are often pruritic, manifested clinically as either scratching, licking or having excoriation marks at these respective sites. If there is evidence of a parasitic infestation that could cause itching, they should be addressed accordingly and patient re-evaluated for persistence of pruritus on a later date. The new class of insecticides, isoxazolines such as fluralaner, sarolaner, afoxolaner and lotilaner offers a safe, efficacious and even sustained efficacy for up to 90 days in one product.[7-10] Understanding the pharmacokinetics and pharmacodynamics of this or other classes of antiparasiticals would aid in its assimilation to the veterinarians' approach to the pruritic patient potentially making it more flexible and robust.

For every pruritic patient, it is important for the attending veterinarian to evaluate the presence of superficial staphylococcal pyoderma and likewise important to determine to what portion of the overall itch is related to staphylococcal infection. This is the same of malassezia dermatitis. This can be done rather easily with routine cytology, collected from representative regions with typical associated clinical signs. The attending veterinarian must remember that recurrent pyoderma or recurrent malassezia dermatitis is often associated with an underlying allergic disease.[11] Due to this high rate of recurrent pyoderma, it is often that patients are frequently exposed to antibiotics which adds selective pressure that culminates in antimicrobial resistance.[12,13] Thus, a robust approach to a pruritic patient and antimicrobial stewardship is indispensable that goes hand-in-hand in modern day practice. Antibiotics are certainly not candies we should freely dispense. After the likelihood of an infestation and/or infection has been ruled out, the possibility of a CAFR must be investigated. Despite its clinical signs are virtually indistinguishable

from canine atopic dermatitis, CAFR does have its own particularities. CAFR is often thought to occur in younger patients, less than one year of age with non-seasonal pruritus, mostly widespread and generalized.[5, 6] This pruritus is usually severe, constant with high variability in its amelioration of pruritus with oral or parenteral glucocorticoids, with results ranging from poor to good.[5,14] The only effective method to distinguish CAFR and CAD is to conduct a food trial either with a home-cooked elimination diet with novel sources of protein and carbohydrate or a commercial hydrolysed diet for 9-10 weeks. It is only with the lack of response from a food trial that we can finally arrive at the diagnosis of CAD.

If a systematic approach to a pruritic patient has been conducted and CAD confirmed, it not only allows the attending veterinarian to appreciate the atopic patients' itch clinical threshold and but also allows the identification flare factors associated with it. The itch clinical threshold is defined as the point where a pet begins to scratch due to a summation of events, where each contributes independently to the itch cascade. As pruritic thresholds are unique for each animal, a systematic approach allows attending veterinarians to build a clinical pruritic impression for the patients under his/her care. Flare factors are biologic or environmental factors which induces the exacerbation of atopic dermatitis. Current recognized flare factors in CAD includes staphylococcal or yeast infections, flea infestation, dietary indiscretion and also environmental triggers.[15]

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ANESTHESIOLOGY

ANESTHESIA FOR POPULATIUON CONTROL: DOING IT WELL AND CHEAP

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ANESTHESIA FOR POPULATIONAL CONTROL: DOING IT WELL AND CHEAP

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Non-client-owned, shelter animals often require sedation, anesthesia and analgesia for orchiectomy (neuter) or ovariohysterectomy (spay) prior to adoption. The Association of Shelter Veterinarians published a guideline regarding the anesthesia of shelter animals with emphasis on patient care and welfare including anesthesia, analgesia and postoperative care. There are also several websites elucidating ways to preserve and protect the animal rights at shelters. They are: www.sheltervet.org; www.aspcapro.org; www.maddiesfund.com; www.sheltermidicine.com and www.sheltermedicine.vetmed.ufl.edu.

Many studies show that the anesthesia-related mortality rate is reduced with in depth understanding of patient's history and pre-op examinations. However, when animals present for surgeries in shelters, they usually lack that important clinical history, performing full blood work for all animals prior to surgery is cost prohibitive and most of feral animals do not allow the veterinarian to approach for a complete physical exam prior to drug injection. Therefore, the anesthesia-related mortality rate in shelters is higher. However, there are several simple techniques that can be used for the anesthesia management of feral and shelter animals in order to improve their survival rate.

1-The most important techniques you can add to your practice in order to increase survival rates in face of lack of pre-op clinical information are: 1- vital signs monitoring during anesthesia; 2- personnel training to recognize any possible cardiovascular and respiratory complication; 3- readiness for aggressive treatments of possible complications.

2-Before the beginning of your anesthesia management, it is important to provide a stress free environment. Stress leads to catecholamine release and arrhythmias

that can be exacerbated by anesthesia. Keep cats away from dogs and dogs away from cats. That alone can help to reduce the stress of those animals significantly. Consider this: Outsmart your patient but do not out power them. You must stay calm, use your brain and be sneaky. Do not use brute force to restrain the animal. That will only cause frustrations for you and your team and stress for your patients. Remember: With cats, the less restraint the better. Also, do not use a catch poll for cats. That causes too much stress for them. Search for a "Freeman cat net" this is a great device to help you to reduce the animal stress. Squeeze cages can also help you get your injections with a minimum stress for both ends: you and your patient.

3-It is acceptable to anesthetize pregnant or lactating patients, animals in estrus, animals that have pyometra, mild infectious or noninfectious diseases (i.e.: upper respiratory tract disease, parasite infestation, or subclinical heartworm infection) however, additional monitoring should be available for those patients. For these cases you should consider running blood work and obtaining a complete physical exam before the beginning of anesthesia, when possible.

4-Fasting helps to reduce the chances of regurgitation and aspiration pneumonia. It is recommended 2-4 hours of fasting for pediatric animals and 6-8 hours for adult animals. Exceptions may be made for feral cats in traps when it is not safe to remove food from traps due to human safety. Removing water is neither necessary nor recommended.

5-Many cats have hyperthermia during anesthesia recovery. The mechanism triggering the post op hyperthermia is not well understood. Stress, opioids and ketamine could be trigger factors. Also, there is a believe that under anesthesia, the more hypothermic the patients are, the more hyperthermic they will be during recovery. Therefore, consider checking the body temperature as soon as possible and throughout the recovery phase as well. Provide heat when necessary. To treat the hyperthermia: non-steroidal anti-inflammatory drugs work well; consider reversing any opioid medication. If you do reverse the opioids with naloxone then the patient may suffer from pain. Butorphanol is a partial antagonist of the mu opioid receptors and can be used for the reversal of the hydromorphone and morphine as well. When butorphanol is used as a reversal agent, the patient will have some analgesia; Use water instead of alcohol since alcohol only causes peripheral vasoconstriction that prevents heat loss. Also a simple fan helps to get the temperature down.

6-When you are planning your anesthetic protocol for your shelter program consider safe and efficient protocols. Balanced anesthesia should always be the goal with analgesia as the first priority. Choose safe drugs with wide therapeutic indexes. Ketamine and

tiletamine are great examples of safe anesthetic drugs that provide potent analgesia. When ketamine or tiletamine is used in combination with a benzodiazepines (diazepam, midazolam or zolazepam) and an opioid (butorphanol, morphine, hydromorphone, buprenorphine) or an alpha2-agonist the animal gets good muscle relaxation, immobilization, effective analgesia and loss of consciousness without patient compromise. Another advantage of ketamine over other induction agents like propofol and alfaxalone is in regard to the necessity of endotracheal intubation. Ketamine leads to a maintenance of the laryngeal reflexes which help the prevention of aspiration pneumonia.

7-After sedation make sure someone trained is observing the animals to have fast treatment when necessary. Oxygen supplementation could significantly reduce shelter mortality

8-Finances are important. When designing anesthetic protocols check local prices and pick the least expensive drugs however, do not forget that anesthesia is not analgesia and to be fair, the shelter animals need both. Think about cheap opioids and non-steroidal anti-inflammatories. Also to reduce cost, check with your local pharmaceutical representative if they have programs to help shelters and other organizations to either provide the drugs for free or at least with a subsidized cost.

9-Eye lube is very important. Animals cannot generate tear while under general anesthesia and corneal ulcers may form if eyes are not well lubricated after anesthesia induction.

10-Besides the surgical procedure, consider the use of flea medicine when necessary. We recommend the identification of the surgical site with a tattoo to prevent possible future surgeries in the same animal. Some areas allow ear notches to identify the castrated animals however that technique tends to reduce the likelihood of adoptions afterwards.

11-Communication between staff is essential. You need a well-trained team where every one involved knows exactly what to do. Someone should be responsible for double-checking drug labels, concentrations and volumes. Usually the total drug volume is based on estimated body weight and to speed up the process there are ways to pre-calculate drug volumes per kilogram.

12-Use local anesthesia. An intra-testicular block with lidocaine is safe, cheap, fast and efficient analgesic protocol. There is no described local analgesia technique for OHE. Incisional local anesthesia have been described but it usually causes a prolongation of surgical site healing and is contraindicated, specially for cats and dogs that will be release after the surgery and no veterinary care will be available.

13-Mask induction can be performed however there is a major concern with environmental air pollution in the OR. Males don't have to be oro-tracheal intubated if the surgeon is fast however females should always be intubated and ventilated. Remember, isoflurane, and other inhalants by it self are do not provide analgesia and should not be used by it self.

References are available upon request

SVA INTEGRATIVE MEDICINE

OSTEOARTHRITIS: THINKING BEYOND NSAIDS - AN INTEGRATIVE APPROACH

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Introduction

Osteoarthritis (OA) is the most common joint disorder in the animals and one of the most common sources of pain and disability in the elderly animals. Chronic wear and tear on the joints is the underlying mechanism for OA. It is inevitable that there will be some degenerative change in animals' joints over their lifetime. The most common sites are the elbows, hips, spine, and any joint that has sustained traumatic injury. Joint pain is the principal symptom, but swelling, deformity, stiffness, and loss of function also occur. Chronic pain is undoubtedly the biggest threat to our pet's quality of life as they age. Fortunately, it is also a great example of a health issue that will benefit markedly from integrative therapy.

The initial step when addressing the arthritic patient is a thorough assessment of range of motion (ROM), weight-bearing status, girth measurements, palpation, overall function and owner perception of problems with home activities. A pain scale may be used to determine the level of pain. Weight-bearing status may be determined through dynamic or static force-plates, or scales. The overall function and goals of the owner should be determined in the evaluation, and progress using the various assessment tools should be monitored. After the problem list and the goals have been determined, integrative therapies can be implemented as part of a multimodal treatment program for OA.

Multimodal Approach to Osteoarthritis

When managing pain in OA patients, a multimodal approach should be used as early as possible. This can include the use of anti-inflammatories, opioids, tricyclic antidepressants, anticonvulsants, NMDA receptor antagonists, nutraceuticals, herbal therapies, acupuncture, laser, electromagnetic field, shockwave, therapeutic exercises, and adjunctive physical modalities. With this approach, both pharmacologic and non-pharmacologic therapies complement one another to increase the effectiveness of any given analgesic drug by intervening at multiple places along the nociceptive pathway. Utilizing a multimodal approach may also allow efficacious use at lower drug doses.

A. Weight Control

- The most important element in the treatment of OA is weight control.
- Overweight or obesity may contribute to the development and progression of OA because of excess forces placed on joints and articular cartilage, which may lead to inactivity and further development of obesity.
- Adipose tissue is recognized as being metabolically active and proinflammatory; therefore, obesity may potentiate the systemic and local effects of inflammation
- Maintaining optimal or slightly lean body condition may be associated with lower risk of developing OA, development of less severe OA if it occurs, and delay of onset of clinical signs of OA in dogs.
- Several commercially available diets formulated for management of dogs with OA. These diets contain higher levels of omega-3 fatty acids and may contain chondromodulators and antioxidants.

B. Acupuncture

- Acupuncture is defined as the insertion of thin, sterile needles into specific points, based on anatomical structures, which leads to a physiologic response. These physiologic responses are due to stimulation of the nervous system and lead to a release of endogenous substances such as β-endorphins, dynorphins, enkephalins, serotonin, epinephrine, gamma-aminobutyric acid (GABA), cortisol, and various hormones. Acupuncture has therapeutic clinical applications in rehabilitation and sports medicine such as pain relief, performance and endurance enhancement, and nerve regeneration.
- Acupuncture is a useful modality to integrate into a pain management due to its analgesic properties and nerve stimulation effects. It also has calming/sedating effects which are useful for patients that need to be cage-rested.

C. Low Level Laser Therapy (LLLT)

- Laser is a light source treatment that generates light of a single wavelength that elicits photochemical reactions in cells to promote tissue regeneration, reduce inflammation, relieve pain, increase fibroblasts and lymphocytes, and stimulates cartilage growth.
- The effect of laser therapy in OA include: inhibition of cyclooxygenase-2 enzyme and prostaglandins, reduces oxidative stress, endorphin release, and cartilage stimulation

D. Transcutaneous Electrical Stimulation (TENS)

- TENS is an electronic stimulus generator that transmits electrical impulses of various configurations to electrodes on the skin for the purpose of pain management. TENS can be effective for pain relief, especially in cases of chronic musculoskeletal and postoperative pain.

  Research suggests:

  - Acute pain: 50-200 Hz, lower pulse width: relief is short-lasting
  - Chronic pain: 2-4 Hz with higher pulse width produces longer lasting pain relief (several hours)
  - Mechanisms of TENS:

- Gate control theory: pain signals are transmitted by small cutaneous A-δ and C fibers. If a TENS current is applied, the large cutaneous (A-β) fibers are stimulated. The signals from the A-β fibers activate inhibitory neurons in the spinal cord dorsal horn and block the transmission of pain impulses from the periphery to the brain.
- Release of endogenous opiates. These endorphins are released from the pituitary in response to low frequency (<10 Hz), high pulse duration (>100 μsec) stimulation, and they produce analgesia when released.

E. Therapeutic Ultrasound (US)

- US is a method of stimulating the tissue beneath the skin's surface using very high frequency sound waves, between 800,000 Hz and 2,000,000 Hz, which cannot be heard by humans. The sound waves to stimulate fibroblast activity, improves blood flow, increases protein synthesis and aids in soft tissue and bone healing. It also provides heat to injured body parts that lie deep within your body that are not able to be heated with a standard hot pack alone.
- Therapeutic US is considered an effective treatment modality for rehabilitating musculoskeletal conditions such as restricted range of motion (ROM) resulting from joint contracture, pain and muscle spasm, and wound healing.

F. Extracorporeal Shockwave Therapy (ESWT)

- ESWT is a new technology using shockwaves to treat chronic, painful conditions of the musculoskeletal system. A shockwave is an intense, but very short energy wave traveling faster than the speed of sound. ESWT may be beneficial as an ancillary treatment for OA as part of the multimodal approach to therapy of this condition. Improved weight bearing and passive range of motion are similar to results expected with NSAID use. In patients that are unable to tolerate the administration of NSAIDs, extracorporeal shockwaves may serve as a useful, noninvasive alternative for treatment of osteoarthritic conditions. Anecdotally, treatment of conditions affecting the elbow, hip, or back may respond better than other joints.
- Mechanism:
  - Gate control theory
  - Induction of cell damage from shockwave application prevents appropriate membrane potentials required for transmission of signals.
  - Production of cytokines and growth factors, endothelial nitric oxide synthase (eNOS) and up-regulation of bone morphogenetic protein (BMP) expression resulting in a decline in inflammation and swelling, with short-term analgesia.

G. Chondroprotective Supplements

Chondroprotectants are products proposed to protect the cartilage (against degradation).

- Glucosamine/Chondroitin

    - Results take 4–8 weeks to develop. These improvements often last for several weeks after glucosamine supplements are discontinued.

    - Studies and clinical experience in people and pets show that glucosamine/chondroitin are equally effective for treating OA when compared to NSAIDs without the side effects.

    - Glucosamine is rapidly taken up by cartilage cells and helps stimulate the synthesis of synovial fluid and cartilage.

    - Chondroitin sulfate is the major glycosamino-glycan found in cartilage; it also helps inhibit enzymes that are destructive to the joint.

    - Glucosamine: ≥ 25 mg/kg, sid

    - Chondroitin: ≥ 15—20 mg/kg

- Polysulfated glycosaminoglycan (Adequan®)
    - Only FDA-approved chondroprotectant in dogs
    - Inhibit serine proteinases, which play a role in the interleukin-1 mediated degradation of cartilage proteoglycans and collagen.
    - Inhibit some catabolic enzymes that degrade collagen, proteoglycans, and hyaluronic acid
    - Inhibit prostaglandin E synthesis.
    - Reduce cartilage degradation by suppression of inflammatory mediators and stimulation of GAG synthesis.
    - 2 mg/kg, twice weekly for 4 weeks, then once a month.
- Omega 3 Fatty Acids
    - Include: Alpha-linolenic acid (ALA), Eicosapentae-noic acid (EPA), Docosahexaenoic acid (DHA)

    - When omega-3 FA replaces arachidonic acid (AA) in cell membranes, the inflammatory cascade is decreased.

    - Diet high in omega-3 FA help with rheumatoid arthritis as well as OA.

    - Implementation of a high-EPA diet together with an NSAID can reduce the amount of NSAID required for the same effect by 25%

    - 70-250 mg/kg, sid.

- Green-Lipped Mussel
    - Natural source of GAGS including chondroitin, as well as a number of other nutrients including complex proteins, amino acids, nucleic acids, naturally chelated minerals, fatty acids, and an inhibitor of prostaglandin synthesis, which makes it effective as an anti-inflammatory supplement, Glycoflex.

    - Studies have confirmed improvement in human OA

    - Study found that some owners perceive huge improvements in OA

    - 30 mg/kg, sid-bid

- S-adenosyl-methionine (SAM-e)
    - Human clinical trials show SAMe effective in treating OA
    - Positive results can persist for at least 2 years after treatment in people
    - Rabbit studies show some chondroprotective effects, by increasing proteoglycan synthesis
    - 18 mg/kg/day

H. Chinese Herbal Supplements

- Traditional Chinese Veterinary Medicine (TCVM) is an effective treatment to help complement current medications and improve pain management. Form the TCVM standpoint, pattern differentiation (Diagnosis) is important for the treatment strategy for OA pain. One diagnosis entity has different kind of patterns. Selections of herbal formulas are based on the pattern differentiation of the patient. As examples:
    - Pain worse in heat/summer: Di Gu Pi San
    - Pain worse in cold/winter: Loranthus Formula
    - More stiffness than pain: Coix Formula
    - Muscle soreness: Body Sore

Conclusions

The conventional medical approach to OA focuses upon the signs and symptoms of the disease. Integrative therapies with multimodal approach are becoming more utilized in veterinary patients and more popular with both clients and practitioners to address OA. When properly administered by adequately trained professionals, these modalities are incredibly effective and safe to patient. They also have less side effects when compared to medications. Often in our integrative practice, we find that adding in these modalities enables lower doses of conventional pain medications, more frequent patient monitoring and follow-up, and clients feel more proactive leading to greater client compliance and satisfaction.

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ORTHOPEDIC SURGERY (SIMULTANEOUS TRANSLATION INTO MANDARIN CHINESE)

PRESERVATION OF CANINE JOINT HEALTH: BEYOND SYMPTOMATIC CONTROL

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It is helpful to think of joint disease as a metabolic imbalance shifted toward catabolism. Joint disease not only affects joint cartilage and synovium, but also subchondral bone, periarticular muscles and tendons, pain perception, cardiovascular fitness, and, ultimately, the patient as a whole. The painful patient is less active and less interested in play and begins to lose muscle mass and cardiovascular fitness while gaining weight. The normal family activity routines are often upset. Not surprisingly, effective joint health management must be directed at the entire patient, rather than simply at the inflamed joint. Further, joint health must be preserved at the earliest notion that it may be at risk of developing OA. In my opinion, every post-surgical joint patient should be placed in a joint health preservation program.

Pet-owner education is the foundation of effective preservation of joint health because comprehensive joint care requires the pet-owning family's active involvement and understanding of treatment goals. It is essential that pet owners understand that joint health preservation, like OA management, is an ongoing, lifelong process that is updated and modified based upon response to treatment and development of new therapies, etc.

Attaining & maintaining a lean body conformation through proper nutrition and feeding practices is the cornerstone of effective joint health preservation. Unfortunately, over-feeding is the single hardest habit for pet-owners to change. Studies have shown the effectiveness of restricted food intake on weight reduction, delaying the onset of and decreasing the lameness associated with OA and increasing patient lifespan. The challenge is accomplishing the restricted food intake through controlled feeding, especially in multi-pet households or where feeding is large part of the human-pet bond.

A lifestyle of regular activity that is moderated away from intermittent extremes of exercise and activities to which the pet is not conditioned is essential. This is VERY difficult for many pet-owners because it, by necessity, impacts their daily schedule. Interestingly, a shift to this desirable lifestyle often benefits the pet-owner simultaneously...everyone wins!

Therapeutic exercise and **physical rehabilitation** under the direction of a certified canine rehabilitation practitioner often contributes to dramatic improvement in comfort, mobility and condition of osteoarthritic dogs.

Therapeutic exercise is a key step in converting the osteoarthritic, overweight and sedentary dog to a lean, physically fit dog that is enthusiastic about activity again.

Diets specifically formulated for the dog with OAhave a practical place in the comprehensive management of OA. Omega-3 fatty acids, a key component of these diets, appear to decrease joint inflammation, improve lameness and patient activity and reduce reliance upon NSAIDs.

Nutritional supplementation with so-called nutraceuticals containing chondroitin, glucosamine and manganese ascorbate have an apparent role in managing OA and preserving joint health. Combined, they function as building blocks to some of the normal constituents of joint cartilage and they decrease the effect of some of the destructive enzymes present within an osteoarthritic joint. Pragmatically, it is important to realize that these nutraceuticals are not subject to the same stringent regulatory guidelines as pharmaceuticals. In fact, one study showed that as many as 84% of the human over-the-counter nutraceutical products vary widely in their composition and fail to meet their label claims. We recommend a high-qualityproduct that combines the glucosamine & chondroitin sulfate ingredients with avocado-soya unsuponifiables (ASU). ASU decreases inflammation and, in human osteoarthritis trials, decreases pain scores and reliance upon non-steroidal anti-inflammatory drugs (NSAIDs). Combined, these nutraceuticals (also referred to as "joint protective compounds" [JPC's]) have a role in joint health preservation and in the comprehensive management of OA.

NSAIDs decrease inflammation and, therefore, typically improve patient comfort and mobility in OA patients, but should not be seen as contributing to joint health preservation. Rare adverse reactions most commonly involve gastrointestinal, liver and/or kidney function. We recommend the medication be discontinued and immediate veterinary consultation sought if diarrhea, vomiting, melena, lethargy or lack of appetite is noted. Often, we recommend blood tests be performed prior to NSAID therapy and periodically throughout treatment.

Adjunctive systemic medicationscan decrease reliance upon NSAIDs or augment their effects in OA patients, but few of them have a role in joint health preservation.

Intra-articular (IA) injectionsof corticosteroids or hyaluronic acid have been advocated for osteoarthritic joints. CorticosteroidIA injection remains controversial with some studies demonstrating adverse effects on cartilagewhile others demonstrate symptomatic relief and chondroprotective effects. In dogs with OA induced by cranial cruciate ligament transection, triamcinolone hexacetonide, 5mg, IA reduced osteophyte size, cartilage erosions, and the histological severity of OA structural cartilage changes with no electron microscopic evidence of increased cell degeneration

or death. Corticosteroid IA administration should be avoided immediately following arthroscopy because increased risk of septic arthritis. Intra-articular injection of hyaluronanhas been advocated for many species including horses and humans, but its effects on naturally occurring OA in dogs has not been extensively reported. Short-term symptomatic relief has been reported in a small-scale study of dogs with naturally-occurring OA following a two injection protocol (3 weeks apart) with a high molecular weight product. Hyaluron's mechanisms of action may be transient improvement in joint lubrication and its longer lasting analgesic, pro-anabolic and anti-catabolic effects. Variation in molecular weight of the product, dosage, frequency of administration as well as species and OA model evaluated may account for the variation in reported outcomes. Disadvantages of its use in dogs may relate to its relatively high cost, the apparent necessity of repeated IA injections, and lack of robust efficacy data reported in dogs.

Biologic therapies such as cytokine therapy, stem cell, and platelet rich plasma (PRP) therapy are gaining advocates, especially when more conventional therapies have failed to restore patient comfort and quality of life. Upset of the critical balance of pro-inflammatory cytokines and anti-inflammatory cytokines is well-recognized in the pathogenesis of OA. Conditioning of autologous serum from a whole blood sample has been used to stimulate the production of interleukin receptor antagonist protein (IL-Ra), IL-4 and other anti-inflammatory cytokines. Intra-articular injection of this autologous conditioned serum has resulted in significant clinical and histologic improvement in OA-affected joints. To circumvent the need for repeated IA injections, viral vector gene transfer technology has been employed to obtain sustained levels of IL-Ra. This approach has resulted in elevated intra-articular expression of IL-Ra, significant improvement in clinical parameters of pain and disease, preservation of articular cartilage, and beneficial histologic effects on synovial membrane and articular cartilage in equine and canine osteoarthritis models. Research and development for improved viral vectors is underway. Stem cell therapy involves stem cell isolation from various tissues including adipose and bone marrow and, in some instances, culture expansion. Injection of these cells into osteoarthritic joints may result in clinical improvement as compared to negative controls (sham injection), but few studies have evaluated this therapy compared to a positive control (current standard of care) therapy in the canine. In the absence of robust evidence for effectiveness over the current standard of care, the expense of stem cell therapy make it difficult to justify for the majority of canine OA patients. PRP is defined as an autologous concentration of platelets in a small volume of plasma. PRP contains a concentration of critical growth factors that are actively secreted by platelets to stimulate cellular proliferation, migration,

differentiation and matrix synthesis. These factors can affect chondrocyte metabolism, chondrogenesis, and improve cartilage healing in vivo. Several human studies show favorable clinical outcomes as compared to intra-articular injection of hyaluronic acid (HA). There is substantial variation in the preparation and formulation of autologous PRP, but affordable point of care preparation is certainly feasible.

Mechanical therapiesfor OA include pulsed ultrasound and shockwave. There is some evidence for the efficacy of shockwave therapy in the management of osteoarthritis. There is a need for more controlled studies in the canine to determine the efficacy relative to the current standard of care and recommended therapy protocols.

Surgical treatment, often required for effective management of OA, may involve joint stabilization, removal of cartilage/bony chips, or joint replacement. The relative importance and timing of surgery is variable depending upon the condition underlying the osteoarthritis. Typically, early intervention is indicated to maximize the surgical efficacy with regard to preservation of joint health.

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CARDIOLOGY

UPDATED APPROACH TO HEART FAILURE IN DOGS

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Congestive heart failure is present when a patient is showing clinical signs as a consequence of retention of an excessive volume of fluid secondary to the presence of heart disease. Heart disease leads to an "underfilling" of the arterial circulation which initiates a cascade of nervous and endocrine compensatory responses. Chronic stimulation of these pathways results in retention of excessive sodium chloride and water leading to an expansion of the circulating fluid volume. This additional volume is primarily retained in the venous vasculature leading to elevated venous and capillary pressures. Ultimately venous and capillary pressures become so elevated that fluid can no longer be retained within the vasculature. This fluid leaks out into tissues in the form of oedema, or into body cavities in the form of effusions such as a pleural effusion or ascites. When a patient is demonstrating clinical signs as a consequence of this excessive retention of fluid they can be said to be showing signs of congestive heart failure.

Some patients with congestive heart failure are concurrently showing signs of inadequate cardiac output. These signs will be apparent as signs of poor perfusion such as pallor, cold extremities, weakness and lethargy. These patients may be found to be hypotensive if blood pressure is measured. The presence of concurrent signs of poor output complicates the treatment of congestive heart failure.

When treating patients with signs of heart disease and heart failure, clinicians should ideally practice "evidence based medicine". There is good evidence to support the use of some agents in the treatment of patients with chronic congestive heart failure; particularly those with common underlying diseases such as degenerative mitral valve disease (DMVD) and dilated cardiomyopathy (DCM). Few if any clinical trials have evaluated the effectiveness of different agents in the setting of acute heart failure management. The most effective treatment of patients presenting acutely with signs of congestive heart failure therefore depends upon an understanding of what can be achieved with the different agents available.

There are a few key questions that clinicians need to ask themselves when managing these patients and the management process will be explained by going through these questions and possible responses.

Are the clinical signs definitely a consequence of congestive heart failure?

Typical signs of congestive heart failure are signs of increased respiratory rate and effort. Sometimes other signs may be present such as abdominal distension. Increased respiratory rate and effort and abdominal distension can also occur secondary to diseases of other body systems. In order for treatment to be effective it must be targeted at the appropriate underlying cause.

To conclude that a patient definitely has congestive heart failure one must establish that the patient has heart disease of sufficient severity to be likely to be the cause of the signs and evidence that those signs directly relate to the patient's heart disease. A diagnosis of heart failure is therefore usually made on the basis of a combination of a history of clinical signs indicative of heart failure, physical examination findings indicative of the presence of heart disease and the results of ancillary diagnostic tests which help to establish that the observed signs are a consequence of the patient's heart disease. Some causes of heart failure are best managed in ways other than through the administration of drugs. The best example of this is in patients with right sided congestive heart failure secondary to a pericardial effusion; patients with this presentation require pericardiocentesis and drug therapy is not necessary.

If a patient is sufficiently stable to undergo diagnostic testing prior to initiation of treatment it may be valuable to obtain a thoracic radiograph with or without echocardiography.

Is the patient showing signs of poor perfusion and low cardiac output at the same time as the signs of congestion?

This is important because drugs that are likely to be used to manage congestive heart failure will at best have a neutral effect on cardiac output and at worst, worsen signs of poor output. Patients with signs of poor output are likely to require additional treatment in an effort to improve perfusion as well as simply treatment aimed at reducing signs of congestion.

Dealing with signs of congestion.

The most effective agents for the control of signs of congestion are diuretics, with or without concurrent administration of venodilating agents. These agents will hopefully reduce the circulating fluid volume and, through dilatation of the veins, increase the capacity of the venous vessels to hold fluid at lower pressures. Reducing the volume of fluid and increasing the capacity of the veins should reduce pressure sufficiently to eliminate the tendency for fluid to leak out of the capillaries. If hydrostatic pressures fall sufficiently the oedema that has established in tissues should be reabsorbed back into the intravascular volume. This will lead to resolution of clinical signs caused by oedema.

Where signs of increased respiratory rate and effort are present due to a pleural effusion, rapid improvement will be obtained by thoracocentesis. Medical management should also be introduced to prevent the reformation of the effusion. Sometimes a large volume of ascites may cause respiratory embarrassment by interfering with the movement of the diaphragm. If this is the case abdominocentesis may also be of benefit. In patients with ascites that is not compromising respiration, medical management alone may be sufficient as reducing circulating fluid volume and venous pressures should lead to the reabsorption of the abdominal effusion.

The most frequently used diuretic is furosemide. It has a wide dose range and can be administered by a wide variety of routes. In the acute heart failure setting intravenous administration is preferable as the onset of action will be rapid. This is usually started as intravenous boluses initially at 1-2 mg/kg every 6-8 hours. In patients with severe signs, or those that have developed signs of congestion despite already being on diuretics, higher doses and shorter intervals between doses may be necessary. Doses of 4 mg/kg can be given as repeated bolus injections with intervals of as little as one hour between doses. The drug can also be given by constant rate infusion. Although daily doses in excess of 12 mg/kg can be administered in the acute setting, if a patient is poorly responsive to repeated boluses of furosemide then adding other types of drug to the treatment regime may well be necessary.

Venodilators can be given in addition to diuretics to reduce filling pressures and relieve signs of congestion. Some nitrate vasodilators e.g. glyceryl trinitrate are thought to have their effect predominantly through vasodilation. Balanced vasodilators such as nitroprusside also have effects on veins as well as arteries. Evidence for the effectiveness of venodilators is not strong and nitroprusside can only safely be administered in an ICU setting where patients can be monitored very carefully.

Dealing with signs of poor output.

Diuretics and venodilators will, at best, have a neutral effect on cardiac output i.e. they will not improve signs of poor perfusion and the best one can hope for is that these signs will not be worsened by aggressive diuresis. Decreasing venous pressures will tend to decrease cardiac filling pressure which in some situations may lead to a worsening of signs of poor cardiac output.

In patients with signs of poor perfusion due to non-cardiac diseases, as a general rule, cardiac output may be improved by the administration of intravenous fluids. In patients with heart failure this is not the case. When heart failure is present hypovolaemia does not underlie the poor perfusion. If the patient is showing signs of congestive failure there is direct clinical evidence that they have an excessive circulating fluid volume. The

problem here is one of distribution of fluid. Intravenous fluid therapy is almost always contra-indicated in patients with active signs of congestive heart failure. The only exception is as a means of delivery of drugs when the patient is requires a constant rate infusion. Giving more fluid will only worsen the signs of congestion and probably not improve the signs of poor perfusion. Other ways of improving perfusion will be necessary.

Signs of poor perfusion may be improved in patients with heart failure by the administration of drugs that increase the force of cardiac contraction (inotropes) such as pimobendan or dobutamine; drugs that decrease vascular resistance (arteriodilators) such as pimobendan, Angiotensin Converting Enzyme (ACE) inhibitors or nitroprusside; and drugs that optimise cardiac rate and rhythm in patients with arrhythmias (anti-arrhythmic drugs). If a patient presents with concurrent signs of congestion and poor perfusion it is likely that at least one of these classes of agent will need to be administered in addition to treatment aimed directly at relieving congestion.

Pimobendan is widely available as an inotropic and vasodilating agent. There is good evidence for its effectiveness in the treatment of more common causes of congestive heart failure in dogs. It should therefore be given to most dogs with congestive heart failure as part of their chronic treatment regime. Pimobendan therapy can be initiated in the acute setting and in some countries an intravenous formulation enables effective initiation of therapy by injection. There is also some evidence of the effectiveness of this agent in the treatment of cats with heart failure secondary to myocardial disease.

Dobutamine is a sympathomimetic inotrope that can be given intravenously. It has to be given by constant rate infusion and may be pro-arrhythmic at higher infusion rates. This limits its administration to the hospital setting where the patient can be closely monitored during administration.

Oral or intravenous vasodilating agents can help improve perfusion by decreasing the resistance to ejection of blood from the left ventricle. Some hypotensive patients may not tolerate vasodilation because they are unable to increase their cardiac output to compensate for the decrease in systemic vascular resistance. This can lead, in some cases to worsening of signs of poor perfusion. Vasodilators should therefore be administered with caution to patients that are already hypotensive and blood pressure should be monitored during their administration.

What are you trying to achieve with your therapy?

It is implicit in the above discussion that drugs are being administered to patients with specific aims in mind. At the outset of therapy these aims should be defined and then the patient should be monitored to see whether these aims are being achieved. Patients should also be carefully monitored for evidence of an adverse reaction to therapy.

As evidence of a response to therapy some or all the following could be monitored

Respiratory rate – this is one of the most useful parameters to monitor as an indication of a response to therapy. Ideally the respiratory rate of a patient with heart failure should begin to decline within the first few hours of treatment. A failure of the respiratory rate to reduce would be a sign of a poor response to treatment.

Systolic arterial blood pressure – in a patient that was hypotensive on presentation a good response to therapy would be an increase in blood pressure. If a patient is not hypotensive then it may still be worth monitoring blood pressure as this may provide an indication of how well the patient is tolerating their treatment. The development of hypotension in a patient undergoing vigorous diuresis will necessitate either a modification of the diuretic regime or the concurrent administration of other therapy targeted at improving output.

Radiographic evidence of heart failure – if a radiograph was obtained demonstrating the presence of changes consistent with heart failure such as a pleural effusion or pulmonary congestion and oedema, it is useful to demonstrate that these changes are resolving after successful treatment. If however a patient's respiratory rate and effort are not improving in response to treatment there may be little point in re-radiographing a patient; unless it is to reconsider the original diagnosis.

Bodyweight – bodyweight should reduce considerably with successful diuresis.

Urination – a desired effect of treatment is to make the patient urinate. If they do not show an increase in urination in response to therapy it may be that they are poorly responsive to diuresis or the treatment may not have been effectively administered.

It is also worthwhile monitoring patients for the development of complications. In addition to the monitoring of systolic arterial blood pressure described above it is also worth monitoring **indicators of renal function and electrolytes**. These are likely to be altered by the administration of diuretics and the reduction in circulating fluid volume. The development of hypokalaemia is particularly common following the administration of furosemide. If detected then concurrent administration of an ACE inhibitor or a potassium sparing diuretic is likely to be necessary and supplementation

of potassium. If urea and creatinine begin to increase significantly this may be a sign of the patient's renal perfusion being compromised by the intensity of diuresis. This may require modification of the diuretic regime or concurrent administration of agents that may improve perfusion such as inotropic drugs.

Have the aims of therapy been achieved?

If the aims of treatment have been achieved – well done! You should now be in a position to transfer the patient from their acute treatment protocol onto a more suitable chronic regime.

If the aims of treatment have not been achieved then intensification of therapy may be necessary. Re-evaluate the original diagnosis and go back through the various steps above to choose which treatment is most likely to help achieve your aims.

Has treatment been associated with the development of any complications?

If complications have developed are they sufficiently severe to warrant decreasing the intensity of heart failure therapy? For instance it may be necessary to reduce diuretic doses in patients that develop azotaemia or hypotension. Other drugs may need to be added to the treatment regime in an effort to address the complications observed.

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WSAVA ONE HEALTH DISASTER MANAGEMENT

ONE HEALTH COMMITTEE REPORT; FOLLOWED BY: COLLABORATIVE INTELLIGENCE FOR VETERINARY PROFESSIONALS

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COLLABORATIVE INTELLIGENCE FOR VETERINARY PROFESSIONALS

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Introduction

When a disaster strikes there is a period of time where there is very little information of who has been impacted, how they have been impacted and what their immediate needs may be. Therefore, decision making is based on assumptions and previous experience until further intelligence is collated and interrogated, allowing for impacts and needs to be supported by "ground truthing". The volume, complexity and fluid nature of this work demands collaboration across a diversity of people, disciplines and organisations.[1] This is achieved by collecting real time data from organisation who are at the coal face. It provides the evidence base for strategic planning, as well as baseline information upon which situation and response monitoring systems will rely. Additionally, the information gathered will allow the response team to analyse and make decisions on support actions, confirm resources and deploy the required level of response for the affected community. It helps to build a picture of current, emerging and diminishing needs. Collectively, this creates situational awareness of the disaster, which requires constant "checking in" due to the fluid nature of disasters.

Veterinary professionals are key actors and are instrumental for animal welfare disaster management decision making. They have local knowledge and the ability to collect raw data through their networks of clients and communities, participate in ground truthing, can identify any emerging issues that may require forethought and are subject matter experts (SMEs). Collaborative intelligence is vital for crisis decision-making. However, for the data to be meaningful it requires an understanding of what intelligence is needed (purpose) and how stress can impact collection and interpretation of the data.

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Purpose of data

As a disaster unfolds, no one has a complete picture of what is happening. In the early stages decision making is based on assumptions and previous experience. However, the later can quickly create an environment whereby the focus of the response is skewed and highly probable that the response will be inadequate. Much more bridging of knowledge and insight is needed to deepen the collective knowledge of how we can, together, more effectively respond to the crises.[2] This in part is achieved by raw data collection at the coal face by those with local knowledge and at varying stages of the disaster. The assessment process occurs in sequential steps and has three phases (rapid initial response, response and recovery) to ensure the information collected is of use. The general process remains the same through each phase, but the detail of information, speed at which it is collected and how it is collected differs. During the early stages, rapid information is required to protect life and reduce further impact on welfare, infrastructure and recovery. This is known as the rapid initial assessment. The next phase is a full needs assessment. This assessment involves assessing the situation to confirm the information being received is an accurate reflection of the event. It also ensures that any subsequent response is co-ordinated, targeted, effective and reduces the likelihood of duplication of effort. This is a crucial phase in any response work as a poorly conducted assessment is likely to lead to poor planning decisions and an inadequate response. This often has flow on consequences affecting recovery efforts.

The information that is pivotal to decision making is intelligence which helps to understand the current impacts on animal welfare and what may impact later on. For example in a flood event, the initial rapid assessment needs to assist with an understanding where the flood impact is and what assistance may be required. Are people self-evacuating? What are your clients doing? Are they asking for help? What is your community doing? Do they have adequate access to veterinary care? Thinking ahead; what are the risks associated with animals being in flood water during the event as well as having access to contaminated grounds once water has started to recede? This is where proactive thinking about zoonotic diseases from a public health perspective should occur. Animals tend to be the initial indicator as animals usually become sick before people do. In New Zealand, like many countries, the regions identified as high risk of leptospirosis exposure is growing at an exponential rate.[3,5] Therefore, if cases of such diseases are presenting at veterinary clinics post flooding this should be communicated as an environmental indicator to the agencies responsible for co-ordinating the recovery. The risk index for exposure to people and animals after a flood event continues to rise and should be regarded in proactive public health measures. A pertinent example of collaborative intelligence.

Stress physiology

When a disaster occurs, people affected by the traumatic event experience a range of early reactions (physical, psychological, emotional and behavioural).[4] These reactions may interfere with their ability to cope and can impact how we observe and interpret what is occurring around us. When in a relaxed state the para-sympathetic system dominates, however, this can change when the fight or flight response is initiated. This is our usual response when we come across an emergency situation. When in an aroused state the sympathetic system dominates to assist with our survival. This is very effective to increase alertness and prepares us for a quick response, however, we need to ensure it is kept at an optimal level as we can tip over the edge and become ineffective due to paralysing stress.

When comparing what our bodies are doing physiologically in a relaxed state as opposed to a fight / flight response we can see where some of the positive and negative responses come in to play. Our brains do not distinguish between physical threat (attacked by a dog) and psychological threat (stress of making decisions in an emergency). When in survival mode the brain is in a stressed state and this reactivity makes it quite challenging, if not outright impossible to be open and receptive to others. We become hyper-vigilant, experience negative thinking, have a sense of urgency "I have to get it done and it has to be done now!" and have tunnel vision. This affects our ability to concentrate, we forget things and start to ruminate (thoughts going around and around). You have no doubt experienced this on occasions and see it often in your clients. This is a dangerous state to be in as we need our smart brain to "click" in, the neocortex. This part of the brain allows us to think and reason along with regulating the limbic system, our emotions. It allows us to see what is going well, gives us patience and the ability to wait. It creates calmness, equanimity (remaining calm and undisturbed), allows us to be able to keep an even keel, see the subtleties and sustain attention. These are all the qualities that are required to be a valuable resource in an emergency situation. This is known as mindsight and is vital for collaborative intelligence. Part of the process of developing mindsight involves reducing reactivity when it is not actually necessary. This can be achieved by stopping, taking deep breaths and focusing on the task at hand. After doing breathing exercises we can start thinking rather than simply reacting.

Actions need to be driven by objective decisions rather than by emotions. Therefore, you need to ask yourself are you thinking or just reacting. The answer could mean the difference between success and failure in an emergency situation.

Conclusion

Collaborative intelligence is the measure of your ability to think with others on behalf of what matters to us all. To access that intelligence, we must learn to dignify differences in how we think and use them to face complex challenges.[2] Expertise in one area is of little use if not open to collaborate with others to make the expertise valuable. Veterinary professionals are key actors in disaster management and are instrumental for crisis decision making through collaborative intelligence.

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WSAVA VACCINATION GUIDELINES

DOG VACCINATION

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Global Canine Vaccination Guidelines

There are two sets of canine vaccination guidelines available: those produced by the American Animal Hospital Association [1] and those from the WSAVA VGG [2-4]. The fundamental principle of both sets of guidelines, as encapsulated by the VGG, is that '**We should aim to vaccinate every animal with core vaccines. Non-core vaccines should be given no more frequently than is deemed necessary.**'

The WSAVA guidelines suggest that we should aim to vaccinate MORE animals. This relates to the phenomenon of 'herd immunity'. Herd immunity suggests that, for some diseases, where a minimum proportion (for example 75%) of a herd of animals is vaccinated, it is difficult for an infectious disease outbreak to occur in that herd. The 'herd' for the canine practitioner is the population of dogs living within his or her practice area – and our aim should be to have as many of these animals vaccinated as possible, in order to reduce the chances of disease outbreak in the herd. This is particularly important in the context of canine rabies. Where a mass vaccination campaign results in at least 70% of the dog population receiving vaccine, there is marked impact on the prevalence of canine and human rabies.

In order to apply the principles of vaccination guidelines, it is firstly necessary to understand the definitions of 'core' and 'non-core' vaccines. CORE vaccines are those that all animals should receive to protect them against potentially lethal diseases of global significance or where legislation may dictate [i.e. canine rabies]. The use of NON-CORE vaccines in certain animals is dictated by geographical location, lifestyle and exposure risk. Some vaccines are NOT RECOMMENDED because there is little scientific justification for their use.For dogs, the core vaccines are those that protect against canine distemper virus (CDV), canine adenovirus (CAV) and canine parvovirus-2 (CPV). In any country in which rabies is an endemic disease, then rabies vaccination is also

considered core for dogs. Non-core canine vaccines include those that protect against leptospirosis, canine parainfluenza virus (CPi), **Bordetella bronchiseptica** and **Borrelia**. Canine coronavirus (CCV) vaccine is not recommended as there is little evidence that CCV is a primary enteric pathogen or that the vaccine can protect against such infection. The WSAVA global guidelines do not consider some vaccines that have very restricted geographical availability (e.g. vaccines against canine herpesvirus, **Leishmania** or **Babesia**).

WSAVA guidelines provide generic advice to practitioners, but it is impossible to ensure that the guidelines are tailored to best fit the local situation in each of the 86 WSAVA member countries. The VGG encourages national associations to adapt and modify the guidelines for local use where appropriate. This process might involve altering the classification of a vaccine. For example, in the UK, **Leptospira** vaccine is generally considered core for the dog and attempts are now being made to provide data that define disease prevalence and characterize locally circulating serovars [5].

Core Vaccination of Puppies

The vaccination of puppies is determined by the transfer of maternally-derived antibody (MDA) from the bitch in colostrum. This antibody is crucial for protection of the pup during early life, but simultaneously blocks the endogenous immune response of the puppy to core vaccination with most available modified live virus (MLV) vaccine products. Canine immunoglobulin has a half life of around 11 days and there is progressive decline in MDA concentrations in pups over the first weeks of life. The 'window of susceptibility' occurs when there is no longer sufficient maternal antibody to provide full protection from infectious disease, but where sufficient antibody remains to block the ability of the pup to make its own immune response to MLV core vaccine. Traditionally, this window has been taken to occur at between 8 – 10 weeks of age, but new evidence shows that higher titre vaccines increase maternal antibody concentrations leading to persistence of MDA for longer periods of time. Studies have now shown that around 1 in every 10 puppies has 'blocking' levels of MDA against CPV at 12-14 weeks of age. For this reason, vaccination guidelines now recommend that puppy vaccination (with MLV core vaccines) starts at 8 – 9 weeks of age, with a second vaccine 3 – 4 weeks later and a third vaccine given at 16 weeks of age or older. The puppy protocol now includes a fourth core vaccine, which optimally would be given at 26 weeks of age (and replaces the vaccine that has previously been given either at 12 months of age or 12 months after the anniversary of the 16 week vaccine). This fourth vaccine might conveniently be given at the time of neutering of the puppy. By this schedule, all puppies (except genetically-determined low or non-responders) will be able to mount protective

immunity by 26 weeks of age. A recent study from Australia has shown that the most significant cause of CPV 'vaccination failure' in puppies is failure to deliver the final vaccine in the puppy series at 16 weeks or older [6]. Where rabies is endemic, pups should receive 1 dose of vaccine at 12 weeks of age or older, but the VGG suggests that in a high-risk situation (i.e. in an endemic area with known local cases), a second dose of vaccine may be given 2 - 4 weeks later.

The 'window of susceptibility' to infectious disease for puppies overlaps with the ideal period to undertake socialization. The WSAVA guidelines endorse strategies to socialize puppies (including 'puppy parties'), but recommend simple measures to minimize the low risk of transmission of infectious disease at such events.

Core Vaccination of Adult Dogs

For adult dogs, MLV core vaccines should be given no more frequently than every 3 years. For CDV, CAV, CPV there is excellent correlation between the presence of serum antibody (as detected by virus neutralization test or haemagglutination inhibition test or in-house serological test kits) and protection from challenge with infectious virus. There are extensive data showing that protective antibody persists in adult dogs, even when they have only been vaccinated as puppies up to 14 years previously [7, 8]. More importantly there are data that underpin the legal registration of canine core MLV vaccines for either 3 or 4 years, based on challenge studies that show that vaccinated dogs resist infection for that minimum period after vaccination (the minimum duration of immunity, DOI). Other experimental data show that dogs vaccinated as puppies only are protected from live virus challenge with CDV and CPV at 9 years of age [7]. On this basis, most of the internationally produced canine MLV core vaccines used in the USA, Europe and some other countries now have a licensed minimum DOI of either 3 or 4 years. However, in other countries the identical international vaccines still carry a 1-year licensed DOI, but may still be used less frequently with informed client consent. This is also true for the internationally produced killed adjuvanted rabies vaccines that may legally be given every 3 years rather than annually. Where such products are available with a 3-year licensed DOI, but governmental legislation insists on annual rabies vaccination, it is beholden on the veterinary profession to lobby for changes to the law in order to prevent unnecessary revaccination of adult dogs. For example, in the USA, state laws changed gradually, such that now every US state stipulates triennial revaccination of dogs against rabies.

The currently accepted core revaccination schedule for adult dogs is therefore revaccination every third year with CDV, CAV, CPV and rabies. Where rabies revaccination is still required annually, the schedule might be CDV, CAV and CPV triennially and rabies

annually. Triennial revaccination is simply better evidence-based medical practice, but also reduces the number of unnecessary vaccines given to adult dogs and therefore reduces the chances of adverse reactions.

Vaccination according to WSAVA guidelines necessitates being able to source vaccine ranges where products are formulated with minimal antigenic content (e.g. combination MLV DAP vaccines, with separate non-core components). In many countries, only multiple combination products are available and so veterinarians in those regions should urge manufacturers to license the minimal component ranges available elsewhere in their countries.

Non-core Vaccination

Non-core vaccines should be selected for the individual dog based on assessment of that particular animal's risk of exposure to the disease and assessment of the benefits of vaccination to that pet versus the risk of adverse reaction. Decision making for non-core vaccines would be facilitated by having available good quality data and disease distribution maps related to small animal infectious diseases. Unfortunately, with the exception of rabies in the USA and Europe, such distribution maps are not widely available. Some national schemes have been developed by industry or academic groups which allow practitioners to input cases of particular infectious diseases into a database that presents the information as disease distribution maps. Monitoring the distribution and evolution of infectious diseases is an important part of vaccinology. An excellent example is canine leptospirosis, which has recently attracted much research interest as the importance of particular serovars in causing canine disease in different geographical locations is determined. This new knowledge has led to the introduction of trivalent or tetravalent canine **Leptospira** vaccines in the US and Europe; the antigenic composition of which is related to the prevalence of serovars in each location. Similarly, in some countries (e.g. the USA and Korea) vaccines are available to protect against different strains of canine influenza virus (CIV). This infection remains an issue for dogs that are intensively kenneled and transported (e.g. racing greyhounds), but the CIV vaccine would not be recommended for general use among pet dogs.

Non-core vaccines may be included into the puppy vaccination schedule if dictated by risk assessment. For example, intranasal vaccines protecting against some elements of the canine infectious respiratory disease complex (i.e. CPi and **Bordetella bronchiseptica**) might be used as early as 4 weeks of age as a single vaccination. The VGG now recommends that where **Leptospira** vaccines are used in puppies, the required two doses of vaccine be co-delivered with core vaccines during the puppy schedule (for example at 8 and 12

weeks of age). A major difference between MLV core vaccines and all of the non-core vaccines is that non-core vaccines (where used) require annual boosters as their DOI is no greater than the licensed 12 month period. Adult dogs given non-core vaccines must therefore receive these annually. In many situations therefore, adult dogs receive 'annual revaccination', but just with fewer components than might have been used in the past. **The Annual Health Check**

All aspects of vaccination should fall under an annual health check programme that reduces the emphasis on vaccination as the primary reason for visiting the practice and considers holistically the overall health and wellbeing of the pet in a preventive healthcare programme. A discussion about which vaccines (or serological tests) might be offered in any one year is just one part of the annual health check. The importance of vaccination can be reinforced by using the VGG fact sheets available from the WSAVA website. Vaccination (or serology) should be appropriately invoiced so emphasis is placed on the professional consultation.

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GASTROENTEROLOGY / LIVER

CHRONIC PANCREATITIS IN DOGS

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Definitions

Pancreatitis in dogs shows a spectrum of disease from mild, subclinical to acute, necrotising and fatal. Pancreatitis is classified based on histopathological appearance. There is no 'gold standard' for histological description of pancreatitis in animals (unlike the liver). The author favours the definitions in human medicine which define pancreatitis as 'acute' or 'chronic' based on its histological appearance and NOT how it appears clinically. These definitions are summarized in table 1 below. Chronic pancreatitis (CP) is defined as: 'a continuing inflammatory disease characterized by the destruction of pancreatic parenchyma leading to progressive or permanent impairment of exocrine or endocrine function or both. The gold standard for diagnosis is histology but this is rarely indicated or performed in dogs (or humans). Noninvasive diagnosis is difficult with the currently available diagnostic imaging, and blood tests have a lower sensitivity than for acute disease, which may explain why its prevalence is often under-estimated.

Table 1: *Proposed histopathological and functional definitions of acute and chronic pancreatitis in dogs based on human definitions*

	ACUTE PANCREATITIS	CHRONIC PANCREATITIS
Histopathological appearance	Neutrophilic inflammation; acinar necrosis, peripancreatic fat necrosis and oedema in varying amounts. Potentially completely reversible with no permanent pancreatic architectural or functional loss	Mononuclear (lymphocytic +/- plasmacytic) inflammation and fibrosis. Permanent, irreversible disruption of pancreatic architecture. Cases with concurrent neutrophilic inflammation and necrosis but underlying fibrosis fall in to this category.
Functional changes	High risk of systemic inflammatory disease and multi-organ failure during acute disease. No permanent pancreatic functional changes on recovery	Characterised by progressive loss of exocrine and endocrine function. Exocrine pancreatic insufficiency (EPI) and/or diabetes mellitus (DM) develop in end stage disease after 80-90% of pancreatic tissue has been lost
Clinical appearance	Spectrum from severe and fatal (usually necrotizing) to mild and subclinical (less common)	Spectrum from mild, low-grade intermittent gastrointestinal signs (most common) to an acute-on-chronic episode indistinguishable from classical acute pancreatitis

Histopathological descriptions are useful to understand the pathology and progression of the disease but are not very useful clinically as most cases do not have a pancreatic biopsy performed and either chronic or acute pancreatitis episodes may be mild or severe and either may be recurrent. However, recognizing the potential for underlying chronic disease in an animal which presents acutely is very important for long term management because it allows the clinical to realize that the dog may develop EPI or DM in the future, and to recognize and treat these appropriately.

Causes of chronic pancreatitis

The cause of CP in dogs is usually unknown. Any age or breed of dog can be affected, but the most typical signalment is a middle-aged to old dog particularly a Cavalier King Charles Spaniel (CKCS), Cocker Spaniel, Collie, or Boxers in the UK. An independent large study of EPI in the UK found an increased prevalence in older CKCS, supporting this breed association. Some cases may represent chronic relapsing cases of acute disease, but many cases are truly 'chronic' from the outset, with an initial mononuclear infiltrate.

Autoimmune chronic pancreatitis in the English Cocker Spaniel

The particular form of chronic pancreatitis recognized in English Cocker Spaniels in the UK is thought to be an autoimmune. As in human autoimmune pancreatitis, it typically affects middle-aged to older dogs, with a higher prevalence in males, and at least 50% of affected dogs subsequently develop DM, EPI, or both. Dogs also often have other concurrent autoimmune disease, particularly keratoconjunctivitis sicca and glomerulonephritis. There is often a mass-like lesion on ultrasound, and biopsies show a typical perilobular diffuse fibrotic and lymphocytic disease centered on perilobular ducts and vessels, with loss of large ducts and hyperplasia of smaller ducts. Immunohistochemistry shows a preponderance of duct and vein-centered CD3+ lymphocytes (i.e.; T-cells). Recent work in humans has identified a strong association with plasma cells that secrete one subgroup of imuunoglobulin G, IgG4. The disease in humans has been redefined as multisystemic because of the frequent involvement of other organs. It is now defined as IgG4 positive sclerosing disease and concurrent keratoconjunctivitis sicca, sialoadenitis, biliary tract disease, and glomerulonephritis are common. Early work in English Cocker Spaniels also shows IgG4-positive plasma cells in the pancreas and kidney (Watson et al, 2012) and also demonstrated an association with a particular DLA (dog leukocyte antigen) in ECS, supporting the theory of autoimmunity (Bazelle et al 2013). The disease in humans responds well to steroid therapy, including a reduction in insulin requirements in some diabetics. There are not yet any controlled trials evaluating the use of immunosuppressive drugs in English Cocker Spaniels with chronic pancreatitis, but there is now enough circumstantial evidence to justify their use in this particular breed. However, the clinician should note that this is very breed- specific; terriers in Britain, for example, have a different histopathologic and clinical picture of disease that does not appear to be autoimmune. The use of steroids in terriers with chronic pancreatitis is not recommended.

Clinical signs

The clinical signs of pancreatitis obviously vary with the severity of the disease. It is only the severe, acute cases in dogs which show the classical signs of acute vomiting, cranial abdominal pain ± "praying" stance. Concurrent colitis with the passage of small amounts of faeces with fresh blood is common due to local peritonitis affecting the transverse colon as it courses close to the left limb of the pancreas. Severe cases may present collapsed and dehydrated with signs of shock and, in very severe cases, there may be renal shutdown, respiratory distress and DIC.

At the other end of the spectrum, low grade acute or chronic cases may show few clinical signs: most commonly, anorexia with or without mild bouts of colitis and occasional vomiting, increased borborygmi and no or mild abdominal pain. Dogs with CP, regardless of the cause, most commonly present with mild intermittent gastrointestinal signs. Typically they have bouts of anorexia, occasional vomiting, mild hematochezia, and obvious postprandial pain, which often goes on for months to years without presentation to a veterinarian. The trigger for finally seeking veterinary attention is often an acute-on-chronic bout or the development of DM or EPI. Dogs may become more playful and less picky with their food when they are switched to a low-fat diet, suggesting they previously had postprandial pain. Chronic epigastric pain is a hallmark of the human disease and is sometimes severe enough to lead to opiate addiction or surgery, so it should not be overlooked or underestimated in small animal patients. In more severe, acute-on-chronic cases, the dogs are clinically indistinguishable from those with classical acute pancreatitis (see above) with severe vomiting, dehydration, shock, and potential multi-organ failure. There is a tendency for the first clinically severe bout to come at the end of a long (often years) subclinical phase of quietly progressive and extensive pancreatic destruction in dogs. It is very important for clinicians to be aware of this, as these dogs are at much higher risk of developing exocrine and/or endocrine dysfunction than those with truly acute pancreatitis; in addition, they usually already have protein-calorie malnutrition at presentation, which makes their management even more challenging. In some dogs, there are no obvious clinical signs until the development of EPI, DM, or both. The development of EPI in a middle-aged to older dog of a breed **not typical for pancreatic acinar atrophy** has to increase the index of suspicion for underlying CP. The development of EPI or DM in a dog of cat with CP requires the loss of approximately 90% of exocrine or endocrine tissue function respectively, implying considerable tissue destruction and 'end-stage' disease.

FASAVA/HILLS FELINE MEDICINE

NEW INSIGHTS INTO TREATING HYPERTHYROID CATS

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NEW INSIGHTS INTO TREATING HYPERTHYROID CATS

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Hyperthyroidism remains the most common endocrine disease in cats with a prevalence of 10 % or more in older cats presenting to first-opinion veterinary practices. The aetiology remains unclear and although several risk factors have been associated with the disease, none are definitive and therefore prevention is not possible. Benign adenomatous hyperplasia or adenoma of one or more commonly both thyroid lobes is the most common lesion within affected thyroid tissue and at least initially, the prognosis is good with effective therapy. Palliative treatment options consist of control of thyroid hormone production through administration of the antithyroid drugs or alternatively through feeding an iodine restricted food. Curative options include removal of affected thyroid tissue surgically or destruction through the use of radioactive iodine.Most endocrinologists consider radioactive iodine as being the gold standard treatment method. However, it is not always available and may not be suitable for all hyperthyroid cats. Most commonly, a treatment choice is individualised for each cat taking into consideration the advantages and disadvantages of each, severity of the illness, facilities and expertise available, cost and owner choice.

Hyperthyroidism is a disease of older cats and it is not unexpected that a proportion of cats present with concurrent non-thyroidal illness. Hyperthyroid cats with pre-existing azotaemia have a reduced survival time. However, many cats do not have azotaemia prior to treatment but develop it after induction of euthyroidism[1]. All treatment options can result in unmasking of kidney disease. Predicting those cats that will develop post-treatment azotaemia is difficult but there is some evidence that measurement of serum SDMA concentrations and urine specific gravity may be useful in predicting its occurrence[2]. Development of azotaemia post treatment does not apparently affected survival[3]. However, there is some evidence that development of hypothyroidism can be detrimental to kidney function and may adversely affect survival[4].

Medical management

Methimazole and carbimazole are the two antithyroid drugs most frequently used for preoperative and long-term medical management of hyperthyroidism because of their consistent and potent effect in lowering thyroid hormone concentrations and relatively limited occurrence of serious adverse effects. Both are actively concentrated by the thyroid gland where they inhibit thyroid hormone production. Carbimazole is converted to methimazole and only methimazole accumulates in the thyroid gland. Methimazole is available as both human and veterinary licenced formulations (Felimazole® (1.25, 2.5 and 5 mg), Dechra Veterinary Products; Thiafeline® (2.5 and 5 mg), Animalcare; Thyronorm® (5 mg/mL liquid), Norbrook). Carbimazole is also available as a preparation for human use and a novel once daily controlled-release formulation (10 or 15 mg tablets) is licenced for cats in Europe (Vidalta® (10 and 15 mg), MSD Animal Health). Administration with food significantly enhances absorption. For long-term management, once euthyroidism has been achieved, the daily dosage is adjusted aiming for the lowest possible dose that effectively maintains control.Long-term monitoring of cats involves regular assessment of clinical signs and serum total T4 measurements every three to six months with reassessment of total T4 concentration 10 days to 3 weeks after each dose adjustment. Whether antithyroid medication is given once or twice daily has little impact on the timing of samples for monitoring purposes[5]. It is generally accepted that total T4 concentration should be maintained within the lower end of the reference interval[6]. In many cats, antithyroid drug therapy results in serum total T4 concentrations below the reference interval. Although overt clinical signs of hypothyroidism rarely develop, and surgical risks are not increased, hypothyroidism should be avoided because if its detrimental effect on kidney function.Compliance with oral medication can be problematic in fractious or inappetant cats. Drug absorption is also potentially affected by concurrent gastrointestinal disease particularly for those cats that vomit. Methimazole and carbimazole can be reformulated for transdermal application and appear equally as efficacious as oral preparations[7]. Recommendations for starting doses and follow-up adjustments are similar for transdermal and oral administration. There are few commercially available transdermal products. Custom formulation increases expense of therapy and stability of the product is not guaranteed. There are health concerns regarding exposure of humans to methimazole.There is growing evidence that medical therapy may not be as efficacious in the long-term. Persistent hyperthyroidism, recurrent hyperthyroidism, increasing difficulty in controlling hyperthyroidism despite increasing drug dosages, and a yo-yo effect between hypothyroidism and hyperthyroidism appear to be common over time.

Continuing efficacy of antithyroid medication is highly dependent on good owner and cat compliance and this can be difficult to maintain over prolonged periods. Importantly medical management does not address the underlying cause of hyperthyroidism and over time the pathological changes in the thyroid gland progress and the prevalence of extreme goitre, multifocal lesions, intrathoracic thyroid masses and suspected malignant transformation increase[8]. Lack of efficacy long-term should be considered as a disadvantage of oral/transdermal medication.

Restricted iodine diets

A restricted iodine diet has been marketed for optimising thyroid health (Hill's y/d). Exclusively feeding this diet has been shown to normalize thyroid hormone concentrations in some affected animals but usually only in mildly affected cases[9]. However, clinical signs may not fully reverse suggestive of persistent hyperthyroidism throughout treatment and the time to develop euthyroidism can be prolonged. There appear to be issues of compliance that may be related to palatability. In cats where other treatments are refused or not feasible it may be a useful option. Other food sources must not be available.

Surgical thyroidectomy

In practice, surgical thyroidectomy is often considered a treatment of choice particularly if radioactive iodine is unavailable. The majority (>70 %) of cats require bilateral thyroidectomy. If a unilateral thyroidectomy is carried out future monitoring for recurrence of the condition is required. Routine bilateral thyroidectomy, whilst increasing the risk of post-operative complications, obviates the need for decision-making at the time of surgery. However, ectopic thyroid tissue should be considered if hyperthyroidism persists after routine thyroidectomy. A major concern is the development of postoperative hypocalcaemia that occurs within one to five days of surgery. It is difficult to accurately assign a risk for hypocalcaemia in a cat undergoing bilateral thyroidectomy as it is highly surgeon dependent. However, with experience, a low rate of postoperative hypocalcaemia (< 10 %) and recurrence (5 %) is expected. As for medical treatment regular postoperative monitoring is required to ensure that permanent hypothyroidism does not develop.

Assessment for hypothyroidism

Irrespective of treatment modality, hypothyroidism is a potential sequel. Although no strict rules exist, suppressed total T4 values should prompt consideration of further investigation for hypothyroidism. Alternatively it is probably best practice to monitor cats through measurement of total T4 and TSH as it appears that TSH concentrations more accurately reflect reduced thyroid function[10]. Given that high TSH values are expected,

use of the canine assay is considered reasonable. A low total T4 and high TSH should be interpreted as indicating hypothyroidism and managed by decreasing the antithyroid drug dosage or implementing L-thyroxine supplementation. Reference interval total T4 concentrations with high TSH may also represent reduced thyroid function and should be carefully monitored particularly if azotaemic.

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TIPS FROM THE EXPERTS FOR THE MANAGEMENT OF…

HIGH LIVER ENZYMES

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TIPS FROM THE EXPERTS FOR THE APPROACH AND MANAGEMENT OF

HIGH LIVER ENZYMES

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Liver Enzymes vs Liver Function Tests

Liver test abnormalities can be categorised loosely into markers of hepatocellular injury, cholestasis, or impaired liver function.

Transaminases ALT and AST

ALT is a cytoplasmic enzyme of hepatocytes that is more specific than AST. ALT has a half-life of 6 hours in a cat and 48-60 hours in a dog, making it a good indicator of continued liver injury.[1]AST is a mitochondrial-bound enzyme found in high concentrations in hepatocytes. Consequently, its increase indicates cell death. However, AST is also found in myocytes and red blood cells. The half-life is 77 minutes in a cat and 22 hours in a dog.[1]

Indicators of Cholestasis

ALKP and GGT typically increase with cholestatic disease or drug induction. Cholestasis increases the surface tension of the bile canaliculi and ductules, inducing enzyme production. ALKP has a half-life of 66-70 hours in dogs and 6 hours in cats.[1]Apart from being hepatobiliary in origin, increased bone isoenzyme may occur in young animals or those with lytic or neoplastic bone lesions. In dogs, ALKP increase is also seen with endogenous or exogenous corticosteroids and anti-epileptic drugs (AEDs), whereas GGT does not increase with bone activity and AEDs.

Liver Function

Liver failure can manifest as decreased synthetic capacity, decreased uptake and excretion of bile acids, and decreased conversion of ammonia to urea. For example, laboratory evidence of decreased synthetic capacity can occur when 70-75% of the hepatic functional mass is lost. Hepatic dysfunction may manifest as decreased albumin, glucose, cholesterol and BUN. Production of coagulation factors, fibrinogen, as well as coagulation inhibitors (e.g. protein C and anti-thrombin) are also affected.

Bile acids are synthesised from cholesterol in the liver and secreted in bile after conjugation. Assessing the liver's capacity to remove circulating bile acids gives an indication of liver function and perfusion. Its use in detection of hepatobiliary disease and acute liver damage in dogs, as well as portosystemic vascular anomalies (PSVA) in dogs and cats, has been described across various studies. [2,3,4] Understanding the limitations of an endogenous challenge help in the interpretation of unusual test results; e.g. higher pre-prandial concentrations. One should also be aware of test vulnerability factors including meal type and size, enteric disease and intestinal motility.[5] Lastly, one should also bear in mind the possible effects of concurrent administration of ursodeoxycholic acid, as well as the spurious increase in bile acids seen in Maltese terriers.[6,7] Fasting ammonia or the ammonia tolerance test serves as possible alternatives to detect hepatic dysfunction and PSVAs. It is less commonly used due to complexities in sample handling.

Protein C is a vitamin K-dependent protein, synthesised in the liver with a short plasma half-life. It serves as an indicator of portal blood flow. It may be used to differentiate PSVA from microvascular dysplasia (MVD), as both disorders may result in increased bile acids.[8] Low protein C activity (< 70%) is a characteristic finding in dogs with clinical signs of PSVA. In contrast, dogs with MVD typically have protein C values > 70%. Protein C deficiency, however, may also be seen in patients with vitamin K deficiency, sepsis, SIRS, and DIC.[9]

Approach
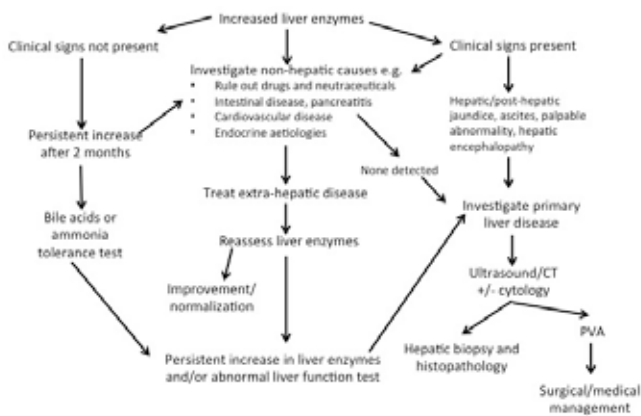
Figure 1: *Approach to Increased Liver Enzymes10*

The decision to investigate is difficult in asymptomatic animals. If no drug history (including nutraceuticals) is found, persistent increase of two-months duration or progression within this period is generally an indication for further testing. The speed at which investigation takes place may further be influenced by the patient's signalment, in conjunction with known breed predispositions.

Investigation is recommended when clinical signs are present, especially if the signs are specific to hepatic disease. Again, history, physical examination and signalment are all critical in establishing the cause of the increased enzymes. Based on clinical signs, extra-hepatic disease should be investigated if indicated. These include intestinal, respiratory and cardiovascular disease, which generally results in mild to moderate increase in transaminase activity if present. Endocrinopathies, e.g. hyperthyroidism in cats, hypothyroidism and hyperadrenocorticism in dogs should be considered in the presence of appropriate clinicopathological abnormalities. Investigation of primary hepatic disease is indicated if hepatic enzymes remain persistently increased despite addressing extra-hepatic disorders.

Investigation of primary liver disease may involve assessment of hepatic function, imaging and +/- histopathology. Ultrasound is a commonly used modality to image the liver, although scintigraphy and computed tomography are generally thought to be more sensitive for detection of PSVAs. However, histopathology is often required for definitive diagnosis, prognosis and to dictate ongoing management.

Cases

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WSAVA ONE HEALTH DISASTER MANAGEMENT

HOW TO DEVELOP DISASTER MANAGEMENT PLANS FOR HOSPITALS AND CLIENTS

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HOW TO DEVELOP DISASTER MANAGEMENT PLANS FOR HOSPITALS AND CLIENTS

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Introduction

Disasters can strike anywhere, at any time, and without warning. Recent events have highlighted how devastating disasters can be on communities, businesses and how local resources can become quickly overwhelmed. Additionally, climate change makes extreme weather events more likely than before with increased frequency and intensity. Albeit with this evidence people are still complacent about personal emergency preparedness which is then reflected equally inadequately in business.

The stark reality of disasters is that it is not a matter of if it will happen; it is a matter of when. The number of people affected by disasters and the costs associated with these events is increasing. Hence there is a requirement to have practice and personal emergency plans in place to mitigate the effects of such disasters, particularly to veterinary business. There are consultants who can help businesses develop emergency and business continuity plans but the aim of this paper is to give a basic outline of how veterinary businesses can prepare for disasters.

Emergency management plans

As veterinary professionals you have a responsibility to your family, clients, their animals, and your communities to be prepared. They will look to you for guidance therefore you have to be prepared to help. Plans are necessary for different scenarios well in advance of a disaster occurring. These scenarios include but not limited:

- Occurring during the working day when you, your staff and clients are at clinic
- Some staff may be out at lunch or on house calls
- Occurring out of business hours, everyone is away from the clinic with in house patients
- Slow onset
- Such as predicted strong winds, heavy rainfall
- Fast onset
- Such as an earthquake or volcanic eruption

Veterinary businesses need to work together with local communities to be incorporated into the disaster management framework. Clients and the community are vital to the survival of your business during the post-disaster economic period. If your community is prepared and resilient, your cliental will still be present.

Standards New Zealand states that "Business continuity management provides the availability of processes and resources in order to ensure the continued achievement of critical objectives". Hence a disaster management plan needs to incorporate all phases (mitigation, preparedness, response and recovery) instead of just being a response plan.

A disaster management plan is:

- about managing risk
- a powerful force for business sustainability and resilience
- provides for business success.

A risk and hazard assessment of your facilities, business and staff is the first step of developing a plan. When risks are known steps should be put in place to mitigate the risks and move onto preparedness, response and recovery. The disaster plan should cover three core elements (financial, logistics and operational) to ensure all components of the business are incorporated.

Financial

Recent work completed by the UN and the World Bank shows that, while specific cases may vary, for every dollar invested in minimising risk, about seven dollars will be saved in economic losses from disasters. Planning for every conceivable disaster is important, although not directly being affected by the event should equally be considered together with its potential negative impacts on business and staff. The true extent of these consequences is often unrecognised and commonly unaccounted for during development of business continuity plans. For example a business could suffer from severe disruption to their operations and financial hardship as a result of collateral effects such as continuity of supply or retraction of a traditional customer base.

A lack of understanding insurance policies particularly business interruption insurance and property damage has caught many businesses. Some business owners are unaware that insurance companies may not cover further damage to a property and equipment if it is considered secondary to the primary event. For example if you do not fix a damaged roof due to initial event (such as a wind storm), i.e. you fail to action covering the damaged roof when it is safe enough to do so and it rains 24 hours later the additional water damage may not be covered as it is not considered the primary event.

Clients are vital for your business to survive the post-disaster economic period. You must find a communication medium (newspaper, radio, newsletters, mail outs, welfare centres, medical centres, churches, cultural groups, fed farmers) that will provide contact for the vast majority of your client base to inform them of clinic plans and where you will be seeing patients until full functionality is restored.

Logistics

In your plan you need to address access to essential services (known as lifelines in disaster management) such as utilities power, water, sewage, gas and medical oxygen. Do you have a backup generator and do you know where to order drinking water or do you have a reserve rainwater tank that could supply you for up to five days? Is your data backed up regularly and stored off site. What is your accessibility like during a disaster? Many large businesses now store data off site at several places regionally and internationally. This gives them the ability to continue with their business if the premises are not accessible.

Businesses need to identify alternative premises where they could continue to run their practice. Start conversations with other practices in your area or you may have to look further afield if your community is severally affected. There are many examples where veterinary practice has shared premises after disasters. What arrangements can you make with fellow colleagues? If you have to evacuate your premises how are you going to transport your patients and where are you going to take them? If it is a slow onset event then you could contact owners to collect their pets or you could offer to shelter in place. If you do offer the latter you must ensure you can guarantee the safety of your patients. There are examples where veterinarians have kept the patients on site but failed to secure the building which has resulted in death of the patients.

Organisation (management & staff)

What roles will staff play during a response in your clinic? What training do they need to fulfil this role and when are you going to exercise a scenario to ensure your plan works? It is important to get everyone involved in writing the plan to ensure all staff understand what you are trying to achieve and they understand the importance of this process.

How are you going to contact your staff after an event? You need to know they are safe and they need to know you are safe. Prior to the event they should be aware of the triggers in your plan to initiate clean-up efforts and they will want to know their employment status post-disaster period. All staff members should be given contact phone tree indicating who to contact after an event via various means. The details should be updated regularly and staff given a copy to have on them at all times. A great way to do this is to make a laminate credit card size copy of the contact details so they can put in their wallet.

The veterinary clinic should have contact details of the local representative for the national veterinary association as they will no doubt have an animal welfare support role to fulfil during and after an event. They will be wanting to know your business functional status. This information can be passed on to the necessary agencies to assist with developing situational awareness and understand the needs of the community and your business. It is ideal to know who your contacts are before an event occurs to understand what their role is and what resources they have to offer.

Conclusion

The priority during any disaster should begin with you. If you are not safe, there is no way you will be of use to your whānau (Maori word for extended family), business and community. Everything needs to be kept in perspective when dealing with a disaster. Human life is addressed first as it should be. This does not rule out preparing for an animal emergency response or for it to occur at the same time as human emergency response.

Well prepared countries are not immune to the destructive impact of the forces of nature. Therefore, there is a need to plan for many different events. As a profession we must always maintain an inherent flexibility to rapidly respond to changing circumstances. Sharing knowledge and experience is an essential element of prevention and preparedness. A resilient community has the ability to return to normality faster than an ill prepared community. To be able to respond to a disaster you have to be prepared. To be prepared you have to have a plan. Now is the time to put a plan in place or revise your current plan.

Resources

American Veterinary Medical Association: Disaster preparedness for veterinarians https://www.avma.org/KB/Resources/Reference/disaster/Pages/default.aspx

Ministry for Primary Industries New Zealand: Animals in Emergencies

https://www.mpi.govt.nz/protection-and-response/animal-welfare/animals-in-emergencies/

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WSAVA VACCINATION GUIDELINES

CAT VACCINATION

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Global FELINE Vaccination Guidelines

Guidelines for feline vaccination are produced by the American Association of Feline Practitioners [1], the European Advisory Board on Cat Diseases [2] and the WSAVA Vaccination Guidelines Group (VGG) [3, 4]. The fundamental principle of these guidelines, as encapsulated by the VGG, is that '**We should aim to vaccinate every animal with core vaccines. Non-core vaccines should be given no more frequently than is deemed necessary.**'

The WSAVA guidelines suggest that we should aim to vaccinate MORE animals. This relates to the phenomenon of 'herd immunity'. Herd immunity suggests that, for some diseases, where a minimum percentage (for example 75%) of a herd of animals is vaccinated, it is difficult for an infectious disease outbreak to occur. The 'herd' for a small animal practitioner is the population of cats living within his or her practice area – and our aim should be to have as many of these animals vaccinated as possible, in order to reduce the chances of disease outbreak in the herd. In order to apply the principles of vaccination guidelines, it is firstly necessary to understand the definitions of 'core' and 'non-core' vaccines. CORE vaccines are those that all animals should receive to protect them against diseases of global significance or where legislation may dictate [i.e. canine rabies]. The use of NON-CORE vaccines is dictated by geographical location, lifestyle and exposure risk. Some vaccines are NOT RECOMMENDED because there is little scientific justification for their use.Core vaccines for the cat are those that protect against feline parvovirus (FPV), feline calicivirus (FCV) and feline herpesvirus-1 (FHV). In rabies-endemic countries, rabies vaccination is also considered core for cats, even if not dictated by legislation. Non-core vaccines are those for which use is dictated by geographical location, lifestyle and exposure risk. Non-core vaccines for the cat are those that protect against feline leukaemia virus (FeLV), feline immunodeficiency virus (FIV), **Chlamydia felis** and **Bordetella bronchiseptica**. Vaccines against feline

infectious peritonitis (FIP) are currently not recommended for use.

WSAVA guidelines provide generic advice to practitioners, but it is impossible to ensure that the guidelines are tailored to best fit the local situation in each of the 86 WSAVA member countries. The VGG encourages national associations to adapt and modify the guidelines for local use where appropriate. This process might involve altering the classification of a vaccine. For example, in the UK, FeLV vaccine is often considered core for the cat and administered routinely to kittens.

Core Vaccination of Kittens

Core vaccination of kittens (FPV, FCV, FHV) begins at 8-9 weeks of age, with a second vaccine given 3-4 weeks later and a third vaccine given at16 weeks of age or older. The 26 or 52 month fourth vaccine is also an integral part of the kitten programme. Increasing evidence suggests that maternally-derived antibody (MDA) may persist for up to 20 weeks in some kittens [5], which underpins the current advice that the third kitten vaccine be given at 16 weeks of age or older. Where rabies is endemic, kittens should receive 1 dose of vaccine at 12 weeks of age, but the VGG suggests that in a high-risk situation (i.e. in an endemic area with recognized clinical cases), a second dose of vaccine may be given 2-4 weeks later.

Core Vaccination of Adult Cats

Most FPV vaccines now carry a licensed duration of immunity (DOI) of 3 years; however, most vaccines against FCV, FHV and non-core products all have a 1-year DOI. A product with licensed 3-year DOI also against FCV and FHV has recently become available. Rabies vaccines (including one non-adjuvanted product) also have a 3-year DOI in many countries. Selecting products with extended DOI allows reduced frequency of administration of that component in a fashion consistent with the legal 'summary of product characteristics' (SPC). Guidelines may still advise triennial revaccination with products carrying a 1-year licensed DOI. For the cat, there are field serological data that show persistent seropositivity for 4 or more years post core MLV vaccination [6] and one experimental challenge study that shows immunity for a minimum period of 7.5 years following vaccination of kittens with killed adjuvanted trivalent vaccine [7]. WSAVA guidelines therefore recommend that adult cats receive MLV core FPV vaccine no more frequently than every 3 years. The frequency of administration of MLV core FHV and FCV vaccines depends on individual risk assessment of the lifestyle of the cat. A low-risk cat (e.g. a solitary, indoor only cat that does not visit boarding catteries) should only require triennial booster vaccination. In contrast, a high-risk cat (e.g. a cat in an indoor-outdoor or multicat household or one that regularly visits boarding catteries)

may benefit from annual FHV/FCV revaccination. Using product ranges that split-out FPV from the respiratory components, such a protocol is entirely feasible. The study supporting the licence of the 3-year FHV/FCV vaccine clearly shows protection from clinical disease (but not virus shedding) in vaccinated cats challenged after 3 years [8]. Rabies vaccination of adult cats may now be performed triennially using products with an appropriate licensed duration of immunity (DOI) of 3 years.

In most situations, MLV ('infectious') core vaccines are preferred over killed ('non-infectious') vaccines. The exceptions to this rule would be: (1) the rare requirement to vaccinate a pregnant queen, (2) vaccination of cats with known retrovirus infection, (3) vaccination of cats in multicat households where there is no circulating respiratory virus, and (4) for rabies vaccination where 3-year DOI is required.

Non-core Vaccination

Non-core vaccines should be selected for the individual cat based on assessment of that particular animal's risk of exposure to the disease and assessment of the benefits of vaccination to that pet versus the risk of adverse reaction. Decision making for non-core vaccines would be facilitated by having available good quality data and disease distribution maps related to small animal infectious diseases. Unfortunately, with the exception of rabies in the USA and Europe, such distribution maps are not widely available. Some national schemes have been developed by industry or academic groups which allow practitioners to input cases of particular infectious diseases into a database that presents the information as disease distribution maps. Additionally, consideration must be given to the vaccine requirements of the individual animal, based on assessment of their lifestyle (e.g. indoor versus outdoor, travel and boarding frequency and location, solitary or multicat household). Vaccination is now an example of 'individualised medicine' and is no longer as simple as having a practice 'vaccination protocol'.

For example, where non-core FeLV vaccination is selected for kittens, an initial dose is given at 8 weeks of age, with a second 3-4 weeks later, followed by a 12 month booster. It is more important to vaccinate kittens against FeLV than it is adult cats, as there is development of some naturally acquired immunity in adult animals. The VGG recommends that adult cats are revaccinated against FeLV no more frequently than every 2 or 3 years, depending on risk.

Minimize Adjuvanted Vaccines

Although it is now recognized that the feline injection site sarcoma (FISS) may be associated with a wide range of injectable or topical products it is clear that vaccines, and particularly adjuvanted FeLV, FIV and rabies vaccines, are one such risk factor in the transformation of local chronic inflammation to neoplasia. A number of strategies have been proposed to minimise the surgical consequences of any FISS that might develop in a cat. The WSAVA has suggested vaccination into the skin of the lateral abdomen, while the AAFP continues to advise vaccination into the distal hindlimb for rabies and FeLV and the distal forelimb for other vaccines. A recent study has shown the efficacy of vaccination for FPV and rabies when vaccine is administered into the distal tail, although there remain some concerns about this procedure [9]. Whichever anatomical site is chosen, basic principles should be to avoid the scruff of the neck, to rotate sites of injection and to record these sites in the medical record of the animal.

The Annual Health Check

All aspects of vaccination should fall under an annual health check programme that reduces the emphasis on vaccination as a reason for visiting the practice and considers holistically the overall health and wellbeing of the pet. A discussion about which vaccines (or serological tests) might be offered in any one year is just one part of the annual health check. The importance of vaccination can be reinforced by using the VGG fact sheets. Vaccination (or serology) should be appropriately invoiced so emphasis is placed on the professional consultation. The use of in-house serological testing can reliably inform the need for FPV revaccination as there is a strong correlation between seropositivity and protection [10]. Any seropositive adult cat does not require revaccination against FPV. This correlation is not as strong for FCV and FHV, as seronegative cats might still be protected by cellular or mucosal immunity.

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GASTROENTEROLOGY / LIVER

CHRONIC ENTEROPATHIES IN CATS: IS IT IBD OR LYMPHOMA?

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Clinical presentation:

Middle-aged to older cats with vomiting, anorexia, diarrhea and weight loss, and general malaise are the most commonly observed clinical signs. However, some cats may have a normal to increased appetite. Unlike in dogs, diarrhea is only occasionally a presenting complaint. Many cats will be presented for hyporexia/anorexia, lethargy and weight loss. These non-specific signs are often waxing and waning, and the owners may seek veterinary attention only late in the course of disease. Abnormal findings on physical exam include loss of body condition, dehydration, possible palpation of thickened bowel loops and/or abdominal pain.

Diagnostic approach:

Because the clinical signs may be very non-specific, the first step is to rule out diseases originating outside GI tract that may present with a similar clinical picture. These include conditions such as hyperthyroidism, chronic kidney disease, liver disease and pancreatitis. A minimal database is recommended that consists of CBC, chemistry profile, serum thyroxin concentration and abdominal imaging (radiographs, ultrasound). Once the GI tract has been confirmed as the origin of the disease, several steps should be considered in view of the most common causes of chronic intestinal diseases in cats. Parasitic diseases should be ruled out with fecal analysis or appropriate empirical treatment with broad-spectrum anthelminthics. Diagnosis of giardiasis using direct fecal smears (trophozoites) or zinc sulfate flotation (cysts) may be difficult. Analysis of several fecal samples may be required due to the erratic shedding of cysts. Several commercially available immunoassays detect Giardia cyst antigen in feces and are helpful diagnostic tools.

Diagnostic imaging:

In chronic enteropathies, the most common abnormality of the small intestine is thickening of the tunica muscularis. It is seen equally frequently in inflammatory bowel disease (IBD) and alimentary lymphoma, and cannot be used to differentiate these 2 diseases. Norsworthy et al. (JAVMA, 2015) reported that jejunal wall thickness >2.8mm at 2 sites or >3.0mm at one site correlates with clinically relevant histopathological disease.

Empirical treatment trial:

Cats with mild to moderately severe clinical signs and maintained appetite may respond to a diet change using highly digestible, novel protein or hydrolyzed peptide diets within 2 weeks. In cats that do not respond to a dietary approach, antimicrobials have been successful in some instances, presumably to correct intestinal dysbiosis (e.g. metronidazole 10-15 mg/kg PO q12 h or tylosin 10-15 mg/kg PO q12h). Improvement is usually noticeable within a few days. In cats that respond to treatment, there are no reports about the optimal duration of antimicrobial treatment, and the author usually attempts to discontinue treatment after 3-4 weeks.

Collection and evaluation of intestinal biopsies:

In cats that do not respond or only partially respond to dietary or antimicrobial treatment, it is often necessary to obtain intestinal biopsies for histopathological evaluation to differentiate between enteropathy-associated T-cell lymphoma (EATL type II) and IBD. Endoscopic mucosal biopsies and surgical full thickness biopsies are both appropriate, and each sampling method has its strengths and weaknesses. In difficult cases when the pathologist cannot easily differentiate between inflammation and neoplasia, immunohistochemistry for T and B-cell markers and PCR assay for antigen receptor rearrangement (PARR) have been used successfully to rule in or out the possibility of enteropathy-associated T-cell lymphoma (EATL) type II. Unfortunately, the sensitivity of PARR is 78% for T cell lymphoma and 50% and for B cell lymphoma, and false negative results are therefore possible.

Inflammation of multiple digestive organs:

IBD, neutrophilic or lymphocytic cholangitis and chronic pancreatitis or any combination of these diseases have been reported to occur concurrently in middle-age to older cats. Simultaneous occurrence of all 3 diseases has been described as "triaditis". This multi-organ inflammation may be a consequence of the unique pancreatico-biliary anatomy of the cat with fusion of pancreatic and common bile ducts prior to the duodenal papilla. It is suspected that bacteria may penetrate and ascend the pancreatic and bile ducts and permit extension of inflammation to these organs. Diagnosis is suspected based on finding changes suggestive of IBD (see above), increased serum liver enzymes and possibly bilirubin, and ultrasound abnormalities in the pancreas and/or increase serum fPL or DGGL lipase. Confirmation of diagnosis relies on histopathologic analysis of intestinal, liver and pancreatic biopsies. Treatment is based on prednisolone at immune-suppressive (IBD) or anti-inflammatory (chronic pancreatitis) doses, and possible broad spectrum antibiotics in case of neutrophilic cholangitis (e.g. amoxicillin and clavulanic acid 20 mg/kg q12h).

Management and prognosis:

- IBD: If dietary or antibiotic trials fail, or in severely affected cats, immune suppressive therapy is the mainstay of IBD treatment. It is best initiated when histological evidence of intestinal mucosal infiltration is available, but could also be the final option of the empirical treatment sequence started with dietary trial and antimicrobials. Prednisolone is generally administered at a dose of 2 mg/kg PO (once daily or divided into two daily doses) for 2 weeks. Once the clinical signs have been controlled for 2 weeks or longer, the dose is reduced by one-quarter to one-half every 2 weeks. The final goal is to maintain the cat on the lowest effective dose, or even to consider discontinuation of steroid treatment if feasible. Refractory cases are usually treated with chlorambucil. Chlorambucil, an alkylating agent, is generally used in combination with prednisolone at a dosage of 2 mg PO per cat every other day (in cats $\geq$ 4 kg body weight) or every 3 days (in cats < 4 kg body weight) and then tapered to the lowest effective dose. Alternately, a pulse treatment with administration of chlorambucil at 20 mg/m^2 body surface area (BSA) q14 days can also be used (for most cats the BSA is between 0.25 and 0.3 m^2). A CBC should be checked 2 weeks after initiation of treatment for signs of myelosuppression. In one study, 80% of 7 cats with IBD treated with diet and prednisolone had a positive response to treatment. Cats with severe histological lesions or eosinophilic inflammation may be more difficult to manage. In addition, failure to respond to treatment may indicate refractory IBD or lymphoma. Owners must understand that feline IBD is a disease that can be managed, but not cured.

- Treatment of small cell alimentary lymphoma (EATL type II) with prednisolone and chlorambucil is associated with a good rate of complete remissions, and survival times between 16 and 30 months depending on the study. Recommended doses are identical to those listed above for the treatment of IBD. When a cat comes out of remission after having initially responded to prednisolone and chlorambucil, it is advised to reinitiate the treatment at the full doses for both drugs. If the cat does not come into remission during induction, alternative protocols include lomustine (CCNU) and prednisolone, cyclophosphamide and prednisolone, COP (cyclophosphamide, vincristine or vinblastine and pred). However, it is recommended to consult with an oncologist or refer the cat prior to starting these more intensive rescue treatments. Large cell lymphomas are associated with a much less favorable response and survival (a few months). Surgical removal may be a pre-requisite in the presence of intestinal masses obstructing transit. Generally, protocols such as COP or CHOP (COP + doxorubicin) are initiated. Consultation with or referral to an oncologist is advised.

- It has been demonstrated that cobalamin (vitamin B12) deficiency may be a consequence of feline gastrointestinal disease due to decreased absorption in the ileum with IBD or EATL type II. Hypocobalaminemia is easily be confirmed by evaluation of serum cobalamin concentration. B12 deficient cats may experience a delayed recovery, or treatment failure after immune suppressive therapy. Cobalamin may be administered SC at 250 m g SC per cat (see http://vetmed.tamu.edu/gilab for full treatment protocol) or orally (0.25 mg/cat PO q24h).

Further reading:

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SVA DERMATOLOGY (SIMULTANEOUS TRANSLATION INTO MANDARIN CHINESE)

DIAGNOSIS AND MANAGEMENT OF COMMON IMMUNE-MEDIATED SKIN DISEASES

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Introduction

In this presentation, we will cover two of the most common autoimmune skin diseases affecting dogs and cats: Pemphigus foliaceus and discoid lupus erythematosus.

1. Pemphigus foliaceus

Canine Pemphigus encompasses four variants; pemphigus foliaceus, pemphigus vulgaris, pemphigus vegetans and paraneoplastic pemphigus. Pemphigus foliaceus is the most common variant and also the most common autoimmune skin disease affecting dogs and cats.

Pathomechanism:

Anti-keratinocyte antibodies induce loss of cohesion (acantholysis) between keratinocytes resulting in formation of pustules that rapidly progress to erosions, crusts and alopecia. In the dog, the major autoantigen is desmocollin 1. Pemphigus foliaceus can occur spontaneously or secondary to triggers such as drugs, vaccine or ultraviolet light.

Signalment:

- Chow Chows and Akitas are predisposed
- Typically middle age
- No sex prediction for dogs; possibly more common in female cats

Clinical signs:

The characteristic lesion in both dogs and cats is a pustule, which evolve rapidly into erosions, crusts and alopecia. Lesions often develop in waves and may wax and wane. Depigmentation affecting nasal planum and footpads are common.

Distribution:

Dogs

The face is the most common initial site of lesions. The disease progresses to affect the dorsal muzzle, nasal planum, peri-ocular areas, ears, trunk and footpads. Lesions are typically bilateral and symmetrical but can be localised (e.g. face or clawfolds). Affected dogs may show pruritus and systemic symptoms such as anorexia and lethargy. Weight loss may be present in severe cases.

Cats

Lesions most commonly affect the face, ears and feet and are typically bilateral and symmetrical. Claw folds may reveal suppurative exudation. Peri-mammary crusting is considered highly suspicious for Pemphigus foliaceus.

Clinical differentiation between Pemphigus foliaceus and bacterial pyoderma

	Pemphigus foliaceus	Bacterial pyoderma
Lesion symmetry	Yes	No
Distribution	Mainly head/face and feet	Mainly ventrum and trunk
Nasal planum involvement	Yes	No
Footpad involvement	Yes	No
Pustules	Large, panfollicular, flaccid and confluent Usually same stage of development (develop in waves)	Single follicular and turgid Various stages of development
Irregular, polycyclic or annular pustules	Yes	No
Epidermal collarettes	Few	Many
Palisading crusts	Yes	No
Paronychia and peri-mammary crusting	In cats	Uncommon

Diagnostic approach:

Cytology

An easy test to increase suspicion for pemphigus foliaceus is to perform cytological evaluation of an intact pustule. The typical findings are "rafts" of acantholytic keratinocytes with non-degenerated neutrophils with or without eosinophils.

The presence of acantholytic keratinocytes does not confirm the dog/cat has pemphigus foliaceus. Acantholysis can also occur secondary to other diseases such as pustular dermatophytosis (especially due to **Trichophyton mentagrophytes**) and bacterial diseases (i.e. bullous impetigo and exfoliative superficial pyoderma).

Histopathology

Biopsies of intact pustules and/or crusts will reveal subcorneal pustules with acantholytic keratinocytes. Special stains (e.g. Gram stain for bacteria and PAS for fungi) can rule out infectious causes for the acantholysis. Tissue cultures should be performed to definitely rule out infections.

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Immunopathology

Direct and indirect immunofluorescence can detect antikeratinocyte autoantibodies and circulating pemphigus autoantibodies respectively.

Treatment options:

Immunosuppressive therapy is used to treat Pemphigus foliaceus. In dogs, the treatment of choice is oral glucocorticoid with or without concurrent steroid sparing therapies such as azathioprine, chlorambucil, ciclosporin and mycophenolate mofetil.

In cats, glucocorticoid (most commonly prednisolone or triamcinolone) monotherapy usually produces a good response. Steroid sparing therapies include ciclosporin or chlorambucil.

Topical glucocorticoids (moderate to high potency e.g. 0.1% mometasone) are also very effective at treating focal lesions.

Veterinarians should also try to identify any potential triggers such as drugs, preventatives and ultraviolet light. If a dog/cat relapses, it is important to rule out development of bacterial pyoderma, dermatophytosis or demodicosis. These diseases can be managed without the need to increase the level of immunosuppression.

Monitoring:

Regular blood and urine monitoring is recommended to identify potential side effects to immunosuppressive therapy.

Outcome

The outcomes for both canine and feline pemphigus foliaceus are generally good. The main reasons for euthanasia are lack of response to treatments and unacceptable side effects to treatments. These can be managed by client education, and regular blood and urine monitoring.

2. Cutaneous lupus erythematosus (CLE) in dogs

Current recognised forms of cutaneous lupus erythematosus in dogs are discoid lupus erythematosus (localised and generalised), vesicular cutaneous lupus erythematosus, exfoliative cutaneous lupus erythematosus and mucocutaneous lupus erythematosus. Localised facial-predominant discoid lupus erythematosus is the most common form seen in dogs.

Localised (facial-predominant) discoid lupus erythematosus (FDLE)

Signalment:
- German Shepherds and their crosses make up about 31% of cases.
- Median age of onset is 7 years (range from 1 to 12 years)
- Female to male ratio is 0.7.

Clinical signs:

The early signs include erythema, depigmentation and scaling most commonly affecting the nasal planum. These progress to erosions and ulcerations, atrophy and loss of the normal cobblestone appearance on the nasal planum. Less commonly, lesions may also affect the skin around the dorsal muzzle, lips, eyes and pinnae. Pruritus is variable and more likely with secondary bacterial infections. The main differential diagnoses include mucocutaneous pyoderma (MCP), uveodermatologic syndrome and localised epitheliotropic lymphoma.

Diagnostic approach:

Depigmentation of the nasal planum without loss of the cobblestone surface architecture may be normal in certain breeds e.g. nasal hypopigmentation in Golden and Labrador retrievers. Conversely, a thorough and immediate examination of the eyes for uveitis should be performed in breeds predisposed to uveo-dermatologic syndrome e.g. Akitas and Alaskan Malamutes.

Cytology:

MCP and DLE can be difficult to differentiate clinically and on histopathology. Furthermore, most cases of DLE are secondarily infected. It is important to perform cytology and treat any secondary infections before proceeding to further diagnostics.

Histopathology:

DLE is characterised by a lichenoid cell rich, lymphocytic interface dermatitis with basal keratinocyte vacuolar degeneration apoptosis, loss of basal cells and basement membrane thickening. The interface reaction may be mild in FDLE but is usually well developed in generalised DLE.

Treatment options

Systemic and/or topical immunosuppressive or immunomodulatory therapies can be used depending on the severity of disease.

For severe cases, oral prednisolone is recommended to achieve remission. The prednisolone doses can then be reduced or potentially withdrawn as less potent immunomodulatory therapies are added. These include combination of tetracycline/doxycycline and niacinamide, fish oils, Vitamin E.

Topical therapies include topical cortisone (starting with higher potency e.g. 0.1% mometasone then changing to less potent e.g. 1% hydrocortisone) or 0.1% tacrolimus.

Distraction techniques should be employed to avoid dog rubbing/licking off topical therapies.

Avoidance of ultraviolet light is important, especially during initial treatment phase. Sunscreen should be applied to non-pigmented areas and the dog should preferably be kept away from sun exposure.

Tattooing of the nasal planum is not recommended. Apart from potential topical chemical reaction to the ink, this method does not protect against actinic damage because the tattoo ink is injected into the dermis but actinic damage occurs mostly on the epidermis.

Generalised discoid lupus erythematosus

Signalment:

In the ten dogs reported, the breeds include two Chinese crested dogs, two Labrador retrievers, and one each of miniature pinscher, Leonberger, Shih Tzu and toy poodle.

The median age of onset is 9 years (range from 5 to 12 years). Both female and male are equally affected.

Clinical signs:

The skin lesions include generalised or multifocal plaques, scaling, follicular plugging and alopecia affecting mainly the neck, dorsum and lateral thorax. The plaques can progress to ulcers with subsequent healing resulting in central atrophic or hypertrophic scars. Both depigmentation and hyperpigmentation may be observed. Four of the ten dogs had plaques around mucocutaneous regions (mainly genitalia). A pattern of reticulated hyperpigmentation affecting the ventral abdomen and lateral thorax was seen in two of these dogs. No systemic signs were reported. Pruritus was reported in four dogs and pain in three. The main differential diagnoses are ischemic dermatopathies and hyperkeratotic erythema multiforme ("Old dog" EM).

Treatment options

A wide range of treatments has been reported to be successful in the few cases including ciclosporin (with initial short course of glucocorticoids), hydroxychloroquine, topical tacrolimus and tetracycline/niacinamide. However, relapses are common after medications were tapered.

Outcome

The prognosis for DLE is good. Progression of FDLE and GDLE to systemic lupus erythematosus (SLE) has not been reported. There was a single case of a DLE variant to "clinical" SLE reported in one dog.

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ANESTHESIOLOGY

HOW TO REDUCE ANESTHESIA-RELATED MORBIDITY AND MORTALITY: EVIDENCE AND EXPERIENCE

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HOW TO REDUCE ANESTHESIA RELATED MORBIDITY AND MORTALITY: EVIDENCE AND EXPERIENCE

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Objectives: Review anesthesia related mortality and morbidity facts with a special focus on how to lower the odds of a patient dying under general anesthesia.

Many studies have elucidated the anesthetic related mortality and morbidity. The most important studies ever published for dogs and cats describe the following mortality rate:

Dogs:
- Overall risk (ASA 1,2,3,4,5, combined)= 0.17% or 1 in 601
- Health dogs (ASA 1 and 2)= 0.05% or 1 in 1849
- Sick dogs (ASA 4 and 5)= 1.33% or 1 in 7

Cats:
- Overall risk (ASA 1,2,3,4,5, combined)= 0.24% or 1 in 419
- Health cats (ASA 1 and 2)= 0.11% or 1 in 895
- Sick cats (ASA 4 and 5)= 1.4% or 1 in 7

Humans:
- Overall risk = 0.001 to 0.0025% = 1 every 100.000 - 250.000The explanation for the major difference between human and veterinary anesthesia-related complication rates are simple. Pre-op evaluation and vital signs monitoring in human medicine is significantly better. The more you know about your patient before the beginning of the anesthetic procedure, the more prepared you will be for a possible complication. With modern monitoring, you can identify life-threatening complications earlier and start target treatment sooner.

The next paragraphs will shine some light on factors that have been recognized as important factors that may INCREASE the odds of anesthetic-related death. In another words, those are the factors that should be avoided:

- Increased physical status increases the chances of having a complication under general anesthesia.
- The more seek the patient is the more complicated the anesthesia management will be and obviously, the mortality rate of those seek patients is expected to be higher

- The American Society of Anesthesiologists (ASA) published a physical status classification that helps us to guide ourselves:

Category	Physical Status	Possible examples
1	Normal healthy patient	Elective procedures, ovariohysterectomy, castration, allergy test
2	Patients with mild systemic disease	Skin tumor removal, repair of fractures or hernias, cryptochirdectomy, localized infections, compensated cardiac disease, obesity, mild dehydration
3	Patient with severe systemic disease	Fever, anemia, dehydration, cachexia, moderate hypovolemia, kidney disease, C-section
4	Patient with severe systemic disease that is a constant threat to life	Uremia, toxemia, severe dehydration and hypoxemia, anemia, cardiac instability, emaciation, high fever, GDV, azotemia, caval syndrome
5	Moribund patient not expected to survive 1 day with or without operation	Extreme shock and dehydration, terminal malignancy or infection, or severe trauma, sepsis
E	Emergency	Any category can receive an emergency status

This classification system is considered subjective. Different anesthesiologists may classify the same patient differently. But that is all right. The ASA classification is not designed to be precise but is a tool used to classify a patient based on its physical status. It is used to help the anesthesiologist to see if there is a possibility to reduce the risk of anesthesia by improving the ASA status of the patient prior to be beginning of the anesthesia. There is evidence that survival rates deteriorate, as the ASA status gets worse.

Factors to consider when assigning ASA status:
- Cardiac reserve – is it compromised? Will anesthesia drugs make it worse? Can it be better with drugs or fluids?
- Pulmonary – what is the drug effect on the pulmonary system? What type of ventilation/oxygenation is required prior to anesthesia induction? Will patient position on the table influence the ventilation status?
- Renal – is dehydration, azotemia or uremia present? Will drugs be eliminated by renal system?
- Neurologic – any signs of CNS depression, behavior change, seizures, elevated ICP, anisocoria, nystagmus?
- Hepatic - is the hepatic function compromised? Liver enzymes, blood glucose, albumin, coagulation disorders?
- Endocrine – any clinical signs of diabetes, thyroid disease, Cushing's, Addison's diseases?
- Hematologic – is anemia present?
- Physical status is independent of the surgical procedure

Urgency of the procedure
- Emergency procedures are associated with higher risks of anesthesia related mortality rate.

Age
- Age is not a disease but is considered a co-factor associated with anesthesia-related mortality rate. Both neonatal and geriatric patients are considered high risk patients for anesthesia.

Intended duration of the procedure

- The longer the procedure is, the higher the odds of anesthesia-related complications are.

Injectable versus inhalant

- Studies are showing that when inhalant anesthetics are used in cats the chances of having a complication increases. However, the authors explain that other co-factors are associated with this observation. For instance, the use of inhalant anesthetics without any co-morbidity is not associated with higher mortally rates. Usually inhalants are used for more complicated and longer procedures and, at the same time, maintenance of anesthesia with injectable anesthetics are often used during short, uncomplicated procedures. Therefore, the length and complexity of the procedure are more important than the anesthetic choice itself.

Endotracheal intubation in cats

- Endotracheal intubation in cats is associated with higher mortality rates. Even though cats are more difficult to intubate than dogs and there are higher chances of tracheal tear in any very small animal, these is not the explanations. Actually it is explained similarly as the previous factor. In the majority of the available studies in cats, endotracheal intubation was associated with longer and more complicated procedures. For example, for a simple castration, inhalants are not necessary usually used.

Fluid administration in cats

- Fluid administration in cats is associated with higher mortality rates. Same as both previous items. Cats receive fluids only when they are undergoing longer and/or more complicated procedures. Which are associated with higher mortality rate.

Obesity

- Obese patients have higher odds of suffering a complication under anesthesia than slim patients. While under anesthesia, obese patients are more likely to suffer from respiratory depression, overdoses, thermoregulatory issues and prolonged recovery.

Brachycephalic

- Brachycephalic patients have higher chances of dying after anesthesia. Usually the brachycephalic patients have stenotic nares, prolonged soft palate, inverted sacules and stenotic trachea. That leads to possible respiratory obstruction and arrest.

Therefore avoiding those previous mentioned factors will help you to decrease the anesthetic related mortality rates in your clinic.

The next paragraphs will then review the factors that have been recognized as important factors to DECREASE the odds of anesthetic-related death. In another words, those are the factors that should be promoted:

Equipment check with protocol and checklist

- Many anesthesia-related complications are related to equipment failure or failures to proper test the equipment. Check lists are available to remind the personnel of all important steps for the safety of the patient been anesthetized.
- Documentation of equipment check also helps to reduce the equipment related anesthetic complication. Knowing exactly when and what happened to the equipment helps to identify reoccurring problems that need to be corrected.

Directly available anesthesiologist + full time nurse

- The availability of well-trained personnel helps to decrease mortality rates when the team is able to treat complications earlier even before they become dangerous to the patient.

No change of anesthetics during the procedure

- When changing personnel during a procedure make sure the information regarding the patient is transferred or possible mistakes can happen. Common examples are: administration of incompatible drugs, implementation of treatments that did not work previously, over dose of fluids and other drugs. Well-trained teams uses standard transfer sheets to help team members to remember the important factors about the patient.

Two persons available during emergency

- The evidence shows that multiple team members should manage CPR events in order to improve survival rates.

Postoperative pain management

- The large majority of anesthesia related complications occur during the recovery of anesthesia. Providing post operative pain management helps to reduce the complications rate.

Pulsoximeter

- Early recognition of complications is the key to improve survival outcomes. The pulsoximeter is usually the first parameter that displaying errors associated with cardio-respiratory complications.

Monitoring

- Most of the anesthesia related mortality and morbidity studies show that monitoring saves lives. Again early recognition and early treatment for anesthesia related complication is the key to decrease the odds of having a complication under general anesthesia

References are available upon request

43RD WORLD SMALL ANIMAL VETERINARY ASSOCIATION CONGRESS
AND 9TH FASAVA CONGRESS

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SVA INTEGRATIVE MEDICINE

APPLICATION OF BLADDER MERIDIAN IN CLINICAL CASES

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APPLICATION OF BLADDER MERIDIAN IN CLINICAL CASES

Introduction

The bladder meridian is the longest channel in the body.

Objective

The objective is to evaluate the feasibility of using acupuncture points along the bladder meridian to treat illnesses of internal organs, neck, back, lumbar and legs.

Methods

Acupuncture points were chosen based on the symptoms in each case and performed twice a week for two months.

Results/Conclusion

Most of the cases have shown that performing acupuncture along the bladder meridian points can be both safe and effective in treating physical and psychological illnesses.

WSV18-0318

ORTHOPEDIC SURGERY (SIMULTANEOUS TRANSLATION INTO MANDARIN CHINESE)

TOP TIPS FOR TREATING LONG BONE FRACTURES IN GROWING ANIMALS: DECISION MAKING AND TREATMENT

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Top Tips for Treating Long Bones Fractures in Growing Animals: Decision Making and Treatment

Alessandro Piras

DVM - Specialist in Veterinary Surgery

Dogs reach skeletal maturity between 5 to 18 months depending on the breed. During this period, all bones exhibit a two-phase growth cycle, an initial rapid phase (20 wk) followed by a substantially slower growth to maturation (48 wk). During the initial part of this biphasic developmental process, both structural and material properties of the immature bone are very different from those of adult bone. Major characteristics are a lower strength and stiffness, lower yield stress and elastic modulus. If we also consider that the diaphyseal cortices in the young dogs are considerably thinner than in the adult, is intuitive that immature canine bone is potentially susceptible to implant failure due to screw pull out

Diaphyseal fractures in immature dogs are usually simple low energy fractures, often incomplete and can be associated with intact paired bones.

Greenstick fractures of the cortex are very common; they are incomplete fractures where only one side of the cortex is fractured while the opposite cortex is bent due to the elasticity and plasticity of the juvenile bone. The intact cortex is along the compression aspect of the fracture. The thick periosteum of immature animals can act as a restraint and stabilizer of the fracture preventing full displacement of the segments. The periosteum also aids in focal hematoma formation, subsequent callus formation and fracture healing. These fractures tend to heal very rapidly due to the their strong and very active periosteum.

Clinical examination

The puppy may be presented with a history of non-weight bearing or partial weight bearing lameness. The affected area can show various degrees of deformity, swelling and local pain. Palpable crepitius is usually absent. Careful physical and radiographic examination (Orthogonal X-ray views of the affected and controlateral limb) is necessary to complete the diagnosis.

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Treatment Options

External coaptation

In case of minimal deformity and minimal or no displacement, treatment consist of a combination of some form of external immobilization, rest and exercise restriction by cage confinement for 3 to 5 weeks.

Coaptation after traction and reduction can yield to good results for suitable diaphyseal fractures.

Cast or splinted bandages require a diligent management to decrease the potential for complications. Depending on the stage and speed of growth of the patient, the cast or splint will require frequent checks and revisions, up to once a week in a very young animal.

Unfortunately, the indications for external coaptation are limited to fractures below the elbow and stifle.

Inappropriate application and incorrect management of external coaptation can lead to a very high rate of complications.

Complete immobilization of the knee in young dogs can result in stiffening of the joint secondary to adhesion formation and quadriceps contracture.

Fracture disease is a syndrome characterized by joint stiffness or laxity, periarticular fibrosis, degeneration of the joint cartilage, osteopenia and muscle atrophy.

Other adverse effects of improperly placed limb splintage are valgus and rotational deformity. Immobilization of the stifle joint should be avoided, particularly in cases of distal femoral fractures to avoid the risk of devastating complications like quadriceps contracture and genu recurvatum. Prolonged immobilization of the hind limb in a non-weight bearing position causes coxa valga and increased anteversion. Unfortunately, the last two conditions are not reversible.

Coaptation of the antebrachiocarpal joint typically causes palmar carpal ligament laxity and consequent carpal hyperextension. This problem is commonly seen in large breeds and usually resolves spontaneously with controlled exercise. The application of any form of supporting bandages, in the attempt of correcting the hyperextension are inappropriate and should be avoided.

Frequent radiographic monitoring of the healing process is advisable. As soon as radiographic signs of clinical union are visible, coaptation should be removed.

Surgical Options

Early recognition of fracture is essential for a successful treatment of this injuries. Delayed repair will necessitate reduction manoeuvres that can damage the blood supply and disrupt the early callus. The goal of the repair of diaphyseal fractures is normal alignment, whereas anatomic apposition is not a priority because of the potential for compensatory remodelling.

Although fractures in immature skeleton can be treated for the most part as fractures in adults, it is mandatory to bear in mind the associated effects on further growth. Intramedullary pins, cross pins, Rush pins, plates, screws or external skeletal fixation, should be applied as to not interfere or restrict the normal growth at the physes.

Intramedullary Pin (IM pins)

The use of intramedullary pins is generally limited to femoral, humeral and tibia fractures.

IM nailing in immature animals, is favored by the high proportion of cancellous bone present in the medullary cavity. IM pins should be smooth and relatively smaller in diameter than those used in adults. They should not occupy more than 25% of the physeal cross sectional area and should be inserted perpendicular to the physis to not disturb the growth potential.

Classic IM pinning of the femur through the intertrochanteric fossa has been associated with serious alterations as malformations of the femoral head and neck ,coxa valga, hyper anteversion and coxo-femoral subluxation.

External Fixator (ESF)

The principles of application of external skeletal fixation in immature animals follows the same general principles as in the adults. However, the intrinsic stability and rapid healing of the of the fractures of immature animals, favour the use of fixators with a low stiffness configuration.

The thin and relatively soft cortices of immature animals, compromise the bone-implant interface for the fixation pins. Fortunately, this do not generally represent a concern as clinical union usually occurs before the ESF failure. Major recommendation in the insertion of the fixator pins are: never bridge a physis, avoid thermal necrosis during insertion, avoid insertion into fissures along the bone. For mechanical and biological reasons, the use of external fixation is poorly suited for the treatment of femoral shaft fractures. Due the anatomical characteristics of the region, the external fixator frame will result in a position, far from the neutral axis of the femur. this remote position will accentuate the bending stresses at the pin bone interface. The biological consideration is that the transfixion of the muscle groups of the lateral aspect of the tight generate pain, decrease the range of motion and can result in quadriceps contracture.

Plates

Although fractures of humerus, radius and ulna, and tibia can be successfully treated either with external or internal fixation, plate osteosynthesis remains the treatment of choice for femoral diaphyseal fractures in juvenile dogs, particularly in large and athletic breeds.

In spite of the strict adherence to the classic AO principles, catastrophic implant failures due screw pull out during the early growth phase has been a commonly reported complication.

The critical evaluation of these failures contributed to the evolution of internal fixation of fractures with a change of emphasis from mechanical to biological priorities. A more flexible fixation encourages the formation of callus while less precise indirect reduction and minimally invasive techniques reduce the operative trauma.

A biological internal fixation avoids the need for precise reduction, especially of the intermediate fragments, and takes advantage of indirect reduction, it also involves the use of long-span bridging plates (locking or non) and fewer screws for fixation in order to achieve a "flexible fixation."

The two techniques that suite this requisite are the Elastic Plate Osteosynthesis (EPO) and Plate and Rod Osteosynthesis. Both techniques can be combined either with an "Open But Do Not Touch" approach, in order to preserve the hematoma and decrease the surgical trauma or with minimally invasive percutaneous plate application (MIPO) in an effort to further decrease the postoperative morbidity.

Suggested Readings

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CARDIOLOGY

STATE-OF-THE-ART LECTURE MANAGEMENT OF PRECLINICAL HEART DISEASE IN DOGS – THE EPIC TRIAL

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Management of preclinical heart disease in dogs – the EPIC Trial

Professor Adrian Boswood, The Royal Veterinary College London

The results of the EPIC study were published in 2016. The full report of the clinical trial is available as an open access publication at the following link

http://onlinelibrary.wiley.com/doi/10.1111/jvim.14586/full

In brief summary – the aim of the trial was to evaluate whether or not dogs with enlarged hearts, secondary to myxomatous mitral valve disease, that had not yet developed signs of congestive heart failure, would benefit from the administration of pimobendan.

We tested our hypothesis by conducting a double blind placebo controlled trial in which 360 dogs were randomly allocated to one of two treatment groups: pimobendan or placebo.

The primary endpoint of the study was a composite of the onset of signs of congestive heart failure or cardiac related death (which ever occurred first).

The study demonstrated a clear benefit associated with the administration of pimobendan. The likelihood of a dog reaching the primary endpoint was reduced by approximately one third if it received pimobendan.

The median time to the primary endpoint was approximately 15 months longer in the treated group indicating, on average, a prolongation of the time to the development of clinical signs by over a year.
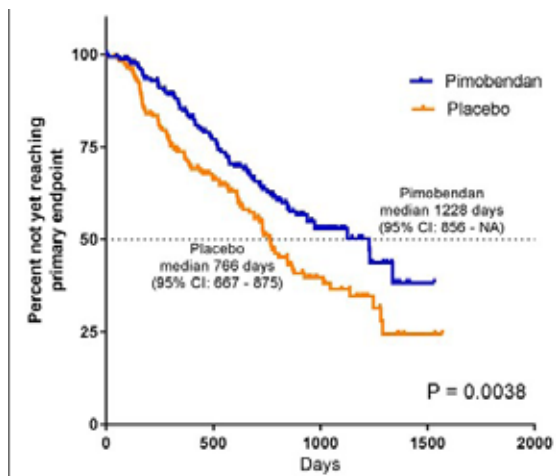
Figure 1 – A Kaplan Meier survival plot illustrating the estimated time to the primary endpoint in the two treatment groups in the EPIC trial.

Ramifications of the EPIC trial

As a result of the findings of the EPIC study we can conclude that there is a benefit of administration of pimobendan before the onset of signs of CHF in dogs with DMVD. We cannot however conclude that **all** dogs with DMVD will benefit. No dogs without cardiac enlargement (stage B1 dogs) were recruited to the study and therefore we can only conclude that there is a benefit associated with the administration of pimobendan to dogs with DMVD **that have enlarged hearts** (i.e. stage B2 dogs).

The majority of dogs recruited to the study were completely free of any outward signs of disease. The majority of the dogs' owners did not consider their dog to be coughing, nor did they feel that their dog's exercise tolerance was in any way compromised. This has the important implication that dogs appear to benefit from therapy before they are showing any signs. This in turn means that taking the approach of waiting until signs develop may now result in some dogs being denied potentially life-prolonging and life-improving therapy.

It is this point which highlights the key change in the management of patients with mitral valve disease which the EPIC study has brought about. Previously taking a "watch and wait" approach was entirely justified on the basis of the available evidence. A patient would be expected to demonstrate some signs – such as coughing, exercise intolerance or increased respiratory rate – either prior to, or at, the time when the introduction of therapy was justified on the basis of the best evidence available. Now if one waits until clinical signs are present before investigating a patient's disease it is possible that a stage of disease during which that patient would have benefitted from therapy would already have elapsed by the time treatment is started. The opportunity to help that patient would have been missed.

Watching and waiting, although a previously sensible approach, is no longer enough to enable you to offer the best evidence-based care to your patients with DMVD. If you are suspicious that a patient may have progressive DMVD, even in the absence of any outward clinical signs, it is worth investigating further. If such a dog is shown to have cardiomegaly, initiating treatment with pimobendan at that stage will probably result in the dog having a longer period before the development of signs of heart failure and prolong the dog's life.

How can dogs that will benefit from early therapy be found?

It is relevant to ask "how can dogs that will benefit from therapy be identified in practice?" The simple answer is "dogs like those recruited to the EPIC study will benefit from therapy" – therefore to determine whether or not a dog may benefit from therapy requires one first to be cognisant of the inclusion (and exclusion) criteria of the study. The most relevant ones are reproduced in the table below.

	EPIC study
Inclusion criteria	• Systolic murmur with maximal intensity over the mitral area (≥ grade 3/6) • Characteristic valvular lesions of the mitral valve and apparatus • Presence of MR on the colour Doppler echocardiogram • Echocardiographic evidence of left atrial dilatation 2D LA/Ao ratio ≥ 1.6 (by the Swedish method) • LVIDDN ≥ 1.7 according to the Cornell formula • Radiographic evidence of cardiomegaly VHS score > 10.5
Exclusion criteria	• Clinically significant supraventricular and/or ventricular tachyarrhythmias • Cardiac disease other than MMVD • Evidence of clinically relevant pulmonary hypertension (RA:RV gradient > 65 mmHg)

When results of a clinical trial are generalised to a larger population of dogs we are assuming that because the drug was shown to be effective in dogs defined by the inclusion criteria of the study – it will be similarly effective in other dogs that share those characteristics. We do not know whether the drug will be effective in dogs that differ from those recruited to the study and therefore we should probably refrain from administering it!

How are dogs with preclinical mitral valve likely to be discovered?

A cardinal sign of MVD is the presence of a left apical systolic murmur in a dog of an appropriate age and breed. The murmur is reliably detected on clinical examination (if dogs are auscultated carefully!) and is usually present for years before clinical signs develop.

All dogs recruited to the EPIC study had murmurs that were at least a grade III/VI in audibility i.e. at least as loud as the underlying heart sounds S_1 and S_2. If a dog is carefully auscultated and does not have a heart murmur, or has one of a low intensity it is unlikely to be a dog that will benefit from therapy. A murmur is usually present in dogs with MVD for years before clinical signs develop. In its early stages MVD is usually slowly progressive. For these two reasons, annual auscultation of at-risk dogs will probably be sufficient to identify the presence of disease well before treatment is likely to be necessary. Only dogs with louder murmurs need undergo further investigation (provided it seems clinically likely that the cause of the dog's murmur is MVD). Dogs with quiet murmurs as a consequence of MVD are very unlikely to have advanced disease. A large majority of the dogs recruited to the EPIC study were considered by their owners to be normal and have no signs attributable to

their heart disease. Therefore it is not advisable to wait for signs to develop before considering the introduction of therapy. Dogs with more advanced MVD may be more likely to have higher heart rates and a regular heart rhythm (i.e. have lost the natural variation in heart rate associated with respiration) on auscultation.

Dogs suspected of having more advanced preclinical MVD should undergo diagnostic imaging to determine whether or not its heart is enlarged. Dogs entering the EPIC study underwent both echocardiography and radiography. Of those two techniques echocardiography is the better test for demonstrating evidence of subtle cardiac enlargement. Ideally therefore dogs suspected of having DMVD should undergo echocardiography to determine their heart size. This is not to imply that radiography is of no value in determination of heart size. It is very likely that a dog with a clearly enlarged cardiac silhouette on a thoracic radiograph will probably have echocardiographic evidence of enlargement. However breed differences in heart size (and VHS) mean that where fine distinctions need to be made between normal and enlarged hearts echocardiography is superior.

The echocardiographic criteria used to recruit dogs to the study were relatively simple and therefore a competent echocardiographer should be able to determine whether a dog has evidence of left-sided cardiac enlargement on the basis of two-dimensional and M-mode echocardiographic images obtained from the right parasternal viewing locations.

It is natural to ask whether in order to initiate pimobendan therapy in a dog with preclinical MVD the dog must first undergo echocardiography. Where a dog has considerable cardiomegaly evident on radiography it is probably safe to assume there will also be cardiomegaly evident on echocardiography. The cut-off of 10.5 for vertebral heart score used in the EPIC study is too lenient to use as a sole criterion for initiating therapy because a number of normal dogs of certain breeds will exceed this value. A group of cardiologists in the US – the "Cardiac education group" – have prepared a useful algorithm.[1] They recommend that where access to echocardiography is limited a VHS of 11.5 or a rapid rate of increase of VHS may be used as a way to identify dogs that may benefit from therapy. It is however worthy of note that more than half the dogs enrolled in the EPIC study had a VHS lower than 11.5; meaning that applying this criterion alone would result in many dogs that may benefit from therapy not receiving it.

Whether there are other methods by which dogs that will benefit from therapy can be reliably identified is currently not known and therefore initiating treatment in the absence of diagnostic imaging is not recommended.

[1] http://cardiaceducationgroup.org/wp-content/uploads/2016/12/CEG_Recommendations_EPIC_121316.pdf

SVA DERMATOLOGY (SIMULTANEOUS TRANSLATION INTO MANDARIN CHINESE)

RECENT ADVANCES IN ECTOPARASITE CONTROL

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Introduction

A new group of parasiticides has recently been introduced to veterinary medicine. Afoxolaner, fluralaner, sarolaner and lotilaner are new insecticide-acaricide molecules from the isoxazoline family that act on the insect aminobutyric acid receptor (GABA) and glutamate receptors, inhibiting GABA and glutamate-regulated uptake of chloride ions resulting in excess neuronal stimulation and death of the arthropod.

Afoxolaner (NexGard®)

Afoxolaner [1-Naphthalenecarboxamide, 4-[5-[3-chloro-5-(trifluoromethyl)phenyl]- 4,5-dihydro-5-(trifluoromethyl)-3-isoxazolyl]-N-[2-oxo-2-[(2,2,2-trifluoroethyl)amino]ethyl]] is one of the members of the isoxazoline family. Afoxolaner has been demonstrated to be a highly effective and safe form of flea and tick control.

Demodex:

Afoxolaner has been shown to be highly effective for treatment of demodicosis in case reports (Chavez 2016, Mueller 2017) and one controlled study. (Beugnet 2016). The controlled published report evaluated eight dogs diagnosed with generalised demodicosis and compared the efficacy with a topical combination of imidacloprid/moxidectin. Afoxolaner was administered at the recommended dose (at least 2.5 mg/kg) on Days 0, 14, 28 and 56 and the topical combination of imidacloprid/moxidectin was given at the same intervals at the recommended concentration. Clinical examinations and deep skin scrapings were performed every month to evaluate the effect on mite numbers and the resolution of clinical signs. The percentage reductions of mite counts were 99.2%, 99.9% and 100% on Days 28, 56 and 84, respectively, in the afoxolaner-treated group, compared to 89.8%, 85.2% and 86.6% on Days 28, 56 and 84 in the imidacloprid/moxidectin-group. Mite reductions were significantly higher on Days 28, 56 and 84 in the afoxolaner-treated group compared to the imidacloprid/moxidectin treated group. (Beugnet 2016).

In a large series of clinical case evaluations at a referral dermatology practice, 102 cases of generalized demodicosis were treated with excellent results. Of the 102 cases, 68 were dogs with adult onset demodicosis. The product was administered at 2.5 mg/kg PO, initially used every two weeks in the first ten dogs. With the high degree of efficacy seen in those dogs, the dosage

was reduced to monthly in the remaining cases. Ninety percent of the cases were negative after two months of treatment, the remaining dogs after three months. The only dog needing every two week administration was a dog on immunosuppressive therapy for pemphigus foliaceus, that became mite positive when the interval was increased to four weeks, but remained mite negative when afoxolaner was administered every two weeks. (Mueller 2017)

Sarcoptes:

Afoxolaner administered at the minimum dose of 2.5mg/kg PO once a month for two treatments is effective for the treatment of infestation with **Sarcoptes scabiei** with a rapid and complete cure of mite infestation in one month and resolution of clinical signs in one to two months. (Beugnet 2016)

Otodectes:

A single oral administration of afoxolaner at the minimum dose of 2.5mg/kg PO is effective (>98%) in treating dogs with induced **O. cynotis** infestations with significantly fewer (P < 0.05) live mites present in the afoxolaner-treated group compared to the untreated group on Day 28. In this study, however, two out of eight afoxolaner-treated dogs were still infested with one and four ear mites, respectively on Day 28. (Carithers 2016)

Adverse Reactions:

Adverse reactions are rare. In a U.S. field study, vomiting was seen in 17/415 (4.1%) cases. Five dogs experienced anorexia during the study, and two of those dogs experienced anorexia with the first dose but not subsequent doses.

Fluralaner (Bravecto®)

Fluralaner (4-[5-(3,5-dichlorophenyl)-4,5-dihydro-5-(trifluoromethyl)-3-isoxazolyl]-2-methyl-N-[2-oxo-2-[(2,2,2-trifluoroethyl)amino]ethyl]-benzamide) is rapidly absorbed after single-dose oral administration, has a long elimination half-life, long mean residence time, relatively high apparent volume of distribution, and low clearance. For optimal absorption, bioavailability and efficacy, the drug should be administered with food. Numerous studies have demonstrated that a single fluralaner dose administered orally as chewable tablet provides flea and tick control for twelve weeks in dogs.

Demodex:

Fluralaner is effective for treatment of generalised demodicosis. In one study comparing a single dose of fluralaner to topically applied Advocate® (imidacloprid/moxidectin) administered once a month; a reduction of 99.8% and 98% in mite numbers was achieved on Day 28 respectively. Skin scrapings were negative in all dogs treated with fluralaner after 56 days. The excellent response of the dogs in this study receiving the imidacloprid and moxidectin spot-on after one month suggests that these dogs may not be comparable to the dogs presented with generalised demodicosis in Europe or North America. (Fourie 2015)

In a larger clinical study, 163 dogs of various breeds with generalised demodicosis (63% with juvenile- and 37% with adult- onset of the disease) were treated with fluralaner once at a single dose of 25mg/kg. The majority of dogs (87%, all of the dogs with juvenile onset and most with adult-onset demodicosis) had negative skin scrapings after one month and all dogs were negative on scraping after two months. Adverse effects were not seen. (Karaś-Tęcza 2015).

Most interesting was the management of breeding bitches of 16 different breed types; German Shepherd, pug, Great Dane, Shih Tzu, shiba, malamute, Italian greyhound, Rhodesian ridgeback, mix breed that had produced repeated litters of puppies with generalized demodicosis. In this trial, all bitches were treated with 25 mg/kg fluralaner ten days prior to scheduled mating and three months later with a second dose. Fourteen bitches gave birth to litters that were clinically unaffected by demodicosis, although two puppies from one litter developed localised demodicosis. (Karaś-Tęcza 2016).

A recent study, that included 67 dogs, also demonstrated that fluralaner when given at the recommended dose for flea and tick prevention is also effective for the treatment of canine generalized demodicosis. In 46 individuals with adult-onset demodicosis; 63%, 85% and 100% cure rates were observed after two, three and four months, respectively. In 21 dogs diagnosed with juvenile-onset demodicosis, in this same study, 81% and 100% cures were observed after two and three months, respectively. (Duangkaew 2017)

Sarcoptes:

Fluralaner at a dose of 25mg/kg once a month for a single treatment is effective for the treatment of infestation with **Sarcoptes scabiei** with a rapid and complete cure of mite infestation in one month and resolution of clinical signs in one to two months. (Taenzler 2016)

Otodectes:

A single oral administration of fluralaner at a dose of 25mg/kg is effective for dogs with experimental **O. cynotis** infestations with no mites present in the fluralaner-treated group compared to the untreated group on Day 28. In this study, however, two out of eight fluralaner-treated dogs were still infested with one to two ear mites on Day 14. (Taenzler 2016)

Adverse Reactions:

Adverse reactions in fluralaner-treated dogs were evaluated in studies evaluating its use for flea and tick control and were uncommon to rare. Transient gastrointestinal-related signs including vomiting and anorexia have been reported in 2% of dogs. Fluralaner can be used without additional risk for collies and other sensitive herding breeds that have the MDR1 mutation. No adverse events were observed subsequent to fluralaner treatment of ABCB1 (-/-) Collies at three times the highest expected clinical dose. Fluralaner seems to be an effective, safe and convenient treatment option for all breeds of dogs with generalized demodicosis.

Sarolaner (Simparica®)

Sarolaner (1-(5'-((5S)-5-(3,5-dichloro-4-fluorophenyl)-5-(trifluoromethyl)-4,5-dihydroisoxazol-3-yl)-3'-H-spiro(azetidine-3,1'-(2) benzofuran)-1-yl)-2-(methylsulfonyl) ethanone) has a very similar spectrum of activity, efficacy, pharmacokinetics and safety to the other isoxazoline molecules.

Demodex:

In a recent study, 16 dogs with generalised demodicosis were treated either with monthly oral sarolaner at 2mg/kg or with a spot-on containing imidacloprid and moxidectin (Advocate®) every 7 days. The sarolaner-treated dogs and the dogs treated with the spot-on had a reduction of over 99% and 96% in mite numbers after one month and negative scrapings after one month and after 11 weeks respectively. There were no treatment-related adverse events observed. The excellent response of the dogs in this study receiving the weekly spot-on suggests that these dogs may not be comparable to the dogs presented with generalised demodicosis in Europe or North America. (Six 2016)

Isoxazolines and Demodicosis

With the advent of these new treatments for demodicosis, as well as chronic use of these treatments as flea and tick preventatives, come new concerns about their impact on normal canine cutaneous Demodex populations. Demodex mites are considered part of the microbiota of most mammals, including dogs. Under normal circumstances, they appear to live as commensals, feeding on their host's sebum and are only opportunistically pathogenic.

A recent study, however to investigate if healthy dogs treated with the isoxazolines, afoxolaner and fluralaner at the labelled dose for flea and tick prevention maintain a normal population of Demodex mites as part of their cutaneous microbiota demonstrated that after 30 and 90 days of treatment, healthy dogs have continued presence of Demodex mites in numbers similar to the population of healthy dogs not receiving these treatments. (Zewe 2017)

These conclusions suggest that dogs on isoxazoline treatment maintain Demodex populations as part of their cutaneous microbiota, despite the apparent ability of these medications to resolve clinical demodicosis. Tested isoxazolines at the labelled dose do not appear to completely eradicate Demodex mites from the skin, but simply reduce Demodex populations to normal numbers in affected dogs. To date, no studies have been performed to detect Demodex DNA post-treatment in dogs with demodicosis. More studies are needed to characterise the response of the Demodex populations in dogs with clinical disease to isoxazolines, independently and in comparison to other treatments for demodicosis.

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FASAVA/HILLS FELINE MEDICINE

USING CTSH IN CATS

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USING cTSH IN CATS

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In human medicine measurement of thyroid stimulating hormone (TSH) is considered the single best test for accurate assessment of thyroid function. There are two important reasons for this. Firstly, there is a log-linear relationship between TSH and free T4 such that a small change in free T4 results in a much greater change in TSH. Secondly and of significant relevance for diagnosing hyperthyroidism, the current assays are capable of measuring TSH values up to 30 times lower than the lower limit of the reference interval. However, TSH is species specific and no dedicated feline assay is yet available. The greater homology of feline with canine (95 %) rather than human (78 %) TSH has prompted widespread use of the canine assay in cats. Whilst of some value in investigating thyroid disease in cats, the limitations of this assay must be acknowledged.

The most commonly used canine TSH assay is chemiluminescent (Canine TSH®, Siemens) and it is suggested that this assay is capable of measuring only approximately 35 % of recombinant feline TSH by contrast to 75 % of similarly derived recombinant canine TSH[1]. As a consequence the upper limit of the reference interval is much lower (approximately 0.3 ng/mL) in cats compared to dogs (approximately 0.6 mg/mL). More importantly the canine assay is already known to be incapable of distinguishing normal from low values with the lower reference limit simply defined as < 0.03 ng/mL. Clearly this has major implications for the cat where suppressed (i.e. low) values are expected in hyperthyroidism. In order to circumvent this problem, results for cats are usually quoted as being detectable (≥ 0.03 ng/mL) or undetectable (< 0.03 ng/mL). Greater imprecision is expected at these limits and could potentially result in the same sample giving different values on the same or different assay runs.

Almost all hyperthyroid cats have undetectable TSH concentrations and diagnostic sensitivity approaches 98 %[2]. A small number of hyperthyroid cats have detectable values including some that are not near the lower limit of the reference interval. The reasons for this are unclear. In some cases prior antithyroid medication may play a role. Many healthy cats and those with non-thyroidal illness may also have undetectable values and diagnostic specificity is only approximately 70 %. This is not surprising given the limitations of the assay and the fact that TSH suppression is likely a feature of non-thyroidal illness in cats as it is in humans. With such assay performance, TSH is not recommended as a sole diagnostic test for feline hyperthyroidism. Rather it may be used to provide support for hyperthyroidism in certain circumstances. Demonstrating undetectable TSH in a cat with appropriate clinical signs with high reference interval total T4 supports a diagnosis of hyperthyroidism. Demonstrating detectable TSH concentrations is more likely to reflect euthyroidism particularly if there are limited clinical signs.

Measurement of TSH may also be helpful in depicting which cats may become overtly hyperthyroid within months. Cats with undetectable TSH values are more likely to become hyperthyroid than those with undetectable values[3].

Despite the limitations of using TSH to diagnose hyperthyroidism, it appears to perform particularly well in diagnosing iatrogenic subclinical and overt hypothyroidism in cats after treatment for hyperthyroidism[4-6]. In such instances measurement of TSH appears to be the most sensitive and specific diagnostic test for hypothyroidism. Diagnosis of hypothyroidism is particularly important given its known relationship with kidney disease and its adverse effect on survival[7].

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APPROACH AND MANAGEMENT OF RED EYE

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Introductory Philosophy

Hyperemia of the surface of the globe or so-called "red-eye" is not only one of the most common reasons for a client presenting their pet for veterinary care, but potentially one of the most critical. As veterinary ophthalmologists, some of the most worrisome cases we see are those were ocular redness was misinterpreted as allergic conjunctivitis or "he must have got some dust in his eye" and thereby serious and potentially blinding diseases such as uveitis, glaucoma, dry eye, or ulcerative keratitis were missed. The goal of this lecture is to heighten awareness of the differential diagnoses that may cause ocular hyperemia and to define methods to differentiate these. We will look at eyes with conjunctivitis, keratitis, uveitis, glaucoma, and orbital disease.

Ocular Blood Vessels are a Diagnostician's Best Friend

In many ways, ocular blood vessels are the diagnostician's best friend since they "always" go to where the problem is. That is, if there is superficial irritation (irritation of the conjunctiva or superficial cornea) then superficial blood vessels will become hyperemic. However if inflammation involves deeper structures – uveitis, glaucoma, scleritis, or deep (stromal) keratitis - then deeper (episcleral) blood vessels become engorged. Thus, clinically differentiating superficial conjunctival vessels from deep episcleral vessels changes the diagnosis, diagnostic testing necessary, treatment, and prognosis.

Clinically Relevant Anatomy and Physiology

For the purposes of this session we will categorize blood vessels overlying the sclera into 2 distinct and clinically useful classes: deep or episcleral and superficial or conjunctival blood vessels. In the normal animal, the blood vessels in the bulbar conjunctiva are so fine that the conjunctiva appears almost transparent permitting the white sclera to be seen through it. The palpebral conjunctiva is normally a pale pink but can become more obvious with hyperthermia or excitement, and should be approximately the same color as other mucus membranes. Bulbar conjunctival vessels extend right up to the limbus. Episcleral vessels – although larger – are usually not prominent when seen through subconjunctival tissues. The exception is some brachycephalic individuals, particularly dogs, in which one or two obvious episcleral blood vessels are sometimes seen in normal, uninflamed eyes. Episcleral

blood vessels supply the intraocular structures via the uveal tract and therefore "dive" through the sclera at the iris root – a millimeter or two behind the limbus. The importance of these anatomical facts will become more obvious when we discuss some of the means to differentiate deep from superficial blood vessels.

Mechanisms of Ocular Hyperemia

In addition to physiologic vasodilation due to hyperthermia, there are (at a mechanistic level) two common ways a blood vessel becomes hyperemic or "injected".

- Vasodilatation due to release of inflammatory mediators (i.e., "inflammation")
- Hydrostatic engorgement due to decreased venous return.

Inflammation. The release of vasoactive mediators at a specific site acts locally to cause (among other things) dilation of the blood vessels that supply that site. Therefore this mechanism for hyperemia dictates that a reasonably "focused" vasodilatation should ensue. That is, if there is uveal inflammation, and release of vasoactive factors within the uvea, then the deep or episcleral blood vessels that supply the uveal tract should become injected. By contrast, conjunctival inflammation should incite only conjunctival vessel hyperemia. This strict rule breaks down somewhat with more significant or major inflammation. Insults such as uveitis that are severe enough to induce episcleral congestion will sometimes also produce some "innocent bystander" hyperemia of the overlying and smaller conjunctival blood vessels; however the inverse is unlikely. Therefore, satisfying yourself that episcleral congestion is not present is the most critical decision whenever "red eye" is a presenting sign.

Hydrostatic congestion. Blood vessels terminating (in the case of arterioles) or originating (in the case of venules) in the conjunctiva and uvea share a common pathway through the orbit to and from the major vessels of the head and neck. Therefore orbital disease can cause enlargement ("injection") of deep and/ or superficial ocular vessels via hydrostatic pressure (decreased venous return) and via local inflammatory effects on these vessels en route to and from the eye. Therefore all eyes with hyperemia should be examined for evidence of altered globe position (strabismus, enophthalmos, or exophthalmos) and - so long as there is no risk of globe rupture - should also be retropulsed.

Differentiation of deep episcleral and superficial conjunctival hyperemia

Conjunctival vessels are superficial, small (fine), branch frequently, move easily with gentle pressure from a cotton-tipped applicator or by lateral motion of the upper eyelid, extend to the limbus, and blanch within seconds after application of 1 drop of a topical vasoconstrictor

such as dilute (1%) phenylephrine. By contrast, episcleral vessels are larger, branch less, appear to "stop short" of the limbus, and blanch more slowly, if at all, with topical vasoconstrictors.

Potential Clinical Diagnoses in Reddened Eyes Since superficial vessels indicate superficial disease and deep vessels indicate deeper disease, it is possible to compile a list of potential likely causes of red eye in association with deep vascular injection (Orbital disease, deep keratitis, uveitis, glaucoma, or – rarely – scleritis) or superficial vascular injection (blepharitis, conjunctivitis, or superficial keratitis). This list guides diagnostic testing and ensures that painful, vision-threatening or potentially life-threatening diseases are not written off simply as conjunctivitis. The only confusion in this list is brought about by the principle of "innocent bystander" inflammation discussed earlier. Subtle (early) glaucoma, orbital disease, or uveitis can cause only mild conjunctival hyperemia before they progress to a stage where they cause episcleral hyperemia.

Diagnostic Tests for "Every" Red Eye

The following is a brief outline of the diagnostic tests that should be considered for all cases of reddened eye.

Retroillumination is a simple but extremely useful technique for assessment of reddened eyes. A focal light source held close to the examiner's eye and directed over the patient's nose from at least arm's length is used to elicit the fundic reflection. Each eye is illuminated equally and the fundic reflex is used to assess and compare pupil size, shape, and equality. Some general rules help interpret retroillumination findings:

- Conjunctivitis – never associated with anisocoria
- Uveitis – often associated with miosis
- Glaucoma – often associated with mydriasis

The Schirmer tear test (STT) should be performed on all reddened eyes but especially those in which there is mucoid discharge. The only exception is those with an obvious deep ulcer in which this test may be unsafe. Normal STT values for dogs are > 15 mm in 60 seconds. However STT values in normal cats range widely (3-32 mm; mean = 17 mm in 60 seconds) and are more difficult to interpret than in dogs.

Tonometry or measurement of intraocular pressure (IOP) is essential in every reddened eye except those at risk of rupture. Its use will permit differentiation of the two major, vision-threatening conditions in which red-eye is the hallmark feature – uveitis (in which IOP tends to be low) and glaucoma (which is defined by elevated IOP) – from conjunctivitis (in which the IOP will be normal). Across large populations, normal canine and feline IOP is reported as 10-25 mmHg. However, some variation occurs. Comparison of IOP between right and left eyes permits application of a reasonable rule of thumb that IOP should not vary between eyes of the same patient by more than ~20%. Perhaps the most important role for tonometry is the monitoring of progress of these diseases and the titration of medications needed.

Aqueous flare occurs as a result of breakdown of the blood-ocular barrier with subsequent leakage of proteins into the anterior chamber. Therefore, is a pathognomonic sign of uveitis and must be performed in every reddened eye. It is best detected using a very focal, intense light source (the small circular aperture on the direct ophthalmoscope works well) in a totally darkened room. The passage taken by the beam of light is viewed from an angle. In the normal eye, a focal reflection is seen where the light strikes the cornea. The beam is then invisible as it traverses the almost protein- and cell-free aqueous humor in the anterior chamber but becomes visible again as a focal reflection on the anterior lens capsule and then as a diffuse beam through the body of the normal lens. If uveitis has allowed leakage of serum proteins into the aqueous humor, then these cause a scattering of the light as it passes through the anterior chamber. Aqueous flare is therefore detected when the beam of light is visible traversing the anterior chamber.

Application of fluorescein dye to the cornea should be routinely used in all reddened eyes to diagnose corneal ulcers. It should be performed after all other parts of the exam are completed so as not to alter the STT result or affect visualization of other structures.

Retropulsion of the globe is a simple but useful method for investigating orbital disease. This is performed by applying gentle digital pressure to both globes through closed eyelids. The resistance to retropulsion and the resilience with which the globes "spring" back against the retropulsive force are subjectively assessed. Retropulsion of the globe in a variety of directions may further localize orbital masses or outline smaller masses that would be missed by direct caudal retropulsion only. This should not be done in eyes at risk of rupture.

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ANESTHESIOLOGY

TOP 5 TIPS FOR SUCCESSFUL ANESTHESIA MONITORING

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5 TIPS FOR SUCCESSFUL ANESTHESIA MONITORING

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When patients are anesthetized the veterinarian needs to take responsibility for appropriate monitoring and supportive measures to avoid mortality and/or irreversible damage to important organs. Anesthetic crisis are unpredictable, and tend to be rapid in onset and devastating in nature. Close attention to vital signs allows us to judge the depth of anesthesia, avoiding overdoses and ensuring a positive outcome maximizing the safety of the anesthetic drugs and allowing us to treat any observed complication as soon as possible.

MONITORING THE CENTRAL NERVOUS SYSTEM

During anesthesia, monitoring the CNS is a simple and reliable method used to determine the stage of anesthesia. This includes monitoring muscle tone, reflexes activity, and eye position. Basically, the swallowing reflex should be absent after induction of anesthesia and return during the recovery of anesthesia. It is usually considered safe to remove the endotracheal tube of a patient after the observation of the second swallowing reflex. The palpebral reflexes are also very useful. The palpebral reflexes can guide you during all phases of anesthesia. They are subdivided into lateral and medial palpebral reflexes. In dogs and cats the lateral palpebral reflex disappears with light anesthesia and heavy sedation. The medial palpebral reflex disappears during the induction of anesthesia and is usually associated with good muscle relaxation. Usually in dogs and cats, it is considered safe to intubate when medial palpebral reflex disappears. Palpebral reflexes should be absent when patient is well anesthetized. However, the swallowing and palpebral reflexes, can be present when ketamine is used for induction. The corneal reflex should always be present unless your patient is too deep or dead. This reflex should not tested regularly due to high risk of corneal damage. Keep this test only for real emergencies, when you are testing if the patient is alive or not. The anal reflex is unpredictable. Jaw tone is an indicator of muscle relaxation and should be relaxed when patients are ready for surgery.

The eye positions in combination with the reflexes are very useful to grade the level of anesthesia in dogs and cats. Central eye position with strong palpebral reflex and strong jaw tone equals to light anesthesia. Ventromedial rotation of the eye with no palpebral reflexes and relaxed jaw tone equals to surgical anesthesia level. Central eye position with absente of palpebral reflex, relaxed jaw tone equals to too deep anesthesia level.

MONITORING THE CARDIOVASCULAR SYSTEM

Ideally, we should monitor cardiac output of patients under anesthesia however, there is no cost effective, non invasive monitor currently available for veterinary patients that could be used during the day to day anesthesia. Since cardiac output is not an option, the veterinarian should focus the cardiovascular monitoring blood pressure (BP), heart rate (HR) and rhythm.

Peripheral Pulse Palpation is unreliable for the monitoring the BP but can help you to calculate the HR and identify arrhythmias. Pulse deficits occur when the pulse rate is higher than the auscultable HR, or some pulses feel weaker than others. If a pulse deficit is detected then an ECG is recommended. Peripheral pulse is only the difference between systolic and diastolic BPs. The presence of a palpable pulse can give you an inaccurate indication of BP. Some authors believe that a systolic BP higher than 50 mmHg is necessary to ensure palpation of pulse. However, pulse palpation cannot substitute the real BP monitoring with non-invasive or invasive techniques.

Mucous Membranes Color may change with room light. To be precise, check both the gum and tongue color. Pink = good perfusion & oxygenation. Pale or gray = vasoconstriction, significant hypotension or cardiac arrest. Bright red = endotoxic or septic shock. Bright pink = hypercarbia.

Capillary Refill Time (CRT) should be <2 seconds. When prolonged a possible hypovolemia, low BP and/or low cardiac output may be present. Also peripheral vasoconstriction due to used of alpha2 agonists or hypothermia can cause prolonged CRT.

Arterial blood pressure: The normal range that is associated with adequate tissue perfusion is: systolic arterial pressure (SAP)> 90 mmHg; mean arterial pressure (MAP)> 60 mmHg (small animals) and diastolic BP (DAP) > 40 mmHg. When values measured are less than these minimum values, we attempt to correct the hypotension with fluids, anticholinergics and sympathomimetic agents

There are several methods of BP measurement:

Doppler ultrasound BP method: usually considered to be low cost. The Doppler sounds indicate HR and rhythm. Dysrhythmias will often sound distinctly "different" on a Doppler. Then, you can confirm the type of dysrhythmia with an ECG. To measure BP, a cuff is placed proximal to the probe on the leg (the cuff width must be 40% circumference of the area to which it is being applied) after that, the cuff is inflated with a sphygmomanometer until Doppler sound disappears, then pressure is slowly released. In theory the pressure at which sound is again heard is the systolic BP. However, research shows that Doppler is not a reliable method for measurement in cats.

Oscillometric BP equipment: This technology is designed to detect oscillations in the cuff pressure that occur with arterial pulsations when the cuff is placed around an area. The monitor is able to electronically measure the pressures and display HR, systolic, mean, and diastolic pressures. Oscillometric BP monitors do not always give accurate pressures, particularly in patients with extremely slow or fast HRs, dysrhythmias and when they are very small animals, such as cats and dogs <10 kg; Again: Cuff width should be 30-40% of limb circumference. Only buy BP a monitor if research is available to prove the accuracy of that particular brand and model. Not all monitors are the same.

Direct (intra-arterial or invasive) BP monitoring technique: This is the most accurate method available. It is considered the "gold standard" for BP measurement and is the preferred method when exact measurements are important. For this technique, a catheter is inserted into a peripheral artery (most commonly, the dorsal-pedal artery or the femoral artery). Then the catheter is connected to an electrical transducer via a fluid filled extension set and the transducer is then connected to the monitor. The transducer then converts the mechanical energy of a pressure wave into an electrical signal that is displayed on the monitor screen as a waveform and as a numerical display of systolic, diastolic, and mean BP, and HR. The resultant waveform on the oscilloscope screen gives an indication of contractility, vasoconstriction or vasodilation, and volume status of the patient. For instance, myocardial contractility is indicated by the rate of upstroke of pulse pressure wave. Stroke volume is indicated by the area under the systolic ejection phase. Vasodilation is associated with steep down stroke and low dichroitic notch. Vasoconstriction is often described by gradual down stroke and high dichroitic notch. Hypovolemic patients can show exaggerated variations in size of the waveform with respiration (generally just see this with IPPV) and arrhythmias can also be detected as an absent or altered pressure wave form.

Electrocardiography (ECG): Cardiac impulse generation and conduction are assessed electrocardiographically. The sinus node is the site of normal impulse generation, and the typical p", "QRS", and "T"-waves are the result of propagation of sinus impulses in the normal, orderly sequence of atrial and ventricular depolarization and repolarization. The appearance of the normal cardiac waveform varies with the choice of lead orientation, but the morphology of electrocardiograms is directly related to the site of impulse generation, and/or the route of impulse conduction.

Remember: ECG provides only HR and rhythm; A normal ECG does not indicate adequate cardiac output or tissue perfusion. Patients with significant arrhythmias identified on ECG (i.e., ventricular premature contractions or ventricular tachycardia) should have BP measurement performed to determine if tissue perfusion is adequate.

MONITORING THE RESPIRATORY SYSTEM

The respiratory system is monitored to insure adequate oxygenation and ventilation. Oxygenation is also dependent on adequate cardiac output, lung function, inspired oxygen levels (FiO_2), ventilation and hemoglobin concentration.

Capnography ($ETCO_2$): The capnograph or end tidal carbon dioxide ($ETCO_2$) will help you to understand the ventilator status of your patient. The normal range is between 35-45 mmHg. If $ETCO_2$ is higher than 45mmHg the patient is hypoventilating (most common reason under anesthesia). This can also be caused by equipment failure such as stuck one-way valve or exhausted soda lime (when patient is re-breathing CO_2) in a rebreathing circuit or the fresh gas flow is too low in non-rebreathing circuit. If the patient is hypoventilating, you can correct the $ETCO_2$ with intermittent positive pressure ventilation (IPPV); if the equipment is malfunctioning, repair the equipment.

When the $ETCO_2$ is less than 30mmHg your patient is hyperventilating. This can be cause by: iatrogenic hyperventilation, V/Q mismatch, sampling of dead space, there is a leak in the breathing system, the patients endotracheal tube is disconnected from the anesthesia machine, the endotracheal tube is in the esophagus or perhaps your patient has low cardiac output.References are available upon request

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SVA INTEGRATIVE MEDICINE

IMPROVING NEUROLOGICAL OUTCOME WITH ACUPUNCTURE: SCIENTIFIC BASIS AND PRACTICAL TECHNIQUES

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Introduction

Intervertebral disc disease (IVDD) is commonly seen in dogs in veterinary practice. The clinical signs of IVDD are back or neck pain, trouble walking, lameness, trouble urinating, paresis and/or paralysis. The most common types of thoracolumbar IVDD, are caused by herniation of the nucleus pulposus into the spinal canal (Hansen's Type I), and protrusion of the intervertebral disc into the spinal canal, with the dorsal annulus still coving the disc material (Hansen's Type II). Acupuncture and rehabilitation can be effective at returning to dogs to an ambulatory state, and return of normal function, when used alone or in combination with Western medicine and surgery, for both types of IVDD. Several studies have shown promising effects of using electroacupuncture at treating IVDD in dogs and rat models. This presentation explains the Traditional Chinese Veterinary Medicine (TCVM) etiology and effective treatment modalities, which include physical rehabilitation used in IVDD cases.

TCVM Etiology and Pathophysiology in IVDD

The nervous system is related to the Kidney (bones and spinal cord), the Liver (joints and smooth flow of Qi and blood), and the Spleen (muscle strength). IVDD are often also considered as a Bi syndrome and are usually accompanied by a Wei (weakness) syndrome. There are 2 Excess Patterns and 3 Deficiency Patterns that are associated with various forms of IVDD. The Excess conditions are invasion of WindColdDamp and QiBlood stagnation, which are often associated with acute trauma in chondrodystrophic dogs (Type I). The Deficiency Patterns, often associated with chronic, Type II IVDD in nonchondrodystrophic breeds, include Qi/Yang Deficiency, Yin Deficiency and combined YinQi Deficiency (Table 1).1

TCVM Treatment for IVDD

1) **Acupuncture**: Acupuncture has been proven to be an effective therapy for IVDD.2-8

A general acupuncture treatment plan for a patient with IVDD is as follows:

a) Dry needle: GV-20, Liu-feng, BL-60, KID-3, SP-10, LIV-3, BL-40, ST-41, GV-1

b) Electro-acupuncture: (20-40 Hz for 10 minutes, 80-120 Hz for 10 minutes, 200 Hz for 10 minutes) at the following pairs of acupoints:

• GV-14 to Bai-hui or GV-1

• Left BL-11 to right Shen-shu

• Right BL-11 to left Shen-shu

• Hua-tuo-jia-ji at or proximal and distal to the suspected or diagnosed disc space, bilateral

• ST-36 to GB-34, or ST-36/GB-34 bilateral

• KID-1 to BL point proximal to IVDD lesion, or KID-1/ Liu-feng, bilateral

c) Aqua-acupuncture (Vitamin B12) at Hua-tuo-jia-ji at or proximal and distal to the suspected or diagnosed disc space, KID-1, BL-40, LIV-3, LI-4, Liu-feng

d) Hemo-acupuncture: use acupuncture needle or 24G hypodermic needle to puncture Jing-well points on the affected limbs and Wei-jian acupoint and get a few drops of blood.

2) **Scalp Acupuncture:**

a) Motor area: A line starts at GV-22 and extends cranial and ventral to TH-23 on the lateral eyebrow

• GV-22: In the small triangular area formed by the ridges of the frontal crest

• TH-23: In the depression on the rim of the orbit at the end of the eyebrow were it extended to the lateral canthus

b) Sensory area: Starts at GV 20 and extends cranial and ventral to Nao-shu

• GV-20: On dorsal midline on a line drawn from the tips of the ears level with the ear canals

• Nao-shu: Over the temporalis muscles ⅓ the way along a line from the cranial ear base to the lateral canthus

c) Long insertion methods: use 32-38G acupuncture needle (½" for small dogs and cats; 1" for big dogs) to penetrated subcutaneously the entire motor line (from GV-22 through TH-23) and sensory line (from GV-20 through Nao-shu). Rotate, lift and thrust, or EA the needles every 2-3 minutes.

3) **Herbal Medicine:** General herbal dosage for dogs is 0.5 g per 10-20 lb body weight BID for 2 to 4 months, and then as needed. (Table 1).

a) Da Huo Luo Dan or Double P II (Da Hua Luo Dan modification) is the primary herbal medicine used to treat IVDD. It may cause loose stool in some cases. It can be used as long as the gut is able to tolerate it. Do not give if patient is sensitive to herbal medications or is prone to diarrhea.

b) Add Bu Yang Huan Wu for Qi or Yang Deficiency (rear weakness, pale and wet tongue, and deep/weak pulse). Better tolerated than Double P II, so can use instead of Double P II if animal is prone to diarrhea.

c) Add Di Gu Pi San for Yin Deficiency (cool-seeking, rear weakness, red/dry tongue, fast/thin pulse).

d) Add Hindquarter Formula for Qi and Yin Deficiency (cool-seeking, rear weakness, red or pale tongue, and fast/weak pulse).

e) Add Stasis Breaker if a tumor or mass is present in the spinal cord

f) Add Jie Gu San for fractures of the vertebra.

Acupuncture Researches in Neurological Conditions

a) Analgesic Effects

- After EA at GB-30 and BL-40 for 25 minutes daily for 7 days, rabbit with injured sciatic nerve had significant higher densities of normal myelinated fibers and more small myelinated fibers as compared to those in the diclofenac and control groups. Their results revealed and confirmed that EA promotes nerve regeneration while diclofenac does not have such an effect.[12]

b) Spinal Nerve Injury

- EA combined with standard Western medical treatment (group 1) was effective and resulted in shorter time to recover ambulation and deep pain perception than Western treatment alone (group 2) in dogs with signs of thoracolumbar IVDD. Overall success rate (all dysfunction grades) for group 1 (23/26; 88.5%) was significantly higher than for group 2 (14/24; 58.3%).[3]
- The proportion of dogs with clinical success was significantly higher for dogs that underwent EAP (15/19 dogs, 79%) than decompressive surgery (DSX) (4/10 dogs, 40%); the proportion of dogs with clinical success for dogs that underwent DSX + EAP was intermediate (8/11 dogs, 73%). EAP was more effective than DSX for recovery of ambulation and improvement in neurologic deficits in dogs with long-standing severe deficits attributable to IVDD.[2]
- Acupuncture at GV26 and GB34 significantly alleviated apoptotic cell death of neurons and oligodendrocytes, thereby leading to improved functional recovery after spinal cord injury (SCI) in rats. Acupuncture also reduced the size of lesion cavity and extent of loss of axons. It also significantly reduced proinflammatory factors after SCI.[13]

c) Brain Injury

- Acupuncture can improve neuranagenesis by promoting the proliferation and differentiation of neural stem cells in brain tissues.[14]
- Electroacupuncture (EA) at ST-36 acupoint improves neurological recovery in rats with intracerebral hemorrhage. EA exerts neuroprotective effects on hemorrhagic stroke by upregulation of Ang-1 and Ang-2.[15]

Outcome Measurement

Using a 0-5 grading scale to evaluate clinical neurological signs of IVDD (Table 2), it can be a valuable tool to help choose the mode of treatment, determine the prognosis, and assess the success of treatment. 9 For the most optimum recovery, it is best to use TCVM with decompressive surgery for cases with grades 4 and 5. IVDD with Grades 1 to 3 may be successfully treated with TCVM alone.[2-8]

A 2010 study evaluated the effectiveness of acupuncture in comparison to decompressive surgery, and a combination of both surgery and acupuncture, in forty dogs that had long standing clinical signs of IVDD (>48 hours). The dogs were re-graded six months after onset of clinical signs, and were considered a success if they returned to ambulation (i.e. they decreased from grade 4/5 to grades 1/2). This research demonstrated that electro-acupuncture had a greater success (79% or 15/19 dogs) than did decompressive surgery alone (40% or 4/10 dogs). Dogs that had both decompressive surgery and electro-acupuncture had an intermediate response (72% or 8/11).[2] This study indicates that the duration of clinical signs prior to treatment appears be an important factor in determining if decompressive surgery will benefit the patient. Therefore if the clinical signs of IVDD have persisted for over 48 hours, and the animal is a grade 5 for a prolonged amount of time, electro-acupuncture is the treatment that shows the most benefit to these patients. In addition, if the client is unable to afford surgery, TCVM may be the only potentially effective treatment option.[2]

Summary

Intervertebral disc disease is commonly seen in small animal clinics. Traditional Chinese Veterinary Medicine (TCVM), including acupuncture, food therapy and herbal medicine, can be an effective singular therapy, or part of integrated therapy with Western medicine and surgery, based on a grading scale of clinical signs and type of IVDD.

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Table 1. TCVM Treatment of the most common Patterns of IVDD

Pattern Type	Clinical Signs	Tongue & Pulse	Herbal Medicine
Qi-Blood Stagnation	• Acute onset; Very sensitive on palpation • Paresis or plegia • Tends to be more Type I IVDD	T: purple P: wiry	Double P II [XE "Double P II"] (Da Huo Luo Dan[XE "Da Huo Luo Dan"])
Wind-Cold-Damp	• Very painful on palpation • Acute onset of paresis or plegia • Tends to be more Type I or II IVDD • Intrinsic Myelopathies	T: purple P: wiry	Xiao Huo Luo Dan[XE "Xiao Huo Luo Dan"] or Double P II [XE "Double P II"]
Qi/Yang Deficiency{ XE "Yang Deficiency" }	• Tends to be more chronic and progressive • Paresis/ paralysis; Lethargy • Tends to be more Type II • Heat-seeking; Cool to the touch	T: pale and wet P: deep & weak (right side weaker)	Double P II [XE "Double P II"] + Bu Yang Huan Wu[XE "Bu Yang Huan Wu"]
Yin Deficiency{ XE "Yin Deficiency" }	• Panting; Cool-seeking; Warm to the touch • Paresis/ paralysis; Lethargy • Tends to be more Type II	T: red and dry P: thin & fast (left side weaker)	Double P II [XE "Double P II"] + Di Gu Pi,[XE "Di Gu Pi"] San[XE "Di Gu Pi San"]
Yin and Qi Deficiency{ XE "Yang Deficiency" } { XE "Bi Syndrome;Kidney Yin and Qi/Yang Deficiency Bi" }	• Tends to be more chronic and progressive • Paresis or plegia • Tends to be more Type II • Dry skin, dry hair • Warm to the touch; Cool-seeking • Difficulty in rising	T: pale or red P: thready and weak at both sides	Double P II [XE "Double P II"] + Hindquarter Weakness [XE "Hindquarter Weakness"]

Table 2. Neurological grading scale in canine intervertebral disc disease

Grade	Grading Scale of Clinical Signs[9]	Recommended Therapy
0	Normal	None
1	Cervical or thoracolumbar pain, hyperesthesia	Acupuncture, herbal medication
2	Ataxia, paresis (muscle weakness) with decreased proprioception, ambulatory (able to walk)	Acupuncture, herbal medication, exercise program
3	Severe paresis with absent proprioception, not ambulatory (not able to walk)	Acupuncture +/- surgery, herbal medication, physical rehabilitation
4	Paralysis [XE "Paralysis"] (not able to stand/walk), decreased or no bladder control, deep pain perception present	Acupuncture and surgery, herbal medication, physical rehabilitation
5	Paralysis, urinary and fecal incontinence, no deep conscious pain perception	Acupuncture and surgery, herbal medication, physical rehabilitation

WSV18-0319

ORTHOPEDIC SURGERY (SIMULTANEOUS
TRANSLATION INTO MANDARIN CHINESE)

TOP TIPS IN USING CERCLAGE WIRE IN FRACTURE TREATMENT

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Top Tips in Using Cerclage wire in Fractures Repair

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Principles and Clinical Application of Cerclage Wiring

Orthopedic wire is used as a supplementation for fracture fixation for the reduction of fragments and the protection of fissures. Never use wire as a sole means of fracture repair. It is most commonly used as an adjunct to intramedullary (IM) pin fixation. The degree to which this device helps to neutralize the remaining forces acting on the fracture site also is directly dependent upon the fracture configuration; for example, full cerclage wires effectively neutralize rotational, compressive and shear forces in a long oblique fracture - but are ineffective in a short oblique or transverse fracture.

Implants

Orthopedic wire is provided as a malleable form of 316L stainless steel. It comes in a range of diameter, from 0.5 to 1.5 mm (2416 gauge). The two types of orthopedic wire available are gauge wire and loop wire. Gauge wire comes in spools or long strips (coil type). Loop wires have a preformed eye at one end for a single knot and are available in 0.85, 1.0, 1.25, and 1.5 mm diameters and in 150 and 300mm length.

Tightening methods

The three most common methods for tightening and securing cerclage are twist, single loop, and double loop knots.

Twist knot (a): To form a twist knot, old needle forceps or pliers can be used. Instruments that lock the two strands may help ensure that the twist forms with an even wrap. The first two to three twists should be formed by hand. The loose twist is then grasped with the instrument and further twists formed while pulling very firmly away from the bone. This will ensure that the twist is tightened at its base, and that the wires wrap around each other NOT one around the other. Once the wire is tight, (meaning that doesn't longer moves if pushed) or the surgeon feels that further twist will cause the wire to break, the twist is cut leaving 3 wraps of wire. It is very important not to wiggle the cerclage wire while cutting it otherwise there is the risk of undoing the twist and loose the tension. Another important recommendation is to not push the twisted wire down to adhere to the bone surface as this maneuver will generally reduce the initial tension.

Single loop knot (b): Single loop knot is formed using a length of wire with an eye at one end. The free end is passed around the bone and through the eye, and tip of the tightener. It is then passed into the wire tightener rotating crank. The wire is tightened around the crank, and by rotating it the wire is tightened. Once the desired tension is achieved, the wire tightener is bent over to lock the free end within the eye. The wire tightener is loosened and the free end pushed down so that it is bent back completely on itself. The wire is then cut, and flattened further if needed.

Double loop knot (c): Double loop knot is formed using a length of wire that is folded over on itself to present a double strand of wire. The fold is compressed but left open enough to be able to pass two strands of wire through the bent end. The folded end is passed around the bone, and the two free ends passed through the loop. The wire is pulled tight. The free ends then are passed into the wire tightener using 2 cranks. Each wire end is loaded into one of the cranks in the same manner as for single loop cerclage. The two cranks are tightened simultaneously until the tension "feels" appropriate, and the wire tightener is bent over, again while maintaining tension in the cranks. Once the bends are sufficient, the cranks are loosened, and the bending completed so that the wire ends lie flat to the bone. The wires are cut, and the arms flattened further if needed.

Clinical considerations

Full cerclage wiring: Full cerclage wiring is when the wire is placed around the bone. This technique is used for long spiral and oblique fractures and to protect fissures. The bone must be completely reconstructable for this technique to work. General "rules" for full cerclage wire application follow:

- The length of the fracture line obliquity should be at least twice the diameter of the bone.

- All wires should be spaced approximately 1.0 cm apart, and all wires should be placed at least 0.5 cm form the end of a fracture.

- All wires need to lie directly on bone (no interposition of soft tissues)

- All wires must be placed perpendicular to long axis of the bone.

- All wires should be placed and tightened before the primary fixation applied.

Hemi-cerclage wiring (Inter-fragmentary wiring): This is used to counteract forces of rotation and shear in transverse and short oblique fracture. Drill holes in each bone segment and pass the wire through the holes. This technique adds little to the mechanical stability of a fracture. The holes in the bone weaken the fragment ends. Inter-fragmentary wiring are useful for repair of some mandibular.

Principles and Clinical Application of Tension Band Wiring

Tension Band Wiring is a means of converting the distractive forces acting at the fracture site into compressive forces between the fragments. Such distractive forces are generally caused by a tendon or ligament being attached to an avulsed bone fragment. Conversion of these forces to create compression at the fracture surfaces will generate to stability and lead to bony union.

Technique

Avulsion fragments can be effectively stabilized using the pin and tension band wire technique.

The fragment is initially stabilized using either 1 or 2 k-wires. To protect this implant from being pulled out or bent by the forces exerted by the pull of the attached ligaments or tendons a cerclage wire is placed to oppose the tensile forces. An additional and very significant benefit of this technique is that the pull in the ligament and the counter-pull in the wire convert these tensile forces to compressive forces across the fracture

In principle the use of two small parallel pins is better than a single bigger pin, however the number of pin size and it size will be defined by the size of the fragment.

Once the fragment is reduced the pin/s are driven perpendicular to the fracture plane and parallel to each other. If possible they should penetrate the trans-cortex (Fig 1 A). To anchor the tension band wire a hole is drilled transversely across the main fragment on the surface away from the ligament or tendon. This hole is usually located approximately the same distance below the fracture line as the pins are above the fracture line. It is important to ensure when drilling the hole that sufficient bone is purchased to prevent the wire from cutting through it and causing a 'stress riser' point that could potentially result in a pathological fracture.

The wire is passed through the hole in the bone, brought across to the original side of the bone, around the ends of the pins and back to the other end of the wire on the starting side of the bone. This creates a figure of eight pattern

(Fig 1 B).

Twist knots are commonly used for tension band wires. To effectively tighten a tension band wire with one knot, the slack in the arm opposite the one with the knot must also draw in by the tightening process. This is very difficult in bigger wire sizes. To avoid this, a twist knot can be tied in both arms of the figure of eight.

Once the wire is tightened, the ends of the K-wire are bent over so that they lie flat against the bone (Fig 1 C).
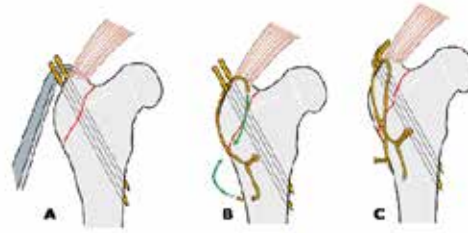
Fig. 1: *A/B/C*

Clinical Applications

The main indications for Tension Band Wiring is the treatment of avulsion fractures such as those involving the olecranon, greater trochanter, patella, tibial tuberosity malleoli, calcaneus.

In all this avulsion fractures the fragment is distracted by the muscle, tendon or ligament which originates/inserts on it.

The tension band is placed so that counteracts the tensile forces acting on the fragments and redirects it to compress the fragment against the adjacent bone. Tension band wiring is also used to repair osteotomies, for example trans-olecranon osteotomy in the caudal approach to the elbow joint or osteotomy of the greater trochanter in the dorsal approach to the hip joint.

Case Example

The treatment of an avulsion fracture of the tibial tuberosity is used here a an example of tension band wiring (Fig 2). This injury is quite common in Greyhounds and Terrier breeds and the fracture occurs through the growth plate. The tibial tuberosity is distracted by the tensile forces exerted by the quadriceps muscle group through the straight patellar tendon.

The fracture is initially reduced and fixation is achieved with two K wires driven through the tuberosity into the metaphysis. The idea behind placing two K wires is that they suppose prevent rotation of the fragment. In reality a well reduced avulsed fragment attached to the tendon has very little tendency to rotate particularly once is under compression, therefore o single pin in in most of the cases more than adequate for the purpose. If the choice lies between one large and two smaller pins the latter is the best choice. A hoe is drilled transversely through the tibia distal to the fracture site.

A length of orthopedic wire is passed through the hole, it is very important to pay attention to use the correct diameter of the wire. As a general indication:

For patients over 20 kg of bodyweight use 18 Gauge (1.2 mm diameter)

For patients below 20 kg of bodyweight use 20 Gauge (1.0 mm diameter)

The ends of the wire are brought across the cranial aspect of the tibia in a "**figure of 8**" pattern and then passed under the straight patellar tendon before being twisted tight. As for cerclage wire twisting technique, is important to insure that the two wire ends are correctly twisted around each other and not one around the other to form a "skip knot."

As the wire is tightened its proximal loop engages on the protruding ends of the Kirschner wires. Each K. wire is than bent and cut leaving a hook of about 5 mm long that is rotated to fit snugly against the tibial tuberosity and insertion of the straight patellar tendon.

Although theoretically a twist should be placed in each side of the "figure of eight" to achieve uniform tension in the wire, adequate tightening is achieved with a single twist on just one side particularly in short tension band constructs.

Fig 2:

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CARDIOLOGY

ESSENTIALS OF EKG INTERPRETATION (E)

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How do we record an EKG and what gives rise to the observed impulses?

The diagnostic electrocardiogram (ECG or EKG) is recorded by the placement of electrodes at various points on a patient's skin surface and then comparing the potentials recorded at those sites either with the potential recorded at another site (in the case of bipolar leads) or with a derived potential that is meant to represent the "average" potential of the patient in the case of unipolar leads.

In canine and feline patients the electrodes are usually placed on the limbs and the three main leads that are recorded are the limb leads that make up Einthoven's triangle. Lead I is derived by comparing the potential between the two forelimbs, lead II by comparing the potential at the right forelimb with that of the left hindlimb and lead III by comparing the potential of the left forelimb with that of the left hindlimb. The deflections displayed in all leads are a consequence of the intrinsic electrical activity of the heart. They represent the potential differences created by the depolarisation and repolarisation of the heart and by carefully characterising the different components of these deflections we can determine if the depolarisation and repolarisation are normal and indirectly derive some information about the size of the heart chambers and the metabolic status of the patient.

Normal depolarisation is initiated at the sino-atrial node and leads to a normal sequence of activation of atrial and then ventricular myocardial tissue. Potential differences between different parts of the heart are generated due to the transmembrane ionic fluxes that occur during depolarisation of the heart.

The pattern of depolarisation that happens under normal circumstances is initiated at the sino-atrial node. The wave of depolarisation initiated here is propagated throughout the remainder of the myocardium by rapid conduction through the specialised conducting tissues. The wave is carried between atria and ventricles through the atrioventricular node. The normal pattern on an EKG is

- P-wave representing the atrial depolarisation
- PQ interval representing the delay in conduction between atria and ventricles caused by the slow conduction at the AV node.
- QRS complex caused by depolarisation of the ventricles.
- T-wave caused by repolarisation of the ventricles.

The cardiac rhythm is normal when these normal deflections occur at a normal rate (for the species being examined). Any divergence from this is considered abnormal.

How do you record a good quality EKG?

In order to be able to interpret an EKG it is first necessary to obtain an EKG of a good quality. A good quality EKG has the following characteristics

1. It is clearly labelled and shows both vertical and horizontal calibration. If the filter is used this should be noted. Ideally a period of 50 mm/sec should be included on the trace and a prolonged "rhythm strip" of lead II recorded at 25 mm/sec.
2. It shows the minimum artefact throughout the majority of the recording.
3. All six frontal plane leads (I, II, III, avR, avL, avF) are recorded. Chest leads may be recorded.
4. It clearly shows details of the case from which it was recorded including the species and breed.

The EKG should be recorded with the patient in right lateral recumbency being gently restrained. Cats may tolerate the recording of an EKG better if they are in sternal recumbency. If right lateral recumbency is not used then this should be noted. Recumbency will not affect the cardiac rhythm but may alter the magnitude of some deflections. Sedation should be avoided if at all possible. Most sedatives will in some way alter the cardiac rhythm. The most profound effects on cardiac rhythm are seen when the alpha-2 agonists are used.

The EKG electrodes should be placed on the patient's limbs, on the forelimbs behind and slightly proximal to the elbow, on the hindlimbs cranial and proximal to the stifle. Good electrical contact should be ensured using the either coupling gel or surgical spirit.

The patient should be held on an electrically insulated surface and as far away from other electrical equipment as possible. Particularly avoid close proximity to strip lighting and computer screens. This should minimise the risk of 50 Hz interference.

Once the ideal recording environment has been achieved then recording can proceed.

Interpretation of the EKG trace

Basic interpretation can be achieved by asking a few simple questions when faced with the EKG trace. The most important aspects of interpretation involve the determination of the heart rhythm and assessment of whether the rhythm is normal or not. The EKG may also provide clues as to the presence of enlargement of some cardiac chambers.

Is the EKG of a diagnostic quality? i.e. free from artefact, labelled and within the boundaries of the paper.

If the EKG is of a poor quality or not properly labelled then less information can be obtained. Subtle changes can be missed when there is considerable artefact. The time to notice artefact is when the trace is being recorded as another trace can be recorded at that time.

Interpretation of rhythm

What is the heart rate?

Many significant rhythm disturbances disturb the heart rate. Arrhythmias that lead to an increase in heart rate are described as tachycardias. Arrhythmias that lead to a decrease in heart rate are called bradycardias. Instantaneous heart rate can be calculated on the basis of the R-R interval from one complex to the next. An average rate can be taken by counting the number of depolarisations within 6 seconds and multiplying by ten. Where the rhythm is very irregular a more accurate rate may be obtained by counting over 12 seconds.

Rates are therefore calculated as below

a) Method of calculation of average heart rate (paper speed 25 mm/sec)

A distance of 15 centimetres from one R-wave is inspected on the lead II EKG strip. The number of R-R intervals in this 15 centimetres is calculated to the nearest half interval. This number is then multiplied by ten to provide the average heart rate to the nearest five beats per minute.b) Method of calculation of the instantaneous heart rate (paper speed 25 mm/sec)

If the R-R interval is x mm the instantaneous heart rate is calculated as HR = $\frac{1500}{x}$ beats per minute.

(Where the EKG is recorded at 50 mm/sec the heart rate is calculated as HR = $\frac{3000}{x}$ beats per minute.)

Determination of Rhythm

Evaluation of the heart rhythm involves examining the EKG for evidence of the normal relationship between the P-waves and the QRS complexes. This can be done in the following way

Is there a P-wave for every QRS complex?

Where a QRS complex arises without a normal P-wave it implies that the atria did not depolarise normally prior to ventricular depolarisation. This can occur when the depolarisation resulting in the QRS complex arises in the wrong place, or the atria are unable to depolarise normally. The possibilities are therefore either

- Ventricular depolarisation
- Junctional depolarisation (The junction refers to the AV node and bundle of His)
- Atrial standstill
- Atrial fibrillation or
- Sinus arrest with escape complexes.

Is there a QRS complex for every P-wave?

If a P-wave is visible on the EKG and it is not followed by a normal QRS complex then there has been failure of conduction of the atrial depolarisation through the atrioventricular node in the normal way. This is described as atrioventricular block and can occur in many forms. There are three types commonly recognised and these are described as first, second and third degree AV block.

First degree AV block is a prolongation of conduction through the AV node. Second degree AV block is an occasional failure of conduction through the AV node and third degree AV block is complete failure of conduction through the AV node. In the latter case there must be an escape focus beneath the AV node to maintain ventricular depolarisation, albeit at a lower rate than normal.

Are the P-waves and QRS complexes consistently and reasonably related?

P-waves and QRS complexes may arise concurrently and yet not be related. This tends to show as an inconsistent relationship between the two and implies the presence of separate ventricular and atrial rhythms. This is described as atrioventricular dissociation.

Are the QRS complexes and the P-waves all the same?

Variation in the appearance of P-waves or QRS complexes may imply that they have originated from a different site or been conducted differently. This would normally suggest an abnormality of rhythm however some variation in P-wave amplitude can be normal in dogs and is described as a wandering pacemaker.

Is the heart rhythm regular or irregular?

If the rhythm is irregular is it regularly irregular or irregularly irregular. Normal rhythms tend to be either regular, or regularly irregular. An irregularly irregular rhythm is almost always abnormal. The most common rhythm of this type is atrial fibrillation; this sounds chaotic. Auscultation is a more sensitive way of determining the regularity of a rhythm.

• Determination of magnitude of deflections.

a = P-wave amplitude, b = R-wave amplitude,
c = P-wave duration, d = P-Q interval,
e = QRS duration, f = QT interval
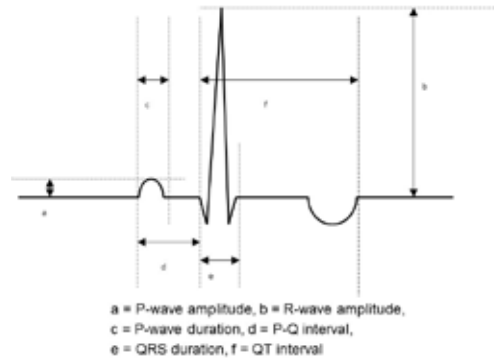
Where normal complexes are visible, i.e. those that appear to have arisen in the normal sequence from sino-atrial node to atrioventricular node to the ventricle, then measurement of the amplitude of the constituents of the complexes may be useful in providing information regarding the presence of chamber enlargement. There are a few patterns of chamber enlargement described.

- Right atrial enlargement (RAE) may be represented by the presence of a tall P-wave, i.e. greater than 0.4 mV. This is sometimes also described as P-pulmonale
- Left atrial enlargement (LAE) may be represented by the presence of a wide P-wave, i.e. longer duration than 0.06 seconds. The P-wave may also become notched or M shaped. This is sometimes described as P-Mitrale.
- Left ventricular hypertrophy (LVH) may be signified by the presence of tall R-waves, wide QRS complexes and a shift of the mean electrical axis to the left.
- Right ventricular hypertrophy (RVH) may be signified by the presence of deep S-waves in leads I, II and III. There may be a shift of the mean electrical axis to the right.

Unfortunately none of these changes is specific to chamber enlargement. There is considerable overlap between normal animals and those affected by hypertrophy. For this reason the chamber enlargement suggested by the EKG must be interpreted in the light of other available information e.g. radiographs, echocardiography or post-mortem data.

What does it mean?

Electrocardiographic abnormalities are rarely significant in isolation. Abnormal findings should be interpreted in conjunction with physical examination findings and the results of other diagnostic tests. The EKG is a relatively poor indicator of changes in size of cardiac chambers but is an excellent indicator of the presence of cardiac arrhythmias.

WSV18-0027

VACCINE SEROLOGY

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THE ROLE OF SEROLOGY IN VACCINATION DECISION MAKING

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profmjday@gmail.comCorrelates of Protection

Protection is the ability of a vaccine to prevent or reduce the effects of infectious disease (depending on the claim for the particular vaccine) when a vaccinated individual encounters virulent infectious agent. In regulatory terms, protection is defined in the context of a challenge experiment. For European vaccine licensing, 80% of vaccinated animals must be protected from disease, while 80% of controls must succumb to the infection. Correlates of protection may be used to indicate whether a vaccinated animal is likely to be protected from challenge with virulent pathogen. It is now well recognized that the presence of virus-specific serum antibody correlates strongly with protection from canine distemper virus (CDV), canine adenovirus (CAV), canine parvovirus (CPV) and feline parvovirus (FPV). In contrast, rabies serology is only used to determine whether vaccinated dogs or cats achieve a mandated antibody titre at a particular time post vaccination for the purposes of pet travel to certain countries.

Numerous studies have shown that dogs that were appropriately vaccinated as puppies (i.e. according to current guidelines) have persistence of protective serum antibody titres for long periods of time and up to the lifetime of the animal. The most recent of these studies shows that dogs last vaccinated up to 9 years previously have protective titres of serum antibody [1] and in an experimental setting; such antibody has been demonstrated up to 14 years after the last vaccination [2]. Similar studies clearly show persistence of serum antibody to FPV in vaccinated cats.

Serological Testing

Until recently, the only means of testing for serum antibody specific for vaccine antigens was to submit samples to a specialist diagnostic laboratory. The 'gold standard' tests for detection of antibody to CDV, CAV and rabies virus is the virus neutralization (VN) test and for CPV and FPV the haemagglutination inhibition test (HAI). Testing laboratories provide an antibody titre and will suggest whether that titre is above a threshold that is considered 'protective'. The titre is defined as the reciprocal of the last serum dilution giving an unequivocally positive reaction in a serological test. Practitioners must remember that the titre indicates a range and is not a fixed number. For example a titre of 640 indicates that the serum sample contains antibody in a range not less than 320 and not more than 1280 in a doubling dilution series.Recently, in-house test kits have become available for the determination of the presence of protective concentrations of serum antibody against vaccine-preventable viral diseases of the dog and cat. The TitreChek™ test is produced by Synbiotics and marketed by Zoetis, while the VacciCheck™ test is produced by BioGal Laboratories. New test kits continue to emerge (e.g. Fastest™, Diagnostik Megacor, Austria). TitreChek determines whether a dog is protected against infection by CDV and CPV, while VacciCheck tests for protective antibody against CDV, CAV and CPV. A separate VacciCheck kit tests cats for the presence of serum antibody against FPV. In some countries the feline VacciCheck kit still includes feline calicivirus (FCV) and feline herpesvirus (FHV), but these antigens are being removed as the correlation between seropositivity and protection is less clear for these infectious agents. The kits are ELISA-based technologies, but while TitreChek uses a familiar microtitration plate format, the VacciCheck system uses an 'immunocomb' in which the reactions occur on spots impregnated into the teeth of a card-like comb and Fastest is a lateral flow procedure. TitreChek and VacciCheck kits have been validated against the gold standards, are simple to use in practice and provide a result within 20–30 minutes. Testing currently costs more than the price of a vaccine. Helpful instructional videos are available on-line (see Maddies Fund: http://www.maddiesfund.org/maddies-laboratory.htm) that work through the performance of each test kit. There are some variations between the two kits: for example both can be run with serum or plasma, but only the VacciCheck kit may be used with a whole blood sample. The 'read out' for the TitreChek system is a simple 'yes or no' (protected or not) answer, while VacciCheck provides a semiquantitative scoring system.

Both tests claim good sensitivity and specificity and there are now several published papers that independently evaluate the kits against gold standard tests. One study of the feline VacciCheck test examining antibody in shelter cats reports a sensitivity of only 49% but specificity of 99% for FPV [3], but another reports 78% and 89%, respectively [4]. In the same animals (in the first study) the sensitivity for FHV was 91% with 97% specificity and for FCV, the figures were 90% and 91%, respectively. The TitreChek kit was evaluated in a study of shelter dogs in Florida, giving a sensitivity of 98% for CPV (specificity 98%) and a sensitivity of 88% for CDV (specificity 95%) [5]. A further shelter study used

the TitreChek kit to evaluate seroconversion one and two weeks after vaccination in a population of proven seronegative dogs entering the shelter [6].

A new UK study has used VacciCheck to test serologically a population of 486 dogs visiting two large practice groups. These dogs were last vaccinated up to 124 months previously and the rates of protection were 96% for CDV, 97% for CAV and 98.5% for CPV-2 [7].

Although this current generation of test kits is a major advance, the ideal test would be rapid (i.e. 5 minutes within the period of a consultation), individual (rather than needing to be batched), cheap (cheaper than a vaccine) and able to give a simple yes/no (protected/not protected) answer.

Applications of In-house Testing

There are several applications of these test kits within the veterinary practice. The first of these is to determine whether puppies have appropriately responded to the primary course of core vaccination. According to current guidelines, the third dose of core vaccine should be administered at16 weeks of age or older. Testing pups at 20 weeks of age will indicate those that are seropositive and therefore protected. Such pups may not require the fourth 26 week (or 52 week) vaccine. Seronegative pups are not protected and may be revaccinated and retested. Those that fail to respond after revaccination may be either 'low responders' or genetically-determined 'non-responders' that are incapable of making an immune response to that antigenic component of the vaccine. Such animals are estimated to be rare within the population, but there are higher risk breeds including the rottweiler that has a known predisposition to mounting less effective immune responses to CPV and rabies virus.

Another indication for serological testing is to determine the protective status (and therefore core vaccination requirements) of a newly adopted dog of unknown vaccination history or a dog which has not been revaccinated for some time. Seropositive dogs remain protected, while seronegative animals should be vaccinated.

When an animal has a history of an adverse event following vaccination, serological testing can be used to determine whether core revaccination is necessary for that animal. If vaccination is suspected as a trigger factor in an adverse event (e.g. an immune-mediated disease), then vaccination should be minimized in that animal in the future. As long as dogs remain seropositive for CDV, CAV and CPV they do not require revaccination. The use of non-core vaccines in such dogs should be considered carefully.

It is becoming increasingly popular for practices that have embraced the 'annual health check' concept to routinely offer serological testing in lieu of triennial core revaccination. Clients are appreciative of this option and it makes greater medical sense to determine whether a core vaccine is required than to give a vaccine unnecessarily. Triennial 'titre testing' is adequate for adult animals, but current advice for geriatric patients (i.e. dogs > 10 years of age and cats >15 years old) would be to perform annual testing.

Finally, in-house serological testing has revolutionized the ability of the veterinarian to manage infectious disease outbreaks in animal shelters – particularly CDV, CPV or FPV outbreaks. Animals within the shelter are tested in order to identify seropositive and protected animals that should be housed together and separated from seronegative (susceptible) animals that will then be vaccinated. Seronegative and vaccinated animals should not be adopted out of the shelter until beyond the incubation period for the disease (2 weeks for CPV or FPV; 6 weeks for CDV) and they have seroconverted. Animals needing to enter the shelter should also be tested. Seropositive (protected) animals may enter and be housed with the seropositive residents, while seronegative animals should not enter the shelter and rather be fostered elsewhere.

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GASTROENTEROLOGY / LIVER

HOW TO TAKE AND INTERPRET A LIVER BIOPSY

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Introduction

The clinical should always give careful consideration to the reasons behind taking a biopsy: you take a biopsy to give you a diagnosis, prognosis and/or guidelines for treatment. There is no clinical justification for undertaking a procedure as invasive as a liver biopsy unless it changes treatment decisions – doing it for 'interest' is certainly not justified. However, because the results of blood and imaging tests are non-specific in liver disease, some form of liver biopsy is usually indicated to give a diagnosis and allow most effective treatment. The clinician must then decide the best way to perform this biopsy considering how stable the patient is, the financial resources of the owner and the relative reliability of the results obtained with different methods.

Biopsy methods

There is also little point in taking a biopsy which is not representative of the underlying disease – if the sample is too small, or from the wrong place, or only from one of a number of organs affected with disease, it may lead to the wrong conclusions being drawn and the wrong, or incomplete, treatment protocols.

Not all biopsies are created equal. The options for liver biopsy are:

- Fine needle aspiration (FNA) cytology – not strictly a biopsy but worth considering here as a potential alternative to more invasive biopsies
- Ultrasound-guided trucut biopsy
- Wedge biopsy – at laparotomy or laparoscopy.

Before undertaking any biopsy except FNA the clinician MUST check coagulation times and platelet count. This is even more important in cats than dogs because cats with liver disease have a higher prevalence of coagulopathies than dogs. This may be because of the high prevalence of biliary tract disease in cats resulting in fat malabsorption and vitamin K deficiency. This problem is compounded if they have concurrent inflammatory bowel disease and/or chronic pancreatitis causing exocrine insufficiency. At least a whole blood clotting time and platelet count on the feathered edge of a blood smear should be performed prior to canine and feline liver biopsies, and there is a rational argument for parenteral vitamin K treatment for 12-24 hours prior to biopsy in ALL cats.

FNAs: beware becoming 'liver FNA happy' for fear of false diagnoses and wasting the client's money! FNA of the gall bladder has a primary indication in the diagnosis of ascending biliary tract infections in dogs and cats – in fact, it is the best (perhaps only effective) way of doing this and is much more sensitive than liver culture or histopathology of a liver biopsy. It allows bile cytology and culture and selection of an appropriate antibiotic based on the results of culture and sensitivity. So, if ascending cholangitis is a differential, a careful bile aspirate should be performed. However, FNAs for anything else can be misleading: this is because of the 'selective' nature of aspirates – only things which will aspirate up a needle will be harvested and there is no anatomical information, so this is a very poor way of diagnosing diffuse liver disease such as chronic hepatitis, which is the commonest liver disease in dogs. There is a moderate indication in suspected hepatic lymphoma and feline hepatic lipidosis but even those cases can be misleading. Willard et al 1999 reported four cats in which fine-needle aspirate cytology suggesting hepatic lipidosis was misleading – in three cases, there was cholangiohepatitis on histology and in one case there was lymphoma. This is not to say that FNA does not have an important place in the diagnosis of feline hepatic lipidosis – cats in the acute phase of the disease on presentation are rarely well enough to have a general anaesthetic and more invasive biopsies. A rapid diagnosis with FNA allows initiation of supportive therapy and tube feeding. However, if the cat fails to respond appropriately after several days of feeding, consideration should be given to obtaining a liver biopsy.

- Ultrasound-guided trucut biopsies: these are less invasive than a laparotomy but do carry some risk – particularly of haemorrhage, so the dog or cat needs monitoring closely for about 12 hours after the procedure. Particular care should be taken in cats and the semi-automatic biopsy guns should be avoided in this species as they can cause significant mortality. Ultrasound-guides biopsies can be less representative than wedge biopsies – probably because of the small size of the sample. This problem is more serious in dogs than in cats with chronic liver disease. Cole et al 2002 compared the diagnosis made on ultrasound-guided needle biopsies with wedge biopsies of the liver in 98 dogs and 26 cats and found the morphological diagnoses agreed only 48% of times.

- Wedge biopsies at laparotomy or laparoscopy. These have the advantages of being more reliable diagnostically and also of allowing concurrent pancreas and intestinal biopsies to be taken to rule in or out concurrent pancreatitis and / or inflammatory bowel disease. A feeding tube can be placed and the patency of the bile duct checked – all very useful. However, there is also a significant morbidity associated with these procedures – particularly with the concurrent gut biopsies – and a small but important risk that the cat may die as a result of complications of surgery. The risk-benefit balance therefore has to be carefully discussed with the owner before the method of biopsy is decided upon.

Interpreting the liver biopsy histology report

On the liver biopsy, the clinician should NOT just read the bottom line diagnosis but should read the whole report. It is important first in dogs to be sure this IS a primary hepatitis and not a reactive hepatopathy. Sometimes the final diagnosis isn't clear for example if the pathologist has written 'reactive hepatitis' without a full explanation, the clinician may believe this is primary liver disease when in fact it is secondary. The presence of inflammatory cells alone does not mean primary disease – if there is no fibrosis and no evidence of hepatocyte death, this is not a primary hepatitis.

In primary hepatitis cases in dogs, the clinician should assess the amount and distribution of inflammation and the types of inflammatory cells involved, the degree and distribution of fibrosis and the presence of any obvious cause including build up of copper. Combining all this information allows optimal treatment of the patient. It is not possible – and indeed is potentially dangerous – to give specific treatments for CH (such as copper chelators and steroid or other immunosuppressive therapy) without biopsy confirmation of disease. It is very important that all canine hepatitis cases have a copper stain performed and if the laboratory has not already done this, it should be requested.

If a significant amount of copper is found in the biopsy, in proportion to the severity of the disease, copper storage disease should be strongly suspected and treated with chelation and dietary therapy. If copper storage disease is ruled out, the type and distribution of inflammation present may suggest a cause for the disease. If there is a strong neutrophilic component, particularly if it is periportal, there may be a chronic hepatic or biliary tract infection and consideration should be given to culturing bile or liver and ruling out chronic partial extrahepatic biliary obstruction or cholecystitis. Ideally, a bile sample should be taken for culture every time a liver biopsy is taken, as a routine procedure: it is very annoying to get back the biopsy result and then wish you had taken a bile culture at the time! If there is a granulomatous inflammatory response, it might be worth considering PCR for bartonella or other unusual infections or submitting a sample to Cornell or Bristol UK for Fluorescent in situ hydridisation (FISH) for bacteria. If there is a dense lymphoplasmacytic inflammatory component, an autoimmune aetiology might be considered. However, it is important to note that lymphoplasmacytic infiltration might also be seen in viral disease and at the current state of knowledge it is impossible to be differentiate between putative viral and autoimmune hepatitis in dogs, so the most difficult decision to make may be over the use of steroids.

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CUTANEOUS MALASSEZIA HYPERSENSITIVITY IN THE DOG

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COLONISATION AND DIVERSITY

Malassezia pachydermatis is a non-mycelial yeast. It is approximately 2 x 4 micrometers in size, has an oval shape with a thick wall and reproduces by unipolar budding. This gives the organism its characteristic shape. There are at least 13 species of lipid dependent yeast including M. sympodialis, M equine, M. caprae, M. cuniculi (colonising animals skin) and the rest: M. furfur, M. globosa, M. obtusa, M. restricta, M slooffiae, M dermatis, M. japonica and M. yamatoensis colonising human skin. M. pachydermatis is present as a commensal of the skin and mucosae of most dogs. In clinically normal dogs, the sites most frequently colonised by M. pachydermatis are the ear canals, anus, lips, chin, interdigital skin, rectum and vagina. It is uncommon on the axilla, groin or dorsum. The anus seems to be the most frequently colonised mucosal site.

PATHOGENESIS: DOGS

It is now clear that under certain circumstances, Malassezia can change from being a commensal organism to a significant pathogen. For this to occur, there has to be alteration in the skin microclimate that allow the organism to proliferate to excessive levels. These changes may involve increased temperature and humidity, changes in skin lipids, alteration in epidermal barrier function and the presence of concurrent bacterial infection. Since S. pseudintermedius and M. pachydermatis are inhabitants of the mucosae, including the oral cavity, they will continually be transferred to the skin, particularly in areas which require cleaning or grooming, and which are pruritic. Thus there is potential for the establishment of microbial overgrowth whenever the skin is damaged or there is underlying disease impairing cutaneous function. These abnormalities may occur in association with:

- **Allergic skin diseases**

- **Epidermal abnormalities** (hepatocutaneous syndrome, zinc-responsive dermatoses)

- **Endocrine diseases** (Hyperadrenocorticoidism, Hypothyroidism)

- **Breed associated susceptibility** (Basset Hound, Dachshund, Cocker Spaniel, West Highland White Terriers, Miniature poodles, German shepherds, Cavalier King Charles Spaniel are at higher risk)

- **Prior treatment with antibiotics**: suggested but not corroborated. Unlike Candida spp, Malassezia is not inhibited by bacteria but is enhanced by the increased growth of S. pseudintermedius.

CLINICAL FEATURES: DOGS

Malassezia pachydermatis most commonly causes as pruritic dermatitis that can affect the lips, muzzle, periocular region, ventral neck, axillae, interdigital areas, body folds, medial thighs and perineum. In the ear canals, Malassezia causes an erythematous, ceruminous otitis. Depending on the sites affected, Malassezia dermatitis may present as foot chewing, frantic face rubbing, ear scratching, perianal licking or scooting or more generalised pruritus.

Commonly affected areas

It is important to note that all these sites may not be affected in an individual dog. Also in severely affected dogs, the lesions can become more generalised. The lesions seen in these areas consist of erythema, often covered by a yellow or slate-grey waxy scale. The skin may feel greasy to touch and there may be an offensive, rancid odour. With time, the lesions may become hyperpigmented or develop into erythematous plaques. Chronic lesions are often lichenified. Pruritus may be quite marked. Malassezia overgrowth can also be detected in the claws of some dogs resulting in a reddish-brown staining of the proximal claw or a waxy exudate in the claw fold, with inflammation of the surrounding soft tissue. In dogs, Malassezia paronychia is commonly associated with atopic dermatitis and paw licking.

HYPERSENSITIVITY

There is good evidence to support the role of M.pachydermatis as an allergen in canine atopic dermatitis. This situation, known as Malassezia hypersensitivity, can occur alongside reactivity to other allergens or it can occur in isolation. Clinically, Malassezia hypersensitivity in dogs manifests as a highly inflammatory and pruritic response mounted to relatively low numbers of yeast organisms, though some dogs may also have overt infection with cytologically evident overgrowth. Clinical diagnosis of Malassezia hypersensitivity in the dog relies upon intradermal allergy testing, or by measuring serum allergen specific IgE. Recent studies have demonstrated good correlation between IDAT reactivity and allergen specific IgE in atopic dogs. In humans, the clinical implications of a

positive IgE result remains controversial, but more recent studies demonstrate a positive correlation between disease severity (of AD) and Malassezia specific IgE titres. This has not yet been demonstrated in dogs. A therapeutic trial is warranted to determine the significance of the cytologic findings and results in marked improvement with resolution of pruritus. Immunotherapy for Malassezia hypersensitivity is available although published studies are limited.

DIAGNOSIS

The diagnosis of Malassezia dermatitis is suggested by the typical history of regional pruritus and the presence of appropriate lesions in typical sites. A definitive diagnosis can be made by demonstrating excessive numbers of Malassezia organisms on the skin surface or in the ears and observing a response to specific therapy. The most useful and readily available tool for the diagnosis of Malassezia dermatitis or otitis is cytology. Samples may be collected from the skin surface by a variety of methods, including direct impression smears, tape impression smears and dry cotton swabs, but the easiest to use is clear adhesive tape. Tape is preferred because the organisms are sometimes not located at the surface of the lesions and repeated application of the tape to the same site will reveal deeper populations. The technique is quick and easy to perform and, with experience, tapes can be examined in the microscope and diagnosis made rapidly. The presence of numbers of Malassezia greater than 2 per high power x1000 oil immersion field is suggestive of microbial overgrowth. Commonly populations are very much higher but the organisms may be found in clusters so at least 20 high power fields should be examined. For diagnosis of Malassezia paronychia, the broken end of a wooden cotton-tip swab can be used to scrape the claw fold, and exudate is pressed and rolled onto a glass slide. For examination of ear exudate in dogs and cats with ceruminous or exudative otitis externa, rolling of exudate in a thin layer on glass slides with a cotton-tip swab is the preferred method.

TREATMENT AND CONTROL

Topical therapy

Malassezia dermatitis responds to topical therapy with antimicrobial shampoo agents containing chlorhexidine and miconazole (Malaseb®), piroctone olamine (Mediderm ®) or econazole (Sebazole ®).

It is critical that the owner is given adequate instructions when prescribing these products. They must be thoroughly massaged into the affected areas, including between the toes and in the axillae. The shampoo must be left in contact with the skin for 10 minutes before rinsing. Initially the shampoo should be used three times a week, until the condition has responded. At that time, repeat tape strips can be assessed to monitor

the improvement. If a good response is seen, the frequency of bathing can be reduced to twice and then once weekly. If the underlying problem is identified and controlled then it may not be necessary to continue long-term bathing. However if an underlying cause cannot be found, routine maintenance baths given one or twice a week can be used to keep the condition under control.

Localised areas of Malassezia dermatitis such as on the feet or lips can be treated with antifungal creams containing 2% miconazole (Daktarin ®) cream, 1% clotrimazole (Canesten®) cream or lotion, nystatin or terbinafine (Lamisil ®) or antifungal wipes containing 2% acetic acid, 2% boric acid q 12hrs or climbazole 0.5%/chlorhexidine 3% pads q 24hrs (Biohex®).

Malassezia otitis usually responds well to treatment with otic drops containing clotrimazole (Otomax® Mometamax ®), miconazole (Surolan®), terbinafine (Osurnia®) or nystatin

(Canaural®) however it is very important to identify an underlying cause to prevent recurrence.

Systemic therapy

Systemic therapy for Malassezia infections may be required for severe cases or for those in which regular topical therapy is not practicable.

Ketoconazole, itraconazole, fluconazole and terbinafine have all been reported effective. Ketoconazole used to be treatment of choice but this is no longer commercially available in Australia and has to be compounded.

Antifungal	Dose
Ketoconazole (compounded)	5 to 10mg/kg PO q 24hrs *
Itraconazole	5 to 10 mg/kg q 24hrs **
Fluconazole	5mg/kg q 24hrs ***
Terbinafine	30 to 40mg/kg q 24hrs PO ****

*A low-dose regimen for large dogs using ketoconazole 5mg/kg q 12hrs PO for 10 days, followed by 5mg/kg q 24hrs PO for 10 doses has been reported to be successful in the majority of cases, and lessens the expense of therapy.

** Itraconazole persists in the stratum corneum and therefore pulse therapy can be used. Dogs treated with 5mg/kg q 24hrs for two consecutive days followed by 5 days without treatment for 3 cycles (3 weeks) responded as well as dogs who had received the medication at 5mg/kg/day for 21 days (Pinchbeck 2002).

*** Anecdotal evidence suggests that fluconazole is not as clinically effective as the other azoles.

****Terbinafine given to dogs with Malassezia dermatitis at 30 mg/kg once daily for 3 weeks resulted in a similar improvement in cytological and skin lesion scores as in dogs given the drug at the same dose twice weekly for 3

weeks; the improvement in pruritus was higher with the daily treatment.

Severe claw fold infections may require longer treatment or higher doses, however, and otitis externa cases may not respond adequately.

There are no veterinary products licensed for the treatment of Malassezia dermatitis in cats. Systemic therapy is the treatment of choice. Itraconazole is preferred to ketoconazole because it is better tolerated in cats. Fluconazole has also been used to treat Malassezia dermatitis in cats and may be a more affordable treatment option.

Prophylaxis for chronic/relapsing Malassezia dermatitis

• Regular shampoo therapy (weekly or biweekly)

• Pulse oral itraconazole or terbinafine: two consecutive days each week

• Monitor for hepatotoxicity with CBC and biochemistry every 6 months

Immunotherapy for Malassezia dermatitis

A commercial Malassezia extract is licensed and available (Greer Laboratories®). A good response to subcutaneous immunotherapy administered for a minimum of 10 months was reported in nine of 16 cases (56%) of Malassezia hypersensitivity confirmed by intradermal allergy testing with both a reduction in use of anti-inflammatory and antifungal medication as well as a reduction in pruritus scores by >50%. No adverse effects were reported (Aberg 2017)

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FASAVA/HILLS FELINE MEDICINE

INITIAL CONSIDERATIONS BEFORE CHASING REMISSION IN DIABETIC CATS

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INITIAL CONSIDERATIONS BEFORE CHASING REMISSION IN DIABETIC CATS

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Diabetes mellitus is a common endocrine disease of cats that is probably increasing in prevalence. There is strong evidence that the majority of cats suffer a disorder similar to human type 2 diabetes mellitus. This is characterised by both insulin resistance and impaired insulin secretion. In cats, as in humans, remission is a possibility particularly if those factors contributing to insulin resistance (such as obesity) and impaired insulin secretion (such as glucose toxicity) are effectively addressed.

Remission rates vary in different studies from 0 to 100 % but none are directly comparable. In practice, remission rates of between 30 and 50 % are probably more realistic. From a review of the literature it appears that dietary therapy (using a low carbohydrate high protein diet) and the intensity of monitoring strategies (particularly those that attempt to tightly control blood glucose concentrations) are extremely important in achieving remission[1]. The type of insulin used may also play a role but this appears to be of lesser importance. Many owners would prefer to know the likelihood of remission before choosing a given treatment protocol with an intense monitoring strategy. Unfortunately it is difficult to predict which cats will go into remission and which will not but generally if remission is to occur it will do so in 3-4 months. Not surprisingly remission is more likely in cats that are less severely hyperglcycaemic at diagnosis. Additionally, remission is more likely in cats intensively treated soon after (< 6 months) first diagnosis, in cats recently treated with glucocorticoids, in older cats and in those without signs of peripheral neuropathy[2]. Gender, weight (including obesity), presence of diabetic ketoacidosis, renal disease or hyperthyroidism, and frequency of hypoglycaemia are not predictors of remission. Approximately 25 – 30 % of cats in remission will relapse and most have evidence of impaired glucose tolerance or inappropriately high glucose concentrations[3]. Repeat

remission although possible becomes less likely. It may also be worthwhile testing for acromegaly early on, given it is associated with insulin resistance thereby significantly decreasing the likelihood of remission. Acromegaly may be an underdiagnosed condition and has been recognised in 25 % of diabetic cats, where only 1 in 4 was phenotypical of the disease[4]. Treatment with insulin should be instituted for two weeks prior to testing for acromegaly.

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ANESTHESIOLOGY

TOP 5 TIPS FOR ANESTHESIA OF THE GERIATRIC PET

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5 TIPS FOR ANESTHESIA OF GERIATRIC PETS

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Age is not a disease therefore, when designing an anesthetic protocol for a geriatric patient, the understanding of the whole physiological status is more important than knowing their actual chronological age. Focus should be given to co-existing diseases that leads to poor functional organ capacity, such as neurological, pulmonary, cardiac, renal, hepatic and endocrine diseases alone or in combination. A complete geriatric pre-op profile includes history, physical examination, thoracic radiography, ECG, blood work and echocardiography. History of CNS depression, polyuria/polydipsia, exercise intolerance, arrhythmias, cyanosis, abnormal pulse quality, cardiac murmurs and/or syncope indicates a need for a more extensive pre-anesthetic evaluation. Even though the focus should be on the specific organ dysfunction of that specific patient, and each patient is unique, here are some general tips for your geriatric patient that needs sedation or general anesthesia:

1- Supplement oxygen for your patient! A minimum of 3 minutes of pre-induction oxygenation via facemask, followed by intra-op and post-anesthetic phases (including after extubation) prevents possible hypoxemia. During anesthesia, manual or mechanical intermittent positive pressure ventilation and oxygen monitoring is recommended (pulsoximetry, capnography and blood gas analysis). Assisted ventilation is often recommended to maintain both normal ventilation (PCO_2 between 35-45 mmHg) and oxygenation (PaO_2 higher than 60mmHg). This recommendation is based on the common physiological changes observed in geriatric patients. They are: Weakening of the respiratory muscles, loss of elastic tissue, pulmonary fibrosis, increased airway resistance, decreased pulmonary diffusion capacity, decreased capillary blood volume and increased susceptibility to respiratory infections.

These changes combined can lead to: decreased chest wall compliance, decreased elastic recoil of the lungs associated with a decreased vital capacity and functional residual capacity, increased predisposition for atelectasis while under anesthesia, reduced efficiency for expiration and gas exchange impairment. All those changes combined can lead to hypoxemia. At the same time, the thermoregulatory center of geriatric patients is weakened and therefore they are more susceptible to anesthesia-induced hypothermia. Hypothermia can be associated with bradyarrythmias, reduced minimum alveolar concentration of inhalants and shivering. Shivering can increase oxygen consumption by 400% also leading to hypoxemia. So, warm them up!

2-Provide cardiovascular monitoring and support! The possible cardiopulmonary disease is always a possibility for the geriatric patient. Anesthesia can induce cardiovascular depression and hypotension. That is not a good combination! Therefore, close cardiovascular monitoring is detrimental to recognize the possible cardiovascular change as early as possible. The most common physiologic changes in the cardiovascular system of geriatric animals are: â baroreceptor activity, â circulation time, â blood volume, hypotension, âcardiac output and limited renal, hepatic and CNS ability to adapt to hypotension. Most of these common changes in the geriatric heart are primary related to myocardial fibrosis, valvular fibro calcification and ventricular thickening. Cardiac conduction system can also get compromised with age, leading to possible cardiac arrhythmias. Therefore, drugs known as negative inotropes and arrhythmogenics should be avoided in the geriatric patient.

Also, be careful with fluids! It is fundamental to ensure adequate venous return and fluid balance to minimize the risk of anesthesia related hypotension. However, due to the decreased cardiac reserve, fluid overload can lead to congestive heart failure and pulmonary edema. Therefore, fluid rate should be prescribed based on the individual need, hydration and physical status. So, lets use that multi-parametric monitor!

3-Use low does and/or short acting, reversible drugs! With age, the hepatic, neurological and renal functions deteriorate. All those possible changes can lead to a prolongation of the drug elimination and possible exacerbation of the drug effects on the CNS. Older dogs and cats commonly experience decreased liver mass and hepatic blood flow secondary to reduced cardiac output. Decreased microsomal enzyme activity, and generalized reduction of metabolic activity are also common. These changes are associated with hypoproteinemia, coagulopaties and hypoglycemia. For all the geriatric patients, liver function analysis and coagulation should be requested prior to the beginning of anesthesia or sedation, especially if highly metabolized drugs are used. Hypotension should be avoided during anesthesia of geriatric patients since it leads to a further decrease in hepatic blood flow, exacerbating the possible ischemic hepatic damage that is already present and associated with advanced age. The aged patient may have also compromised cognitive, sensory, motor and autonomic functions and that is usually correlated with decreased requirement for anesthetic drugs (inhalants, benzodiazepines, opioids, barbiturates) and prolonged recovery time.

Other possible problems commonly observed in elderly dogs and cats are: chronic kidney disease, urinary incontinence, bladder tumors and prostate problems. Those changes are associated with decreased renal mass, tubular size, weight and glomerular numbers leading to a reduced filtration function. Reabsorption of protein, water and sodium, secretion of aldosterone, secretion and reabsorption of anionic and cationic compounds, formation of vitamin D, renin and elimination and metabolism of protein-bound compounds are all compromised. That can influence the regulation of blood pressure, acid-base, erythropoietin, resulting in hyperphosphatemia, azotemia dehydration and hypoproteinemia. Now, general anesthesia can lead to a 40% reduction in renal blood flow and glomerular filtration. That is not a good combination! Now, that can be worse if cardiac output is already compromised by any cardiac disease. Consequently, the effects of anesthesia on the kidney can be exacerbated in geriatric patients with pre-existing cardiovascular or renal condition. Hypoxemia, hypovolemia, hypotension, and hypercarbia are factors that contribute to renal failure following anesthesia and should be avoided to decrease the chances of worsening organ dysfunction. These factors reinforce the justification for close cardio-respiratory monitoring of older pets under general anesthesia since early recognition and treatment are key to prevent further compromise of the kidney disease.

4-Pay attention to the patient's history! Hyperadrenocorticism, diabetes mellitus and hypothyroidism are common conditions in the geriatric patient. Older patients may have decreased adrenal responsiveness to ACTH stimulation when compared with younger dogs. It has been suggested that corticosteroid supplementation in the pre-anesthetic period may be beneficial for the geriatric animal because of the possibility of adrenal exhaustion in response to stress of anesthesia and surgery. So, knowing the patient history, understanding the physiology of the possible co-existing disease and working hard the stabilize the patient prior to the anesthesia induction will ensure a higher survival rate!

5- Anesthesia recovery is even more important in geriatric patients! The anesthetist should be ready to provide oxygen, heat and continuous cardio-respiratory

monitoring. It is common to see aspiration pneumonia in elderly patients. So, create the habit of aspirating the esophagus and stomach of these patients before endotracheal extubation. Maintain an open airway via intubation until the animal is swallowing, provide proper nursing care for recumbent animals with soft beds and frequent changing of decubitus, add a source of heat, provide human touch, and compassionate verbal encouragement. It is ok to wait for the pet to recover from anesthesia, rushing will not make it better. However, sometimes reversing the effects of drugs that are not analgesics (e.g. benzodiazepines and alpha2-agonists) can help to speed up the recovery time. The AAHA senior care guidelines for dogs and cats also recommends that clients should receive postoperative instructions with clear, concise, verbal and written take-home instructions that includes information about possible complications, drug effects, nursing care, nutritional management, home monitoring, and after-hours veterinary phone contact.

In conclusion, the anesthesia of geriatric patients is related with higher risks however, safe anesthesia can be performed if these guidelines are followed.References are available upon request.

SVA INTEGRATIVE MEDICINE

TREATING COGNITIVE DYSFUNCTION SYNDROME WITH ACUPUNCTURE AND HERBAL MEDICINE

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I. Introduction

With increasing age, dogs and cats may develop cognitive impairment or cognitive dysfunction syndrome (CDS), a form of neurodegenerative disorder, which shares some analogies with Alzheimer's disease in humans.[1-3] Although a declining of learning and memory may begin in dogs as young as 7 years of age, clinical cases of CDS are seldom identified until the age of 11 years or older, when dogs started to show signs of DISHA (Disorientation, Interaction changes, Dleep/wake disturbances, House soiling and Activity changes).[4,5] In a study of 180 dogs aged 11 to 16 years with no identifiable health problems, 28% of dogs aged 11-12 years, and 68% of dogs aged 15-16 years showed at least 1 sign consistent with CDS.[6] One study reported that 50% of cats older than 15 years of age had possible CDS.[7] CDS can adversely affect the quality of life in both dogs and their owners. Treatment is aimed at slowing the advancement of neuronal damage and cell death and improving clinical signs. Drugs, diet, and supplements are often used concurrently to improve neurotransmission and reduce oxidative damage and inflammation.[1]

Acupuncture and Chinese herbal medicine can be an excellent adjuvant to conventional therapy CDS patiens, as it has been used in animals for thousands of years in China.[8] Clinical anecdotal evidence indicates acupuncture and Chinese herbals may greatly benefit patients with CDS.[9,10] Acupuncture has shown to significantly improve cognitive impairment and showed to be effective in improving intelligence and ameliorating depression and anxiety in various pathological conditions in humans and lab animals.[9,11,12] Many Chinese herbs or herbal formulas have been found to posses calming effect and are able to enhance stimulate blood circulation in the brain and to promote adult neurogenesis in the dentate gyrus in animals. Thus, these herbs may be beneficial for patients with CDS.[10,13]

II. TCVM Etiology And Pathology

In Traditional Chinese Veterinary Medicine (TCVM), CDS is due to a loss of Shen. The Shen is the Spirit or Mind. Shen rules mental activities, memory and sleep. Shen also refers to the outward appearance of the vital activities of the whole body. It provides an animal with awareness and mental clarity. When Shen is healthy,

it produces inner peace. The animal with a healthy Shen will exhibit normal behaviors and will be alert and responsive to environmental stimuli. When Shen is lost, the animal will show poor memory, disorientation, confusion, restlessness, palpitation, anxiety and hyperactivity.

The Shen is housed in the Heart. The Heart Qi plays an important role in mental activities and brain functions. The plaques formed by beta amyloid peptides in the brain are considered Phlegm or local Blood Stagnation. Phlegm is often generated by the abnormal amount of accumulated fluids produced by Spleen Qi Deficiency. Phlegm in the brain tends to mist and block the Mind, leading to a loss of Shen. The Shen also requires nourishment and anchoring from Heart Yin and Blood to remain healthy. When Heart Yin and Blood are Deficient, the Shen lacks anchoring and nourishment, leading to a Shen Disturbance and abnormal behavioral changes. Thus, CDS can be divided into 5 main patterns: the 3 Excess patterns include Phlegm/Phlegm Fire Misting the Mind, Qi-Blood Stagnation, and Liver Qi Stagnation; the 2 Deficiency patterns are Heart and Spleen Qi Deficiency and Heart Yin and Blood Deficiency. Due to that most CDS cases are often already chronic by the first time the owner seeks medical attention, a combination of Excess and Deficiency is not uncommon. The most common combinations are (a) Phlegm Misting the Mind with Heart and Spleen Qi Deficiency, and (b) Heart Yin and Blood Deficiency with Qi-Blood Stagnation.

III. General TCVM Treatment

A. Acupuncture

a) Common points for CDS in animals: PC-5/6, HT-5/7, BL-14/15/43/44, GV-20, CV-15, Da-feng-men, Nao-shu, An-shen, GB-20, LI-4, LIV-3

b) Methods of Stimulation:
- Dry needle for 20-30 minutes
- Electroacupuncture: PC-6 bilateral, BL-15/44 bilateral, BL-23/52 bilateral, GV-20 to Nao-shu, Da-feng-men to Long-hui, 20Hz for 10 min followed by 80-120Hz for 10 min
- Aquapuncture: vitamin B12 injected into 5 acupoints (An-shen, Da-feng-men, GB-20, CV-14, CV-17, ST-40, BL-15/44, BL-23/52), 0.2-0.5cc per acupoint
- Acupressure or laser acupuncture

B. Tui-na Massage

a) Mo-fa (Touching skin & muscle) or Ca-fa (Rubbing) from Long-hui to GV-15 for 3-5 min

b) Clockwise thumb Rou-fa to stimulate An-shen, GV-20, Nao-shu, GB-20, PC-6, PC-8, BL-15/44, BL-23/52, KID-1, KID-3 and KID-7 for 3-5 minutes

c) Ca-fa (Rubbing) on each ear with the thumb rubbing the midpoint of the inner ear

d) Rou-Fa (Rotary-Kneading) along the back-shu Bladder meridian (BL28 to BL11) from caudal to rostral back and forth 12 times

e) Rou-fa along the Conception Vessel meridian (CV8 to CV2) back and forth 12 times

C. Herbal Medicine

a) If you do not know or cannot figure out the TCVM pattern of your patient, you may always start with Shen Calmer (from Jingtang Herbal Inc.), 0.5 g per 1020 lb body weight BID-TID

b) It is functioning to calm the mind, nourish Heart Yin and Blood, and soothe Liver Qi

IV. Scientific Evidence

A. Acupuncture

a) Acupoints that have been found to be effective in improving cognitive function and reducing stress in human and animal models: ST-36, GV-14, GV-20, HT-7, HT-9, PC-9, CV-6, CV-12, CV-17, SP-10, BL-23, GB-34, LIV-3, KID-1, LU-11, Ex-HN-3, LI-20[11,12,14,15]

b) These acupoints improves cognitive function through[11,12,14,15]
- Improving cerebral blood flow
- Promoting cholinergic neural transmission
- Facilitating dopaminergic synaptic transmission
- Enhancing neurotrophin signaling and nerve growth factor in the hippocampus
- Protecting cerebral neurons from apoptosis and oxidative damages
- Regulating glucose metabolism
- Reducing the expression of microglia in the hippocampus
- Suppressing acetylcholinesterase in the hippocampus
- Decreasing the levels of extracellular amyloid b (Aβ) proteins in the hippocampus and relevant brain regions

c) In Alzheimer's human patients, brain fMRI demonstrates that acupuncture stimulation at HT-7, ST-36, ST-40, KID-3, LI-4, and LIV-3 increased in the activity in the temporal lobe (including hippocampus) and prefrontal lobe, which are related to the memory and cognitive function, as well as some regions of parietal lobe, and cerebellum.[14]

B. Chinese Herbal Medicine

a) Single Chinese Herb10,16-18

- Huperzia serrata (Qian Ceng Ta or Jin Bu Huan) is a potent, reversible, selective inhibitor of acetylcholinesterase (AChE). A systematic review and meta-analysis of randomized clinical trials demonstrated that Huperzine A from Huperzia serrata appears to have beneficial effects on improvement of cognitive function, daily living activity, and global clinical assessment in human patients with Alzheimer's disease
- Ginseng and Ginkgo biloba increase the uptake of choline in CNS, release acetylcholine from hippocampus, and reduce the level of Aβ
- Salvia miltiorrhiza (Dan Shen), Herba Erigerontis (Deng Zhan Hua), Radix Morinda Officinalis (Ba Ji Tian), Coptidis Rhizome (Huang Lian), Houttuyniae Herba (Yu Xing Cao), Uncaria rhynchophylla (Gou Teng), and Lycium barbarum (wolfberry; Gou Qi Zi) have been found to protect the brain against Aβ cell toxicity in different types of neuronal cells
- Wolfberry (wolfberry; Gou Qi Zi) was able to enhance the neuronal differentiation of the hippocampal neurogenesis and reverse the depression-like behavior caused by 50 mg/kg corticosterone injection

b) Chinese Herbal Formula16,19,20

- Liu Wei Di Huang Tang/Wan, at a dose of 100 mg/kg, was proved to improve cognitive function by promoting hippocampal neurogenesis in adult rats
- Jia Wei Wen Dan Tang at a dose of 50 mg/kg improved the cognitive functions via enhancing neurogenesis in the hippocampus of mice treated for 2 weeks
- Jia Wei Xiao Yao San was able to reverse the impaired neurogenesis in the hippocampus in stressed rats
- Bu Yang Huan Wu, 5g per Kg per day by mouth for 2 weeks, displayed a stimulating effect on neurogenesis and improved the neurological scores and functional recovery in stroke rats
- Ba Wei Di Huang Wan, 2g 3 times a day by mouth for 8 weeks, significant improved the cognitive function scores in human patients with dementia as compared to the placebo group
- Yi Gan San, 2.5g (1.5g of extract) 3 times a day by mouth for 4 weeks resulted in significant improvement of behavioral and psychological symptoms of dementia and activities of daily living in dementia patients as compared to the control group

V. Pattern Differentiation & Treatment

1) Phlegm Misting the Mind

- Signs: mental confusion and depression, lethargy, change of voice, vomiting of mucus, dull eyes. Tongue is pale to pink with thick sticky coating, may have swollen with teeth marks or a midline crack reaching the tip. Pulses are slippery with normal strength
- Treatment strategy: Resolve Phlegm, open the Heart orifice
- Acupuncture treatment: Common points (PC-5/6, HT-5/7, BL-14/15/44/45, GV-20, CV-15, Da-feng-men, Nao-shu, An-shen, LI-4, LIV-3); Add ST-40, BL-20, SP-6, CV-12 for Phlegm
- Herbal formula: Di Tan Tang, 0.5 g per 1020 lb body weight BID-TID

2) Phlegm Fire Misting the Mind

- Signs: Agitation or irritability, rash/manic behavior, aggressive, barking, thirsty, warm to the touch, cool seeking, mental depression, dull eyes. Tongue is red, the tip is redder or has red points, yellow sticky coating, maybe swollen with teeth marks. Pulses are full, rapid, slippery or wiry
- Treatment strategy: Resolve Phlegm, cool Fire, soothe the Liver
- Acupuncture treatment: Common points (PC-5/6, HT-5/7, BL-14/15/43/44, GV-20, CV-15, Da-feng-men, Nao-shu, An-shen, GB-20, LI-4, LIV-3); add ST-40, BL-20, SP-6, CV-12 for Phlegm; add PC-7, HT-8, GV-14, LIV-2, Wei-jian, Er-jian to clear Heat/Fire
- Herbal formula: Wen Dan Tang or Zen Xin San, 0.5 g per 1020 lb body weight BID-TID. Use Zhen Zin San in severe cases

3) Qi-Blood Stagnation

- Signs: Agitation or irritability, stop social interaction or become less responsive, poor memory, dislike of lying down, weak and cool limbs, wandering through the house, household accidents, getting lost in corners, step over anything, sleep less. Tongue is pale purple, or purple on the sides. Pulses are wiry or choppy
- Treatment strategy: Move Heart Qi-Blood, eliminate stagnation, open the Heart orifices
- Acupuncture treatment: Common points (PC-5/6, HT-5/7, BL-14/15/43/44, GV-20, CV-15, Da-feng-men, Nao-shu, An-shen, GB-20, LI-4, LIV-3); add LU-7, LI-4, LIV-3, CV-14, BL-21, LI-10, BL-17, SP-10 to move Qi-Blood and clear stagnation
- Herbal formula: Stasis in Mansion of Mind or Xue Fu Zhu Yu, 0.5 g per 1020 lb body weight BID-TID. Use Stasis in Mansion of Mind in severe cases

4) Liver Qi Stagnation

- Signs: Irritability/aggressiveness, depression, fluctuation of mental state, burping, nausea/vomiting, constipation to diarrhea, finicky/poor appetite, thirst, red conjunctiva, dislike to be touch on the thoracic flank region. Tongue is pink to red/lavender or red on the sides. Pulses are wiry, especially on left side

- Treatment strategy: Soothe Liver Qi, strengthen Spleen/Stomach, calm the mind

- Acupuncture treatment: Common points (PC-5/6, HT-5/7, BL-14/15/43/44, GV-20, CV-15, Da-feng-men, Nao-shu, An-shen, GB-20, LI-4, LIV-3); add BL-18/19, LIV-3/13/14, GB-3 to soothe the Liver Qi, and BL-20/21, LI-10 and ST-36 to strengthen Spleen and Stomach

- Herbal formula: Yi Gan San, Chai Hu Shu Gan, Liver Happy, or Xiao Yao San, 0.5 g per 1020 lb body weight BID-TID. Use Liver Happy in severe cases; Use Xiao Yao San if patients have digestive problems

5) Heart and Spleen Qi Deficiency

- Signs: Reduced responsiveness, stop any social interaction or become less responsive, become more aloof or fearful, poor memory, having indoor accidents, decreased appetite, lassitude, a desire to lie down, weakness of the limbs, sleep more, panting due to shortness of breathe, stare blankly at a roof, wall or air, signs often worsen during day time, fail to recognize their owners and friends at the end. Tongue is pale wet. Pulses are deep, weaker in the right side

- Treatment strategy: Tonify Heart and Spleen Qi, calm the Mind

- Acupuncture treatment: Common points (PC-5/6, HT-5/7, BL-14/15/43/44, GV-20, CV-15, Da-feng-men, Nao-shu, An-shen, GB-20, LI-4, LIV-3); add CV-6/17, BL-20/21, ST-36, SP-6, LI-10 to strengthen Qi

- Herbal formula: Yang Xin Tang, 0.5 g per 1020 lb body weight BID-TID. Add Si Jun Zi Tang or Liu Jun Zi Tang if there is decreased appetite

6) Heart Yin and Blood Deficiency

- Signs: Listlessness, anxiety, poor memory, reduced responsiveness, bark or abnormal behavior at night or late evening, pacing at home at night, sleep less at night, awake at night, having household accidents, cats may howl at night for no reason, signs often worsen during evening. Tongue is red or pale and dry. Pulses are deep and thin, weaker on left side

- Treatment strategy: Nourish Heart Yin and Blood, calm the Mind

- Acupuncture treatment: Common points (PC-5/6, HT-5/7, BL-14/15/43/44, GV-20, CV-15, Da-feng-men, Nao-shu, An-shen, GB-20, LI-4, LIV-3); Add CV-4, BL-17, BL-20, SP-10, KID-3/7, SP-6/9 to nourish Yin and Blood

- Herbal formula: Shen Calmer or Tian Wang Bu Xin Dan, 0.5 g per 1020 lb body weight BID-TID. Use She Calmer in severe cases

VI. Summary

Acupuncture and herbal medicine could serve as an effective, safe, well-tolerated and inexpensive form of care for dogs with CDS

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WSV18-0231

ORTHOPEDIC SURGERY (SIMULTANEOUS
TRANSLATION INTO MANDARIN CHINESE)

HOW TO EXPLORE THE STIFLE JOINT: TIPS, TRICKS, AND ALL YOU NEED TO KNOW

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Learning Objectives

At the end of this session you will be able to:

- Identify the instruments that simplify stifle joint exploration and how to use them
- Recognise differences between the medial and lateral menisci
- Identify the location of a meniscal injury

The keys to simplifying stifle joint surgery are an understanding of:

- the **surgical anatomy of the stifle joint** – familiarity with surgical anatomy aids confidence, intraoperative decision-making and simplifies surgery overall
- the **preparation, positioning and intraoperative manipulation of the stifle joint** that will simplify stifle joint exposure and maximize visualization
- the **equipment that will simplify exposure**
- **how to perform a stifle joint arthrotomy** and effectively examine the joint structures
- **common surgical procedures** involving the stifle joint

Preparation and positioning

Surgical preparation and draping are most easily performed with the limb suspended with adhesive tape from a drip stand or roof bolt. The limb should be clipped circumferentially from the proximal thigh to just proximal to the hock joint for most stifle joint surgeries. After routine surgical preparation the animal can be moved into the operating room with the limb still suspended.

Positioning the animal in **dorsal recumbency** simplifies stifle joint exploration. "Free draping" rather than field draping will allow the surgeon to have maximum vision and intraoperative manipulation of the stifle joint.

It is important that the final drape layer is of waterproof material as lavage is necessary in most stifle joint surgeries. Non-waterproof drapes will increase the risk of bacterial contamination through strike-through.

Equipment for effective stifle joint exploration includes:

- 1 Gelpi self-retaining retractor
- 1 stifle joint distractor (or alternatively a narrow-bladed 10-12mm Hohmann retractor
- 1 sharp-pointed Senn retractor
- 1 meniscal probe
- 1 mosquito haemostat or meniscal forceps
- Frazier suction tip (#6 or 8) and suction
- effective surgical lighting
- sterile lavage solution

Surgical approach for stifle joint exploration

The specifics of the various surgical approaches to the stifle joint are beyond the scope of this session. Arthroscopy has been shown to provide superior meniscal detail, greater sensitivity in detecting meniscal damage, reduced patient morbidity and a significantly lower occurrence of late or subsequent meniscal injury than for arthrotomy.

This session will focus on arthrotomy. Both the lateral and medial approaches have relative advantages and disadvantages. Both provide equally good vision and access to the menisci. Which side the surgeon chooses is personal preference and is usually determined by the type of cruciate stabilization surgery that is to be performed.

In both cases a "**mini arthrotomy**" rather than a full arthrotomy is completely adequate to provide adequate visualization to confirm the presence of cruciate disease and explore the menisci and adequate access for any necessary meniscal surgery.

The mini arthrotomy incision is from the level of the distal pole of the patella to the tibial plateau. Recognise that the infrapatellar fat pad is cranial to or **outside** the synovial part of the joint, so it is not necessary to incise through the fat pad. The incision through the synovial membrane runs immediately proximal to the fat pad and extends proximally to the level of the distal pole of the patella.

In a typical cruciate disease case there is chronic thickening of the synovial membrane and so the use of electrosurgery to control small arterial vessels in the thickened capsule in combination with suction is very useful.

A Gelpi retractor is inserted to transversely retract the capsular incision.

To properly inspect the menisci when cranial cruciate ligament rupture exists and the joint is unstable it is essential that distraction or cranial drawer of the tibia is achieved and maintained throughout the procedure.

Effective inspection of the menisci when **no cruciate instability** is present is not possible without arthroscopy. It should be noted that when **no instability exists** such as in early cases of cruciate disease it is very uncommon to have isolated meniscal injuries that are significant.

In these cases where there is **no instability** in either flexion or extension when examined under general anesthesia it is reasonable to assume that there is no significant meniscal injury. An arthrotomy (or arthroscopy) to confirm that early cruciate disease with partial tearing of the cranial cruciate ligament is present however would be necessary. Careful probing of the cranial cruciate ligament with the meniscal probe will usually identify torn cruciate fibres in these cases.

In cases where cruciate instability **is present** 2 methods exist to create and maintain distraction or cranial drawer of the tibia at arthrotomy.

The first method is the use of a self-retaining **stifle joint distractor**. The proximal tip of the distractor is placed in the proximal part of the intercondylar fossa. The intercondylar fossa is the origin of the caudal cruciate ligament so care should be taken when placing the proximal tip to avoid damage to the ligament.

The distal tip of the distractor is positioned caudal to and under the intermeniscal ligament. The intermeniscal ligament is not able to be visualized because it is covered by the fat pad. Gentle retraction of the Gelpi retractor distally helps retract the fat pad distally and provide better access to place the distal tip of the stifle distractor immediately caudal to the intermeniscal ligament. Alternatively, the Senn retractor can be used for this purpose.

The intermeniscal ligament lies immediately cranial to the insertion of the cranial cruciate ligament. Even in cases of complete cranial cruciate rupture the insertional end of the cranial cruciate is visible and can be used as a guide to find the intermeniscal ligament.

Once both tips of the distractor are in the correct location the joint is distracted. Provided the distal tip is correctly positioned caudal to and loading the intermeniscal ligament it will not pull out. If the distractor is pulling out distally it is because the tip has been placed into the fat pad instead of caudal to the ligament. Slight extension of the leg with the distractor in place will partly open the caudal aspect of the femoro-tibial joint and facilitate examination of the caudal horn of the menisci. The caudal horn of the medial meniscus is the area where the vast majority of significant meniscal injuries occur.

Various stifle distractors and sizes are available. A "speedlock" / "spinlock" locking mechanism is preferable to a ratchet locking system.

The stifle joint distractor simplifies exploration of the stifle joint in nearly all cases. Combined with a Gelpi retractor, meniscal probe and good lighting and suction it allows single-handed examination of the stifle joint.

The second method for creating effective distraction of the stifle joint to examine the menisci involves a combination of a narrow-bladed (12mm or less) **Hohmann retractor** and a sharp-pointed **Senn retractor** and necessitates a surgical assistant.

A Gelpi retractor is placed as previously described. The Senn retractor is placed into the infrapatella fat pad and the tibia pulled cranially.

The point of the Hohmann retractor is inserted through the intercondylar space of the femur and carefully hooked over the caudal aspect of the tibial plateau taking care not to damage the caudal cruciate ligament.

The Hohmann retractor is then used to lever the tibia cranially and the femur caudally by pushing the handle of the Hohmann retractor in a caudal direction against the femoral trochlea. Use of a narrow bladed Hohmann is necessary to avoid damaging the articular cartilage of the trochlea ridges.

Single-handed exploration is not possible with this method.

Relevant meniscal anatomy: what do you need to know to diagnose and treat meniscal injury?

The menisci are biconcave, C-shaped fibrocartilaginous discs with their open part directed towards the axis of the bone. The medial and lateral menisci are

remarkably different to each other.

In cross section the menisci are wedge-shaped being thickest on their convex abaxial border and thinnest on the concave axial border. The menisci are held in position by **6 meniscal ligaments.** To treat a meniscal tear to the caudal horn of the medial meniscus you will need to cut part or all of the caudal meniscotibial ligament. So..... you need to be very familiar with the meniscal anatomy if you are going to safely and effectively treat meniscal injuries in cruciate disease cases.

Both menisci are attached to the tibia by a **cranial and caudal meniscotibial ligament.** Each of these 4 ligaments is a short strong ligament on the axial or central end of each meniscus.

The menisci are attached to each other by an **intermeniscal ligament** that joins their cranial horns and lies immediately cranial to the tibial insertion of the cranial cruciate ligament.

The caudal horn of the lateral meniscus is also attached to the caudal part of the medial femoral condyle by the **meniscofemoral ligament of the lateral meniscus**. The medial meniscus lacks <u>any</u> femoral attachment.

The medial meniscus is also firmly secured abaxially or peripherally to the joint capsule and the medial collateral ligament. Conversely the lateral meniscus has <u>no</u> attachment to the lateral collateral ligament and has limited caudal capsular attachments, especially in the region of the popliteal tendon**.** It is only the cranial part of the lateral meniscus that has a firm capsular attachment.

It is this **difference in attachment of the menisci** that renders the medial meniscus less mobile than the lateral meniscus and explains the much higher incidence of damage to the medial meniscus.

Why are significant lateral meniscal injuries rare?

The lateral meniscus, because of its meniscofemoral ligament and minimal capsular attachments, moves **with** the lateral femoral condyle and is not subject to

significant abnormal shear forces after cranial cruciate rupture.

The medial meniscus however, being firmly attached to the tibial plateau, is subject to shear force when cranial cruciate instability exists. In this situation the medial femoral condyle moves caudally on flexion and cranially on extension of the stifle joint. This movement of the medial condyle is resisted by the caudal horn of the medial meniscus and subjects it to abnormal shear forces which ultimately results in damage. Because menisci function as stabilizing "shock absorbers" they are designed to take compressive loads but are unable to withstand shearing forces.

When using the meniscal probe to detect meniscal injuries (nearly all significant injuries occur to the caudal horn of the medial meniscus) the right-angled probe is turned flat to pass between the meniscus and the tibia. In a normal medial meniscus, the probe cannot be passed caudal to the caudal edge of the meniscus because it is attached to the capsule. The probe is turned with the tip facing dorsally and gentle traction on the caudal horn confirms no capsular tearing when there is no luxation of the horn cranially.

The probe tip is then used on the dorsal sloped surface of the caudal horn of the medial meniscus with the tip pointing down towards the meniscus. If a bucket handle (partial circumferential) tear is present the probe tip will drop into the tear and the torn piece can be dislodged.

Significant lateral meniscal injuries are rare. The same procedure with the meniscal probe can be used on the caudal horn of the lateral meniscus however this must be done in full recognition of the completely different attachment of the lateral to the medial meniscus.

It is normal to be able to pass the probe caudally between the ventral surface of the caudal horn of the lateral meniscus and the tibia because there is no tight capsular attachment as there is on the medial side.

Bucket handle tears can be identified in the same way as for a medial meniscus however.

Small radial tears of the cranial horn of the lateral meniscus are common however these are of little clinical significance.

There are 5 key points to remember about meniscal anatomy when you are doing meniscal surgery:

- **The medial and lateral menisci are <u>different.</u>**
- **The caudal meniscotibial ligament of the medial meniscus.** Very commonly either a part of this ligament or all of this ligament needs to be transected to remove either a partial tear or a complete caudal pole tear respectively. There is no capsular attachment at the area of the caudal meniscotibial ligament; the meniscal probe can pass freely dorsal and ventral to the ligament.

- **The menisco-<u>femoral</u> ligament of the lateral meniscus**. This is the largest of the meniscal ligaments and while normal is very different to the medial meniscus.
- **The medial meniscus is firmly attached to the tibial plateau.** The medial meniscus is firmly attached to the tibial plateau through peripheral attachments to the joint capsule and the medial collateral ligament. The lateral meniscus has no attachment to the lateral collateral ligament and has no caudal capsular attachments. Only the cranial third of the lateral meniscus has capsular attachments. It is this difference in attachment of the menisci that renders the medial meniscus less mobile than the lateral meniscus and explains the much higher incidence of damage to the medial meniscus in the cranial cruciate deficient stifle joint. When using a meniscal probe to inspect the menisci it is important to recognise that the capsule should be firmly attached to the entire periphery of the medial meniscus.
- **Normal menisci are gloss white.** Damaged menisci typically have a matt or roughened appearance and are discoloured.

Treatment of meniscal injury

Surgery is the treatment of choice for meniscal injury. Conservative treatment is not recommended due to the avascular nature of the majority of the meniscus and consequent lack of healing. Only the peripheral 10% - 15% of the meniscus has a significant blood supply. The remainder of the meniscus receives nutrition from the synovial fluid. Dogs with untreated meniscal injuries remain with significant lameness despite treatment of their cruciate disease.

It is important to assume a meniscal injury is present in all cruciate ruptures where instability is present <u>until proven otherwise</u> on exploratory

arthrotomy / arthroscopy. Meniscal injury has been shown to be present in dogs with cruciate instability in 30-60% of cases.

Meniscal injury is rare in dogs with early cruciate disease **before instability develops.** Early diagnosis of cruciate disease before instability develops is key to preventing meniscal damage and the problems associated with that.

Meniscal injury – treatment goals: Remove all of, **BUT ONLY THE DAMAGED PART** of the meniscus. Most commonly it is the caudal horn of the medial meniscus that is damaged.

There are three common types of meniscal injury;

- **bucket handle (partial circumferential) tears**
- **peripheral capsular detachment**
- **radial tears**

  All occur predominantly only in the caudal 1/3 of the medial meniscus between the medial collateral ligament and the caudal meniscotibial ligament.

  Bucket handle tears are the most common

meniscal injury. These are longitudinal tears (parallel to the circular orientation of the collagen fibres) named as the inner part of the meniscus is axially displaced resembling the handle of a bucket. Probing with a meniscal probe may be necessary to "unmask" a bucket handle tear. When probing it is important to remember that in the normal medial meniscus passage of the probe dorsally and ventrally over the meniscus will not cause damage or separation and that (other than at the area of the caudal meniscotibial ligament) the probe can not be passed caudally beyond the meniscus due to the close attachment of the joint capsule to the medial meniscus. (This is NOT the case with the lateral meniscus where the probe passes freely dorsally and ventrally). Multiple bucket handle tears can be present in the caudal horn and careful probing is necessary.

Peripheral capsular detachment is similar to a large bucket handle tear with complete detachment of the caudal pole of the meniscus from the joint capsule.

Radial or transverse tears are full thickness tears radiating from the inner concave (axial) border.

Partial meniscectomy (removal of only the damaged part of the meniscus) is strongly preferable to total meniscectomy.

Axial partial meniscectomy is the removal of the "bucket handle" part of a bucket handle tear. This leaves the periphery of the meniscus intact and, unless the bucket handle is very large, preserves some of the load bearing capacity of the meniscus.

Caudal pole hemimeniscectomy is the removal of the entire caudal pole of a detached medial meniscus. While this is necessary in cases where the entire caudal horn is detached it inactivates the shock absorbing capacity of the meniscus and increases the severity of subsequent osteoarthritis similar to total meniscectomy.

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GASTROENTEROLOGY / LIVER

GALL BLADDER MUCOCOELE: AN UPDATE

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Pathophysiology

A gall bladder mucocoele describes cystic mucinous hyperplasia of the gall bladder wall with accumulation of thick mucus. A mucocele can be subclinical and an incidental finding on diagnostic imaging but can cause obstruction of the biliary tract secondary to the thick mucus and when it becomes very large, it can also stretch the gall bladder wall and cause pressure necrosis and rupture.

Gall bladder mucoceles were first reported in dogs in 1995. They are uncommon but appear to be increasing in frequency.

The underlying cause of gall bladder mucoceles remains poorly understood, but there does appear to be a strong association with hypertriglyceridemia and hypercholesterolemia in dogs. Other proposed associations include biliary tract infection; disruption to gall bladder motility and drainage and genetic mutations. A very interesting recent study has suggested an association with neonicotinoid (imidacloprid) use particularly in Shelties – which is an attractive explanation for the apparent sudden appearance of this new disease. Studies repeatedly report an increased incidence in small breed dogs with a strong over-lap with breeds reported to suffer from familial hyperlipidemia: for example, Aquirre et al 2007 report gall bladder mucocele in 38 Shetland sheepdogs and Malek et al 2013 report gall bladder mucocele in 43 dogs including 10 cocker spaniels; 5 Shetland sheepdogs and 4 miniature schnauzers. A Japanese case-control study of gall bladder mucocele showed a significant association with increased cholesterol or triglycerides and miniature schnauzers were one of the breeds predisposed to mucocele.

Aguirre et al 2007 proposed an association between gall bladder mucocele and 'dyslipidemia' with an increase in serum triglycerides and/or cholesterol reported in all cases in which they were measured. Six out of 38 of their cases had hyperadrenocorticism, 5 had hypothyroidism and one had diabetes mellitus. Malek at el 2013 also reported dogs with hyperadrenocorticism, diabetes mellitus and hypothyroidism and a study of 30 dogs with gall bladder mucocele included seven dogs with hyperadrenocorticism and two dogs on long term steroid therapy. Another retrospective case-control study of gall bladder mucocele found a very significant association with hyperadrenocorticism but questioned

the association with hypothyroidism since many dogs were tested after the mucocele was diagnosed, introducing a significant source of bias. However, in a study of iatrogenic hyperadrenocorticism in 12 beagles, no increase in biliary sludge and no mucoceles were reported about 3 months of treatment with 8 mg/kg hydrocortisone every 12 hours.

There are plausible reasons why high serum lipids should predispose to gall bladder disease. Studies in prairie dogs fed high cholesterol diets have shown an increase in bile viscosity, a reduction in bile flow and gall bladder mucosal hyperplasia presumably due to the irritant effects of the bile acids in the thick, stationary bile. However, similar studies have not been reported in dogs and cholesterol and fat metabolism differ between species.

The pathophysiological relationship between hyperlipidemia and gall bladder mucocele therefore remains poorly understood, but evidence to date suggests that any dog with persistent hypertriglyceridaemia and/or hypercholesterolaemia should be investigated for clinically significant biliary tract disease.

Gall bladder mucoceles are rarely reported in cats. There are two single case reports in cats and one case report was a cat with concurrent hepatic lipidosis, which does suggest a potential association with hyperlipidemia in this species also, albeit rare.

Treatment

Clinically significant gall bladder mucocoeles should be treated and most authors recommend surgery for cholecystectomy. The prognosis is reasonable if the gall bladder has not ruptured and if biliary diversion surgery is not necessary. However, there is a real risk of perioperative mortality and bile leakage and cholecystectomy is generally considered to be a referral procedure. Successful medical management has been reported – particularly when the underlying cause is also identified and treated. Complete resolution has been reported in two hypothyroid dogs after supplementing thyroid hormones together with medical management. Medical management should only be attempted where there is no evidence of bile leakage or gall bladder wall thinning or necrosis on ultrasound. The owners should be warned of the potential risk of gall bladder rupture and the mucocoele should be regularly monitored for this with ultrasound. Medical treatment involves feeding a low fat diet and giving ursodeoxycholic acid and anti-oxidants.

It is unknown whether there is any benefit to medically managing asymptomatic mucocoeles. Surgical management would not be advisable in the absence of any clinical or clinicopathological abnormalities because of the risk of perioperative mortality. It is not known if asymptomatic mucocoeles progress nor if they do, how fast. It is also unknown if medical management will delay progression but the author uses ursodeoxycholic acid routinely in asymptomatic cases and monitors them with repeat ultrasound examinations every 2-4 months.

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43RD WORLD SMALL ANIMAL VETERINARY ASSOCIATION CONGRESS AND 9TH FASAVA CONGRESS

WSV18-0084

ROUTINE PYODERMA VS MRSP INFECTION IN THE DOG

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Introduction

Pyoderma is a pyogenic skin bacterial infection and the most common skin disease seen in dogs. **Staphylococcus pseudintermedius**is a commensal as well as the most frequent bacterial pathogen causing bacterial pyoderma in the dog

Underlying causes for bacterial pyoderma

Bacterial pyoderma is always secondary to an underlying cutaneous or systemic disease that disrupts the skin's defence mechanisms.

- Hypersensitivity dermatitis (adverse food reactions, atopic dermatitis, flea bite hypersensitivity)
- **Demodex spp, Sarcoptes scabiei,**fleas)
- Endocrinopathies: hypothyroidism, hyperadrenocorticism, diabetes mellitus
- Follicular dysplasia
- Keratinisation disorders (sebaceous adenitis, zinc responsive dermatosis)
- Malignancies

Diagnosis of bacterial pyoderma

Clinical lesions

- Pustules
- Crusts
- Epidermal collarettes
- Papules
- Focal to multifocal alopecia
- Erosions, ulcers
- Nodules and draining tracts

Infections due to methicillin resistant **Staphylococcus pseudintermedius**(MRSP) and methicillin susceptible **Staphylococcus pseudintermedius**(MSSP) look the same except MRSP infections do not respond to appropriately selected empirical antibiotic.

Cytology

A clinical diagnosis is made using a combination of consistent clinical lesions and cytological evidence of bacteria with suppurative inflammation i.e. degenerate neutrophils with intracellular cocci. Sampling techniques include direct and indirect (e.g. cotton buds) impression smears and adhesive tape cytology. The slides are stained with a Romanosky stain such as **Diff-Quik**, and examined under the microscope using the 100X oil immersion field.

Methicillin resistant *Staphylococcus pseudintermedius***(MRSP)**

MRSP infections are a major concern worldwide. MRSP infections are resistant to the antibiotic methicillin, which is used to deal with B lactamase producing staphylococci. Oxacillin is a replacement for methicillin in laboratories due to availability and stability. Cefoxitin is used to test for methicillin resistance in **Staphylococcus aureus**but is not reliable in diagnosis of MRSP.

Methicillin resistance is due to the mecA gene, which encodes for modified penicillin binding protein (pbp2a) that has low affinity for all B lactam antibiotics. The mecA gene is contained within a mobile genetic element called the Staphylococcal Cassette Chromosome mec (SCCmec), which can be transferred between and within staphylococci species. Horizontal gene transfer is frequent in **S. pseudintermedius**and multidrug resistant MRSP evolved rapidly through acquisition of a very limited number of mobile gene elements and mutations.

MRSP clones

Molecular testing using multilocus sequence typing (MLST) has allowed differentiation of MRSP into clones, which enables researchers to monitor the spread of MRSP worldwide. The major clones worldwide are the ST 71 in Europe, ST 68 in USA and ST45 in South East Asia.

Prevalence

The prevalence of MRSP in dogs varies geographically and on the studied populations. Published studies in Asia reported frequencies as high as 28% in Thailand and 66.5% in Japan. The prevalence of MRSP in healthy dogs is generally low.

Transmission

The suggested routes of transmission of **S. pseudintermedius**between dogs include vertical transmission from bitches to puppies (including milk) and horizontal transmission between dogs.

Risk factors for developing MRSP infections

- Increased number of antibacterial drug prescriptions
- Exposure to multiple drug classes (especially beta-lactams)
- Recent (30 days) antibiotic usage
- Misuse/excessive use of antibiotics
- Breeding dogs
- Hospitalisation/veterinary visits
- Concurrent immunomodulatory therapy
- Male? (Finland)

When to culture for MRSP? Modified from Hillier *et al*, **2014**
- Less than 50% improvement in lesions within 2 weeks of appropriate systemic antimicrobial therapy
- Appearance of new lesions consistent with bacterial pyoderma 2 or more weeks after starting appropriate systemic antimicrobial therapy
- Presence of residual lesions and cytological evidence of bacterial pyoderma after 6 weeks of appropriate systemic antimicrobial therapy
- Prior history of multidrug resistant infection in affected dog or from in contact dog in same household
- Recent history of hospitalisation
- Breeding dogs?

Bacterial culture collection and antibiotic susceptibility testing

A culture swab is used to collect samples from the surface and superficial lesions. Primary lesions such as intact pustules are preferred over secondary lesions such as epidermal collarettes and crusts. For deeper lesions such as nodules and draining tracts, the skin surface should be surgically prepared and a deep tissue biopsy collected for bacterial culture and antibiotic susceptibility testing.

Antimicrobial susceptibility testing is most commonly performed using the disk diffusion or dilution methods. While methicillin resistance is identified using oxacillin antibiotic testing, the gold standard to diagnose methicillin resistance is polymerase chain reaction (PCR) detection of mecA gene. When there is a clinical suspicion for MRSP, the author request for an extended panel of antibiotics including oxacillin. This is because some MRSP may show apparent **in vitro** susceptibilities to beta lactams i.e. cephalexin and amoxicillin-clavulanate acid but are ineffective **in vivo**.

Key steps for successful treatment of bacterial pyoderma
- Correct diagnosis of pyoderma
- Use topical antibacterial therapy and if necessary select an appropriate systemic antibiotic
- Correct antibiotic administration i.e. correct dose and frequency until clinical cure
- **Diagnosing and treating any underlying disease(s) causing bacterial pyoderma**

Treatment considerations

Topical antibacterial therapy

The skin lesions are readily accessible to topical therapy. Topical antibacterial therapy can be effective as a sole treatment against surface and superficial bacterial pyoderma regardless of methicillin susceptibility.

Benefits of topical antibacterial therapies
- May avoid the need for systemic antibiotics
- More rapid resolution of infection
- Reduce duration of systemic antibiotics required
- Break up biofilms, remove crusts, debris, bacteria and allergens from the skin
- Restore normal skin structure and function against infections
- Control against reinfections when underlying disease is being investigated and managed
- Reduce environmental contamination
- Reduce risk of transmission to other dogs and humans

Topical antibacterial options include 2-3% chlorhexidine, benzoyl peroxide, bleach (sodium hypochlorite), miconazole, fusidic acid, mupirocin, triclosan, bacitracin and polymixin B. They are available in shampoos, conditioners, lotions and sprays for more generalised infections, and creams, lotions, gels and wipes for more localised infections.

Systemic antibiotic

These are indicated for generalised, severe or deep bacterial pyoderma, if the dog is not amendable to topical therapy or owner unable to perform topical therapy. The results of antibiotic susceptibility testing guide the veterinarian in antibiotic selection. The final choice of antibiotic would depend on several factors including availability, safety, costs and patient factors (e.g. concurrent disease or drug therapy, drug reactions). Guidelines for selecting antibiotic and their doses have been published (Hillier **et al**, 2014) and will be discussed during the presentation.

Duration of treatment
- Superficial bacterial pyoderma: 3 weeks or 1 week beyond clinical resolution
- Deep bacterial pyoderma: 4-6 weeks or 2 weeks beyond clinical resolution
- If treatment duration is less than 3 weeks, then the patient should be examined prior to stopping antibiotics to ensure resolution of infection

Controlling against reinfections

Bacterial pyoderma is always secondary to underlying diseases. Veterinarians play an important role in identifying and managing these underlying diseases so that we can avoid reinfections, repeated antibiotic treatments and reduce risks of development of antibiotic resistance.

Prognosis

Fortunately, there is no difference in treatment outcomes between dogs treated for MSSP and MRSP with both groups having an overall good prognosis. Dogs can continue to carry MRSP for more than one year after clinical resolution of infection. These dogs can pose a risk to susceptible in contact animals (both dogs and cats) and humans, as well as contaminating the environment.

Transmission to in contact dogs and owners

Staphylococcus pseudintermedius do not typically colonise humans although we can betransient

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carriersespecially veterinary professionals or those in close contact with dogs. Dogs and their owners can harbour genetically identical strains of **S. pseudintermedius**including MRSP.

Veterinarians and owners should focus on reducing direct and indirect spread of MRSP through direct contact and contamination of environments respectively. It is reasonable to restrict MRSP infected dogs from contact with other dogs and humans, especially those who are immune-compromised, until they receive treatment and show clinical improvement. Veterinarians should also educate owners on the importance of hand hygiene and how to decontaminate the environment.

In dogs, MRSP carriage has been identified as a risk factor for developing surgical site infections after tibial plateau levelling osteotomy (TPLO).   Routine infection control practices are effective in controlling the potential transmission of staphylococci and MRSP between animals and to owners, veterinary and nursing staff. Personal protective equipment (PPE) prevents contamination of clothing and skin and subsequent transmission to other animals and staff.

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FASAVA/HILLS FELINE MEDICINE

LYMPHOMA IN CATS – COMMON CONUNDRUMS

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LYMPHOMA IN CATS: COMMON CONUNDRUMS

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Alimentary (gastrointestinal) lymphoma: The most important distinction is small cell/low grade (indolent clinical course) versus large cell/high or intermediate grade or large granular lymphoma (aggressive clinical course).

Conundrums

- Diagnosis of aggressive subtype versus small cell GI lymphoma: Generally, diagnosis of aggressive GI lymphoma is more straightforward than that of small cell GI lymphoma. Cytology of enlarged lymph nodes or intestinal masses is often diagnostic with surgical biopsies infrequently required. In small cell GI lymphoma, clinical presentation and results of diagnostic tests e.g. abdominal imaging, cytology and histopathology overlap with inflammatory bowel disease. Cytology cannot reliably diagnose small cell GI lymphoma because of the difficulty in distinguishing neoplastic lymphocytes from a normal/reactive population and because the typical diffuse mild to moderate intestinal wall thickening precludes effective sampling for cytology.

- Collection of biopsies in small cell GI lymphoma: The distal small intestine is commonly involved, and thus the diagnosis may be missed if only the stomach and duodenum are biopsied. For endoscopic biopsy collection, therefore, both upper (stomach and duodenum) and lower (colon and ileum) GI studies should be performed. The jejunum cannot be accessed in routine endoscopy procedures in small animals. Alternatively, exploratory laparotomy and full thickness biopsies of multiple GI sites can be performed. The major benefit of endoscopy is its relative non-invasiveness, but potential drawbacks compared to exploratory laparotomy include inability to assess other abdominal organs or the serosal surface of the GI tract, inability to access the jejunum and higher risk of suboptimal biopsies (e.g. due to superficial sampling or crush artefact) hampering diagnosis. Full thickness intestinal biopsies carry a risk of dehiscence and subsequent septic peritonitis, though this does not appear to be necessarily increased in the setting of alimentary lymphoma specifically (1).

- Additional tests on biopsies: No matter the method of biopsy, histopathological diagnosis of small cell GI lymphoma can be challenging. Neoplastic lymphocytes cannot be distinguished from inflammatory infiltrate on the basis of morphology

alone. Criteria suggestive for lymphoma over IBD include a more monomorphic lymphocyte population, specific patterns of epitheliotropism and/or lamina propria invasion, extension deep to the mucosa, and intravascular infiltration. In addition to routine H&E, immunophenotyping with immunohistochemistry (IHC) and assessment of clonality via PCR (PARR) can assist in making a final diagnosis of lymphoma. In one study (2), approximately 50% of cases that were initially diagnosed as IBD on H&E alone were re-classified as T cell lymphoma when IHC and PARR were assessed. Although less common, misdiagnosis of IBD as lymphoma can also occur. This study suggested a diagnostic algorithm for these cases - if initial histopathology diagnoses IBD but there is poor response to treatment then IHC +/- PARR should be pursued for definitive diagnosis. If initial biopsy is suspicious for lymphoma then IHC should be performed +/- PARR, if IHC is suspicious but not diagnostic for lymphoma. If initial biopsy diagnoses lymphoma, IHC is still recommended for immunophenotyping as prognosis may differ for B versus T cell disease, and because of the small risk of false positive lymphoma diagnosis. PARR should not be relied upon alone for diagnosis as sensitivity in cats can vary (as low as 60-70%, especially with B cell lymphoma) therefore a negative result cannot conclusively rule out lymphoma.

- How much does the distinction of IBD versus small cell lymphoma matter? Prognosis for IBD is better than small cell lymphoma, and a definitive diagnosis should always be pursued. However, in cases where IBD or small cell lymphoma is strongly suspected based on initial diagnostic tests, and histopathology is either not definitive or is declined by owners, initiating treatment directed at IBD and escalating if response is inadequate (e.g. diet and B12 supplementation if indicated ➡ prednisolone ➡ chlorambucil) may be, in my opinion, very reasonable. Distinguishing between aggressive GI lymphoma and small cell lymphoma is of vital importance as the treatment options and prognosis are dramatically different.

- Does a prior diagnosis of IBD increase the risk of developing lymphoma in the future? In cats with a previous diagnosis of IBD who are later diagnosed with small cell lymphoma, it is unclear whether 1) IBD has 'transformed' into lymphoma or the chronic inflammation has potentiated the development of lymphoma or 2) the initial diagnosis was incorrect and the cat had lymphoma all along. In people, inflammatory bowel disease is strongly associated with an increased risk of colorectal cancer. These cancers are typically carcinomas, although there may be an increased risk of lymphoma as well. Chronic immunosuppression may also play a role in cancer risk in these patients.

Nasal lymphoma: Lymphoma is the most common intranasal tumour in cats. Clinical signs do not specifically distinguish it from other intranasal diseases. In most cats, it is localised to the nasal cavity at diagnosis, though systemic involvement can occur. Advanced imaging (CT or MRI) and biopsies for histopathology are recommended for diagnosis - while cytology may be diagnostic, false negatives are common due to concurrent inflammation. Radiation therapy is the treatment of choice for localised disease. Complete staging including regional lymph nodes, thoracic and abdominal imaging and ideally bone marrow assessment, is recommended if radiation therapy is to be pursued.

Conundrums:

- Definitive diagnosis: IHC is the most reliable way to distinguish nasal lymphoma from carcinoma (the second most common feline intranasal tumour) and should be considered for definitive diagnosis in all feline nasal tumours (3). Although the recommended treatment for both is radiation therapy and the error rate of routine H&E is not especially high, lymphoma has a higher risk of systemic involvement and therefore more extensive staging is indicated prior to treatment, and the prognoses differ (generally longer survival time with lymphoma than carcinoma). IHC on feline nasal tumours, especially those diagnosed as carcinoma on H&E, would therefore seem prudent.

- Treatment options: Although radiation is standard of care, it is not widely available. In that case, chemotherapy can be effective. The majority of cats will respond to COP/CHOP chemotherapy and extended survivals can be seen, especially in those achieving a complete response (approximately 1-2 years) (4).

- Combining radiation with chemotherapy: Given the risk of systemic disease and the effectiveness of chemotherapy, it is tempting to treat with both radiation therapy and chemotherapy. In one study using radiation therapy and chemotherapy for cats with localised nasal lymphoma, the median survival time was approximately 2.5 years, longer than that published for either treatment alone (5). The one study comparing cats treated with radiation therapy, chemotherapy, or both, for nasal lymphoma did not show a statistically significant difference between the treatments, though when cats receiving any radiation therapy were compared to cats receiving chemotherapy alone, radiation therapy was found to improve overall survival time. Again, improved survival was seen in cats with a complete response to treatment (6).

Peripheral nodal lymphoma: Lymphoma limited to the peripheral nodes represents < 10% of all feline lymphoma cases. Nodal lymphoma in cats includes both small and large cell variants, and there is a specific subtype referred to as "Hodgkin's-like" which typically involves one or more of the lymph nodes of the head and neck.

Conundrums:

- Diagnosis: Unlikely canine nodal lymphoma, cytology alone is often insufficient for diagnosis and histopathology is often required. IHC should be pursued to assist in diagnosis, especially of Hodgkin's-like lymphoma and in any equivocal cases. The histological designation 'T cell rich B cell lymphoma' is used interchangeably with Hodgkin's-like lymphoma by some pathologists, but the clinical presentation (single or regional lymph nodes, usually not disseminated disease) must be considered to diagnose Hodgkin's-like lymphoma and choose appropriate treatment. I have seen a case of a drug reaction in a cat with marked peripheral lymphadenopathy that was diagnosed as emerging large B cell lymphoma on H&E, however IHC showed reactive hyperplasia. The inciting drug was discontinued with rapid resolution of lymphadenopathy.

- Treatment: In general, although data is lacking, nodal lymphoma in cats is treated as for dogs i.e. depending on whether it is a large or small cell variant. In the specific case of Hodgkin's-like lymphoma, surgery is often pursued as an initial treatment, but whether or not chemotherapy should be used at all, as an adjuvant treatment, after progression, or instead of surgery is undefined.

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OPHTHALMOLOGY

STATE-OF-THE-ART LECTURE FELINE HERPESVIRUS: CONTROL AND THERAPY

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Clinical Presentations

Feline herpesvirus is a ubiquitous virus that varies very little worldwide with respect to their clinical virulence. And yet, we see a huge range of clinical signs in cats infected with this virus. There are probably a large number of reasons for this; however principle among these is likely the host's response to this virus. FHV-1-naïve kittens infected in the first few weeks of life against a backdrop waning maternal immunity almost inevitably get severe upper respiratory and ocular disease with high morbidity but rare mortality. By contrast, adult cats can undergo viral reactivation with viral shedding and can infect in-contact cats; all without demonstrating clinical signs themselves. These two scenarios represent just the two extremes of infection; within your clinic you see cats with a huge diversity of clinical signs in between. For this reason, I like to consider clinical signs associated with FHV-1 under one of three broad categories: primary infection, recrudescent infections, and FHV-1-associated syndromes.

Primary Herpetic Disease

Primary ocular FHV-1 infection is characterized by blepharospasm, conjunctival hyperemia, serous ocular discharge that becomes purulent by day 5-7 of infection, mild to moderate conjunctival swelling, and often conjunctival ulcers. Corneal involvement is not reliable; however some cats develop corneal ulcers which are transiently dendritic at the very earliest phase only. These dendrites quickly coalesce to become geographic ulcers. The ocular signs are seen in association with typical signs of upper respiratory infection. The uncomplicated clinical course is typically 10-14 days; however it is critical to realize that almost all cats become latently infected within ganglia for life. Reactivation from latency is likely in at least 50% of cats, sometimes with viral shedding.

Recrudescent FHV-1 Syndromes

Despite the frequency with which latently infected cats undergo viral reactivation at the ganglia and viral shedding at peripheral epithelial sites, recrudescent disease occurs in a minority of these. Further, disease severity and tissue involvement can range very widely between individuals and even between episodes in the same cat. Recrudescent conjunctivitis is usually milder than in acute infections, but can become chronic and "smoldering". Although recrudescent conjunctivitis is

usually nonulcerative, substantial conjunctival thickening and hyperemia can occur secondary to inflammatory cell infiltration. Corneal involvement is relatively frequent in recrudescent disease compared to primary infection and may involve the corneal epithelium or stroma. With epithelial involvement, dendritic and later geographic corneal ulceration may be seen just as in primary infections. Corneal stromal disease is typically immunopathological (i.e., immune-mediated, but not necessarily autoimmune) in origin and includes stromal neovascularization, edema, stromal cell infiltration, and ultimately fibrosis usually under an intact epithelium. Consensus has not been reached regarding the antigens responsible for the subepithelial immunological response within cornea and/or conjunctiva. Some believe the process is driven by viral antigens, while others are suspicious that altered self-antigens are the focus of the immunological response.

FHV-1-Associated Disease Syndromes

The following diseases have been associated with detection of FHV-1 in affected tissues; however the causative role of the virus in each syndrome has been variably proven.

Symblepharon. There is little question that symblepharon can be a sequela to severe primary FHV-1 infection. It is commonly seen in young animals, and presumably occurs as a result of widespread ulceration with exposure of the conjunctival substantia propria and sometimes also the corneal stroma. FHV-1 is almost certainly the predominant cause of symblepharon formation in cats and other infectious agents are unlikely to cause symblepharon formation.

Corneal sequestration. Experimentally, FHV-1 inoculation (in cats receiving corticosteroids) can result in corneal sequestration. However, the prevalence of detectable FHV-1 in samples collected from cats with sequestra has varied widely in the clinical setting and the link between FHV-1 and sequestra has not been shown to be causative. It seems likely that sequestration is a non-specific response to stromal exposure or damage and that FHV-1 is just one possible cause of this disease. This is borne out in a study by Nasisse et al who reported identification of FHV-1 DNA in 86 of 156 (55%) of sequestra analyzed (compared with only 6% of clinically normal corneas). A lower prevalence of FHV-1 DNA was found in corneas of Persian and Himalayan cats with sequestration, suggesting that other non-viral causes of sequestration are more likely to be operative in these breeds.

Eosinophilic keratitis. Prior clinical studies have suggested a link between FHV-1 infection and eosinophilic keratitis. PCR testing of corneal scrapings from cats with cytology-confirmed eosinophilic keratitis has revealed 76% (45/59) of cases to be FHV-1 positive.

However, PCR performed on tears collected onto a STT was negative in 10 cats with cytologically proven eosinophilic keratitis. As with corneal sequestra, the role of the virus in the initiation or exacerbation of this disease has not been determined; however anecdotally some patients with this syndrome improve with antiviral therapy alone.

Dermatitis. Periodically, FHV-1 has been identified as a cause of dermatological lesions, particularly those surrounding the eyes and involving nasal skin of domestic and wild felidae. This is not surprising when one considers the marked epithelial tropism of this virus and the reliability with which HSV-1 causes dermal lesions. We have recently examined the diagnostic utility of FHV-1 PCR for this disease. FHV-1 DNA was detected in all 9 biopsy specimens from 5 cats with herpetic dermatitis but in 1 of 17 biopsy specimens from the 14 cats with nonherpetic dermatitis, and was not detected in any of the 21 biopsy specimens from the 8 cats without dermatitis. This is in sharp contrast to the use of this technique in ocular tissues where the extent of viral shedding in normal animals dramatically reduces the sensitivity of a positive test in affected animals. When results of histologic examination were used as the gold standard in this study of cats with dermatitis, sensitivity and specificity of the PCR assay were 100% and 95%, respectively. We concluded that FHV-1 DNA can be detected in the skin of cats with herpetic dermatitis, that the virus may play a causative role in the disease, and that this PCR assay may be useful in confirming a diagnosis of herpetic dermatitis.

Diagnosing Cats with Keratoconjunctivitis

One of my least favorite questions is "What is the best laboratory test for cats with corneal or conjunctival disease?". In reality there is not one. Explaining this position requires an understanding of an essential fact about feline herpesvirus (FHV-1) - clinically normal cats (and lots of them) can shed FHV-1 at their ocular surface. Because PCR is more sensitive than IFA or VI, this assay exacerbates this problem. In fact, in some humane shelter-based populations, about **half** of all normal cats are shedding FHV-1 DNA as determined by PCR. Therefore, in some circumstances, the number of false positive test results we can expect is extraordinarily high and we may be better to flip a coin than to run that PCR assay! Given the predictably high rate of false positive (particularly with serology and PCR) and negative test results (particularly with VI and IFA), I now no longer conduct laboratory tests for FHV-1 or **Chlamydia felis** (previously **Chlamydia psittaci** and, before that, **Chlamydophila felis**) in individual cats with keratoconjunctivitis. Rather, I resort to good old fashioned clinical acumen. My diagnostic "tests" now are (i) the history and clinical exam findings followed by (ii) response to therapy. This requires acceptance

of a couple of critical facts: first I have to be willing to be wrong when making an educated guess regarding the etiological diagnosis and, second, I have to use the absolute best therapeutic trial and demand excellent owner compliance in executing that trial.

Diagnosing Keratoconjunctivitis Using Clinical Signs as Your Guide

Using clinical signs of surface ocular disease as a "diagnostic assay" requires a philosophical approach that I liken to adding pebbles to one of two sides of an old-fashioned scale or balance. I start with the paradigm that feline keratoconjunctivitis is infectious till proven otherwise and that by far and away the most commonly implicated infectious organisms are FHV-1 and **Chlamydia felis**. I then consider the clinical signs outlined in the table. Using each feature as a discerning feature I aim to place one of my "diagnostic pebbles" on the herpetic or chlamydial sides of the balance, thereby making a clinical judgment at the end of the examination as to which of these 2 organisms is more likely to be the cause of the disease seen.

Clinical Signs	FHV-1	C. felis
Conjunctival hyperemia	+++	+
Chemosis	+	+++
Conjunctival ulceration	+/-	-
Keratitis	+/-	-
Dendrites	Pathognomonic	-
Respiratory signs/malaise	++	+/-

Note that both agents cause some of the signs and that it is a weighted assessment. This introduces a notable element of subjectivity into the assessment. I unashamedly tell clients this and explain that I still believe that this is better than wasting their money on a laboratory test. I also use this time to introduce the concept that the clients themselves will form the critical next step in the diagnostic process – "response to therapy". We will discuss this more fully in the next session.

Antiviral Therapy

If we are to use response to therapy as a "diagnostic test", then we must choose the optimum therapeutic approach possible for each cat. Although a large variety of antiviral agents exists for oral or topical treatment of cats infected with feline herpesvirus type 1 (FHV-1), some general comments regarding these agents are possible:

- No antiviral agent has been developed for FHV-1; although many have been tested for efficacy against this virus. Agents highly effective against closely-related human herpesviruses are not necessarily or predictably effective against FHV-1 and all should be tested in vitro before they are administered to cats.
- No antiviral agent has been developed for cats;

although some have been tested for safety in this species. Agents with a reasonable safety profile in humans are not always or predictably non-toxic when administered to cats and all require safety and efficacy testing in vivo.
- Many antiviral agents require host metabolism before achieving their active form. These agents are not reliably or predictably metabolized by cats and pharmacokinetic studies in cats are required.
- Antiviral agents tend to be more toxic than do antibacterial agents since viruses are obligate intracellular organisms and co-opt or have close analogues of the host's cellular "machinery". This limits many antiviral agents to topical (ophthalmic) rather than systemic use.
- All antiviral agents currently used for cats infected with FHV-1 are virostatic. Therefore, they typically require frequent administration to be effective.

The following antiviral agents have been studied to varying degrees for their efficacy against FHV-1, their pharmacokinetics in cats, and/or their safety and efficacy in treating cats infected with FHV-1.

Trifluridine (TFU or trifluorothymidine) is too toxic to be administered systemically but topically administered trifluridine is considered one of the most effective drugs for treating HSV-1 keratitis. This is in part due to its superior corneal epithelial penetration. It is also one of the more potent antiviral drugs for FHV-1. It is commercially available in the USA as a 1% ophthalmic solution that should be applied to the affected eye 5-6 times daily. Unfortunately, it is expensive and is often not well tolerated by cats, presumably due to a stinging reaction reported in humans.

Idoxuridine (IDU) is a nonspecific inhibitor of DNA synthesis, affecting any process requiring thymidine. Therefore, host cells are similarly affected, systemic therapy is not possible, and corneal toxicity can occur. It has been used as an ophthalmic 0.1% solution or 0.5% ointment. This drug is reasonably well tolerated by most cats and seems efficacious in many. It is no longer commercially available in the USA but can be obtained from a compounding pharmacist. It should be applied to the affected eye 5-6 times daily.

Vidarabine (VDB) interferes with DNA polymerase and, like idoxuridine, is non-selective in its effect and so is associated with notable host toxicity if administered systemically. Because it affects a viral replication step different from that targeted by idoxuridine, vidarabine may be effective in patients whose disease seems resistant to idoxuridine. As a 3% ophthalmic ointment, vidarabine often appears to be better tolerated than many of the antiviral solutions. Where it is not available commercially, it can be obtained from a compounding pharmacist. Like idoxuridine, it should be applied to the affected eye 5-6 times daily.

Acyclovir (ACV) has relatively low antiviral potency against FHV-1, poor bioavailability, and is potentially toxic when systemically administered to cats. Oral administration of 50 mg/kg acyclovir to cats was associated with peak plasma levels of only approximately one third required for this virus. Common signs of toxicity are referable to bone marrow suppression. However, acyclovir is also available as a 3% ophthalmic ointment in some countries. In one study in which a 0.5% ointment was used 5 times daily, the median time to resolution of clinical signs was 10 days. Cats treated only 3 times daily took approximately twice as long to resolve and did so only once therapy was increased to 5 times daily. Taken together, these data suggest that very frequent topical application of acyclovir may produce concentrations at the corneal surface that do exceed the reported concentration required for this virus but are not associated with toxicity. There are also in vitro data suggesting that interferon exerts a synergistic effect with acyclovir that could permit an approximately 8-fold reduction in acyclovir dose. In vivo investigation and validation of these data are needed.

Valacyclovir (VCV) is a prodrug of acyclovir that, in humans and cats, is more efficiently absorbed from the gastrointestinal tract compared with acyclovir and is converted to acyclovir by a hepatic hydrolase. Its safety and efficacy have been studied in cats. Plasma concentrations of acyclovir that surpass the IC_{50} for FHV-1 can be achieved after oral administration of this drug. However, in cats experimentally infected with FHV-1, valacyclovir induced fatal hepatic and renal necrosis, along with bone marrow suppression, and did not reduce viral shedding or clinical disease severity. Therefore, despite its superior pharmacokinetics, valacyclovir should never be used in cats.

Ganciclovir (GCV) appears to be at least 10-fold more effective against FHV-1 compared with acyclovir. It is available for systemic (IV or PO) and intravitreal administration in humans, where it is associated with greater toxicity than acyclovir. Toxicity is typically evident as bone marrow suppression. It has been released as a new topical antiviral gel in humans. There are no reports of its safety or efficacy in cats as a systemic or topical agent, although anecdotal reports from Europe (where it is much less expensive) are very promising.

Famciclovir (Famvir® and generic) is a prodrug of penciclovir; however metabolism of famciclovir to penciclovir in humans is complex; requiring di-deacetylation, in the blood, liver, or small intestine, and subsequent oxidation to penciclovir by aldehyde oxidase in the liver. Unfortunately, hepatic aldehyde oxidase activity is nearly absent in cats. This has necessitated cautious extrapolation to cats of data generated in humans. Indeed data to date suggest that famciclovir and penciclovir pharmacokinetics in the cat are

extremely complex and likely nonlinear. For example, an approximately 6-fold increase in dose produced only an approximately 3-fold increase in plasma concentration. However, in a masked, prospective, placebo-controlled study of efficacy, experimentally infected cats receiving 90 mg/kg famciclovir TID had significantly reduced clinical signs, serum globulin concentrations, histologic evidence of conjunctivitis, viral shedding, and serum FHV-1 titers, as well as increased goblet cell density. Importantly, no important adverse clinical, hematologic or biochemical changes were associated with famciclovir administration. More recently, we have shown that 90 mg/kg PO BID produces almost identical plasma and tear concentrations as did the TID dose that was so successful. Do not compound, do not taper the dose when seeing improvement. Rather, treat beyond clinical resolution and then stop.

Cidofovir (CDV) is commercially available only in injectable form in the USA but has been studied as a 0.5% solution applied topically twice daily to cats experimentally infected with FHV-1. Its use in these cats was associated with reduced viral shedding and clinical disease. Its efficacy at only twice daily (despite being virostatic) is believed to be due to the long tissue half-lives of the metabolites of this drug. There are occasional reports of its experimental topical use in humans being associated with stenosis of the nasolacrimal drainage system components and, as yet, it is not commercially available as an ophthalmic agent in humans. Therefore, at this stage there are insufficient data to support its long term safety as a topical agent in cats.

Lysine

The literature regarding lysine has become very interesting recently with some data that at first glance appear contrary to earlier study outcomes which suggested efficacy. This requires a more detailed assessment.

Lysine limits the in vitro replication of many viruses, including FHV-1. The antiviral mechanism is unknown; however, many investigators have demonstrated that concurrent depletion of arginine is essential for lysine supplementation to be effective. This finding suggests that lysine exerts its antiviral effect by antagonism of arginine. Meanwhile, results of 2 early independent in vivo studies have supported the clinical use of l-lysine in cats. Lysine-treated cats undergoing primary herpetic disease had significantly less severe conjunctivitis than cats that received placebo, while latently infected adult cats receiving lysine had reduced viral shedding. In both studies, plasma arginine concentrations remained in the normal range, and no signs of toxicity were observed, despite notably elevated plasma lysine concentrations in treated cats. A subsequent study examined the effects of lysine in 144 shelter cats receiving oral boluses of 250 mg (kittens) or 500 mg (adult cats) of lysine once daily

for the duration of their stay. No significant treatment effect was detected on the incidence of infectious upper respiratory disease (IURD), the need for antimicrobial treatment for IURD, or the interval from admission to onset of IURD. A subsequent pair of studies assessed the safety and efficacy of l-lysine incorporated into cat food. Perhaps not unexpectedly, food (and therefore lysine) intake decreased coincident with peak disease and viral presence. As a result, cats did not receive lysine at the very time they needed it most. Surprisingly though, clinical signs and viral shedding in cats fed the supplemented ration were worse than in cats fed the basal diet.

Taking all of this into account, I administer 500 mg lysine per os q 12 hours **therapeutically** at the time of recrudescent disease and encourage owners of cats that have frequent recurrences to administer this same dose over the long term as a **prophylactic** measure. More recently I have strongly recommended that client-owned animals receive lysine as a twice daily bolus; not sprinkled on food.

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SVA REHABILITATION

INTRODUCTION TO THERAPEUTIC EXERCISE

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INTRODUCTION TO THERAPEUTIC EXERCISE

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The rehabilitation therapist assesses the patient by gathering subjective data from the client, medical history from the referring veterinarian, and objective data from an in-depth physical examination that includes assessment of posture, gait, strength, flexibility, AROM, PROM, and neurologic function. From this data, a problem list is created and an assessment narrative is written. The treatment plan addresses each of the items on the problem list and includes manual therapies, physical modalities, and therapeutic exercises.

Therapeutic exercise plans are based upon the weight-bearing status of the patient. Weight bearing is classified as: Non-Weight Bearing (NWB), Partial Weight Bearing (PWB), and Full Weight Bearing (FWB). Animals that are FWB are then classified by strength: Minimal (Less than 3 on a scale of 5, shown here as <3/5), Moderate (3 out of 5, shown here as 3/5), and Good (Greater than 3 out of 5, shown here as >3/5). Exercise plans for each of these weight-bearing statuses will include work on Proprioception, Strength, Flexibility, and Endurance. The therapist must then focus upon the structures being treated: the muscles. There are two basic muscle types: Joint stabilizer muscles and Mobilizer muscles. Stabilizers tend to be short bellies with short excursion length. They tend is sit deep to the mobilizers and insert close to the joint. These muscles tend to have more Type 1 muscle fibers. Mobilizers have longer bellies and tendons, sit more superficially in the limb, have more Type 2 muscle fibers, and are more frequently diagnosed with strain injuries.Patient evaluation requires a thorough examination to assess for areas of pain and/or weakness. The patient's emotional and intellectual abilities must be assessed as well, as this will determine the types and intensities of treatment plans that will be optimal. With all information gathered, the therapist will create both short-term and long-term goals for the patient, based upon age, injury acuity, weight bearing status, and client's goals.When designing a therapeutic

exercise plan, many variables must be controlled. These include: Frequency, Intensity, Duration, Environment, and Degree of impact. These variables are then applied to each type of exercise (proprioception, strength, flexibility, and endurance). At the start of each exercise session, the patient is first trained how to complete the desired movement. The correct movement is then demonstrated to the client, who is then asked to complete the movement with the patient. The therapist must be aware of possible avenues for the patient to avoid doing the correct movement and be prepared to prevent or correct each while instructing the client how to observe these actions.There are some basic rules that should be observed when carrying out an exercise program:

1) Always start within the patient's comfort zone. Any exercise that causes pain will no longer be a viable option for the anxious patient. Intensity of the exercise is gradually increased until the short-term goals are met. Once the patient is able to easily complete the prescribed exercise, the frequency, intensity, duration, environment or degree of impact can be increased.
2) Strength work should be done 3 to 5 days per week with days off alternating with work days. For the client that insists on training daily, the plan will work on thoracic limbs one day, core the next, and pelvic limbs the next.

3) All patients have a daily stretching routine after working out.Therapeutic exercise equipment for dogs includes physioballs, therapy bands, rocker/wobble boards, inflatable discs, cavaletti poles, and treadmills. Cavalettis were introduced from equine training and are used to enhance proprioception, strengthen flexor muscles, and increase stride length. Land treadmills are popular today. The concern when working canine patients on human treadmills is that the treads are relatively short (1.5m). For breeds larger than border collies, the tread needs to be at least 2m long. A short tread can lead to a shortened/altered gait that persists when off of the tread. This can have very detrimental effects on working dogs. There are several companies manufacturing canine-specific treadmills.Inflatable discs are very valuable tools. They are used as low unstable surfaces when working on proprioception and balance. Rocker/wobble boards come with interchangeable devices on the underside that allow the therapist to convert from uni-directional rocking to multi-directional wobbling.As stated above, exercises are developed to address proprioception, speed, strength, and endurance. Proprioception can be addressed using cavaletti poles in patterns on the ground, weave poles, inflatable discs, and rocker/wobble boards. Once the patient is proficient in one activity, the exercises can be combined into a circuit. The goal for speed work is to increase stride length, efficiency and burst speed. Cavaletti poles are used to increase stride length and efficiency, while interval training is used to increase burst speed. The goal for endurance work is to increase the duration

and intensity of exercise as fitness improves. Here, the therapist must introduce cross training to prevent overuse injury or patient boredom.Unilateral or focal strengthening exercises are beneficial for patients who have gait asymmetry, focal atrophy, or a regional issue such as poor core tone. Two of our favorite exercises for this category include the Sit>Beg>Stand>Beg>Sit exercise and the "Commando Crawl". The first is a great core strengthener. The patient is taught to sit squarely, then is trained to sit up ('beg'). Once the patient is capable of holding this posture, even with mild to moderate perturbations by the therapist, the next step is added: The patient is encouraged to transition from the beg posture to standing upright on the pelvic limbs. The therapist must be ready to keep the patient's focus sufficiently up and toward the back to prevent him from dropping down into a stand position. From this posture, the patient is trained to return to the beg position without allowing the front paws to return to the ground. From the beg, the dog is trained to slowly return to the sit position. This is a challenging but very rewarding exercise, especially for our geriatric patients.Commando crawl involves the patient crawling under a set of obstacles or through a tunnel. This requires great eccentric control of the triceps, quadriceps, supraspinatus, and hamstring muscles.In conclusion, therapeutic exercise programs are designed based upon the patient's weight bearing status. There are no 'cookbook' protocols....each patient requires strategic problem solving each day that they are treated. The therapist must remain focused upon both the patient's and the client's motivation, skill, and long-term goals. Most importantly, the therapist must keep it fun for the patient, always being prepared to make changes quickly as dogs can become bored or frustrated.

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UPDATE ON IMMUNE-MEDIATED HEMOLYTIC ANEMIA IN DOGS

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Immune-mediated hemolytic anemia (IMHA) is a common hematological disorder in dogs, may be primary (idiopathic, autoimmune) or occur secondarily to underlying diseases and is often associated with life-threatening complications. The diagnostic approach and its management with immunosuppression and transfusion support will be discussed with emphasis on evidence and controversies.

Introduction

Immune-mediated hemolytic anemia (IMHA) is one of the most common and serious hemolytic anemias in dogs, but occurs rarely in other animal species. In IMHA an immune response, including anti-erythrocytic antibodies, complement and macrophages, targets directly or indirectly erythrocytes and a hemolytic anemia ensues. There are many triggers for IMHA such as infections, drugs and other agents, and cancer leading to secondary IMHA, but in many dogs no cause is identified (so-called idiopathic, autoimmune or primary IMHA) or a genetic predisposition has been proposed (Cocker spaniels). Furthermore, alloimmune hemolytic anemias, such as hemolytic transfusion reactions, both acute and delayed, and neonatal isoerythrolysis (only litters from transfused bitches), are caused by specific anti-erythrocytic alloantibodies. In contrast to other species, dogs with IMHA also develop an often overwhelming inflammatory response resulting in thrombosis and necrosis of various organs. And while the anemia can be corrected with transfusions, these complications in dogs are causing severe morbidity and mortality despite aggressive immunosuppression and antithrombotic interventions.

Immune Destruction of Erythrocytes

Regardless of the underlying cause, IMHA results from a breakdown in immune self-tolerance or from a deficit in the control mechanism that regulates B and T lymphocyte activity as well as macrophage reactivity. Immune destruction of erythrocytes is initiated by the binding of IgG or IgM antibodies to the surface of erythrocytes. Under most clinical circumstances, immune destruction is an extravascular process that depends on recognition of erythrocytes opsonized with IgG, IgM and/or complement by specific receptors on reticuloendothelial cells. Macrophages with engulfed erythrocytes may be noted on cytological examination of blood and tissue aspirates as erythrophagocytosis, but this is not definitive proof of an immune-mediated process. Antibody-coated erythrocytes may also be lysed by complement fixation and the membrane attack complex, which is clinically noted as intravascular hemolysis.

A diagnosis of IMHA must demonstrate accelerated immune destruction of erythrocytes. Evidence of a hemolytic anemia is suggested clinically by icterus and a regenerative anemia with hyperbilirubinuria, and hemoglobinemia and hemoglobinuria refers to an intravascular process. However, the erythroid response in the bone marrow may be blunted by the immune and inflammatory process or the underlying disease thereby leading to non-regenerative anemias. Besides documenting a hemolytic anemia, one or more of the following three hallmarks must be present to support a diagnosis of immune-mediated hemolysis: persistent autoagglutination, marked spherocytosis and a positive direct Coombs' test result. As in human medicine, the Coombs' test should be considered the best test to definitively diagnose IMHA, although marked spherocytosis and persistent/true autoagglutination (after 3x washing of EDTA blood with saline) are other important parameters indicating immune-destruction of erythrocytes.

Autoagglutination

Anti-erythrocytic IgM and in large quantities IgG antibodies may cause direct erythrocyte autoagglutination. The autoagglutination may be seen by naked eye in an EDTA tube or on a glass slide or may become apparent as small clumps of erythrocytes on blood smears. For yet unexplained reasons, canine erythrocytes have a tendency to unspecifically agglutinate in the presence of plasma and colder temperatures as well as possibly with excessive EDTA anticoagulant. Mixing blood with one drop of saline may break up rouleaux formation but not other forms of unspecific red cell agglutination. It is, therefore, important to determine whether the agglutination persists after "saline washing", which has been coined persistent or true autoagglutination. This is accomplished by adding physiologic saline to the tube containing a small amount of EDTA-anticoagulated blood, mixing, centrifuging and removing the supernatant including the plasma and repeating this saline washing 3 times. True or persistent autoagglutination is indicative of an immune process, but precludes the performance of Coombs' test or blood typing and crossmatching procedures which are based upon an agglutination reaction as result. Those based upon chromatographic techniques do not seem to be affected by autoagglutination as free red cells can move along the strip. If the agglutination breaks up after washing, the Coombs' test is expected to be positive, if it is a case of IMHA. There is no evidence for washing away red cell bound antibodies in dogs.

Spherocytosis

If erythrocytes are only partially phagocytized or lysed by complement in circulation, erythrocytes with reduced surface area to volume ratio, known as spherocytes, are formed. They appear spherical and microcytic with no central pallor and are considered fragile. Note proper areas on the blood smear needs to be reviewed to find spherocytes in between single regular discoid red cells. Large numbers of spherocytes (>20/microscopic high power field) are nearly diagnostic for IMHA, whereas small numbers may be seen with other conditions including DIC, endotoxemia and zinc intoxication. In our experience all dogs with marked spherocytosis and suspected to have IMHA also had a positive Coombs' test. However, only 60-80% of dogs with a positive Coombs' test or clinically diagnosed with IMHA had marked spherocytosis. Hereditary spherocytosis due to genetic membrane defects has rarely been seen in dogs, but should be considered as a differential diagnosis in dogs with negative Coombs' test results.

Because of the difficulties with the Coombs' test (see below), Slappendale had proposed to use the erythrocytic osmotic fragility test at specific saline concentrations as a mean to diagnose IMHA and this test is currently used in various clinics in Europe. However, there are many other reasons for increased fragility of erythrocytes beside IMHA including hereditary red cell defects. This test is not used in human medicine and has not been shown to be superior to determination of marked spherocytosis and a positive Coombs' test in dogs with IMHA. The osmotic fragility test is also a cumbersome and not well standardized technique.

Positive Direct Coombs' Test Result

The direct Coombs' test is also known as direct antiglobulin test (DAT) and is used to detect antibodies and complement on the surface of erythrocytes when the anti-erythrocyte antibody strength or concentration is too low to cause spontaneous agglutination (subagglutinating titer). Separate canine-specific IgG, IgM, and C3b antibodies as well as polyvalent antiglobulin reagents are available. They are added at various concentrations after washing the patient's erythrocytes free of plasma (3x as shown above) and mixtures are generally incubated at room temperature or 37°C (cold agglutinins appear to be rarely of clinical importance and rarely cause hemolysis). The strength of the Coombs' reaction does not necessarily predict the severity of hemolysis, but reaction changes are useful in monitoring the disease.

Typically tube or microtiter methods have been used exclusively in the reference or teaching laboratory setting, but a flow cytometric method has also been introduced in a couple of places. A standardized, sensitive, and simple gel column method was available by DiaMed (Switzerland), but unfortunately the company was sold to another company which decided to not pursue the veterinary market. A novel standardized antiglobulin test method has just been developed by Alvedia (France) similar to the immunochromatographic strip technique for blood typing of dogs and cats (see updates on blood typing and crossmathing). Although many commercial laboratories offer Coombs' testing for dogs, clinicians have questioned the tests sensitivity and specificity and often forgo the test and/or use response to therapy as a diagnostic. However, negative Coombs' test results may be seen because of technical reasons, insufficient quantities of bound antibodies, the presence of weakly bound antibodies, or the disease in remission. The Coombs' test stays positive for days to months after initiating treatment. A few days of immunosuppressive therapy will likely not reverse the Coombs' test result, as unlikely a transfusion would cause a positive Coombs' test result. Thus, dogs with negative Coombs' test results should be reevaluated for other causes of hemolytic anemia.

In a recent prospective study of anemic and non-anemic dogs we compared various direct Coombs' test methods including microtiter plate assays, gel column, capillary, and immunochromatographic techniques using polyvalent antiglobulins in a laboratory setting and found excellent correlations between tests and with spherocytosis and without noticeable interference by immunosuppressive or transfusion therapy in anemic dogs.

In conclusion, a diagnosis of IMHA requires the documentation of red blood cell destruction and an immune process. While regenerative anemia, icterus, and hyperbilirubinuria are suggesting a hemolytic anemia, evidence of true autoagglutination, spherocytosis, and/or a positive direct Coombs' test are required to document immune destruction. The authors also recommend monitoring IMHA patients for the disappearance of these immunological parameters to adjust and taper therapy.

Therapeutic Considerations

A diagnosis of IMHA requires the documentation of red blood cell destruction and an immune process. While regenerative anemia, icterus, and hyperbilirubinuria are indicating the presence of a hemolytic anemia, evidence of (1) true autoagglutination after washing, (2) marked spherocytosis, and/or (3) a positive direct Coombs' test are required to document immune destruction. The prognostic factors for IMHA are poorly defined unless IMHA is secondary to an underlying disease. Severe anemia, icterus, leukocytosis, hypoalbuminemia and thrombotic evidence are unfavorable findings. Because the severity of IMHA ranges from indolent to life-threatening disease and serious complications seen with IMHA, therapy has to be tailored for each patient and depends in part on whether the IMHA is primary or

secondary in nature. Removal of the triggering agent or treatment of the underlying condition can bring the IMHA rapidly under control.

Fluids, Blood Transfusions, Oxygen and Oxyglobin in IMHA

Restoration and maintenance of tissue perfusion with crystalloid fluids is important, even when it results in further lowering of the hematocrit. When severe anemia and a dropping hematocrit lead to signs of tissue hypoxia, packed red blood cell transfusions appear beneficial. The increased oxygen-carrying capacity provided by the transfused red blood cells may be sufficient to maintain the animal's hematocrit for a few days, while other treatment modalities have time to become effective. The notion that transfusions pose an increased hazard to animals with IMHA has been overemphasized and is not supported by retrospective clinical studies. Fresher blood products are possibly an advantage. However, the common occurrence of autoagglutination may make blood typing and crossmatching of the patient impossible. In these cases DEA 1- blood should be transfused. Additional blood types are being recognized which may be also important.

If compatible blood is not available, the bovine hemoglobin solution Oxyglobin, a highly purified bovine hemoglobin solution, if available, may be administered and provides increased oxygen-carrying capacity and plasma expansion. The original FDA study documented the beneficial effects of Oxyglobin in dogs, whereas recent retrospective studies do not allow any conclusions. In contrast to blood and Oxyglobin, oxygen inhalation therapy is of little benefit, unless the animal with IMHA is suffering form pulmonary disease such as pulmonary thromboembolism. Thanks to adequate transfusion support, animals with IMHA rarely die because of anemia, but because of secondary complications such as thromboemboli and infections.

Immunosuppressive Therapy for IMHA

The insufficient understanding of the pathogenesis, the generally guarded prognosis, the lack of good therapeutic trials, the serious drug side effects, and the high costs of intensive care greatly hamper the successful management of dogs with IMHA. The main goal of immunosuppressive therapy is to reduce (1) phagocytosis, (2) complement activation, and (3) anti-erythrocytic antibody production. Glucocorticoids are the initial treatment of choice for canine, feline and human IMHA. They interfere with both the expression and function of macrophage Fc receptors and thereby immediately impair the clearance of antibody-coated erythrocytes by the macrophage system. In addition, glucocorticoids reduce the degree of antibody binding and complement activation on erythrocytes, and only after weeks, diminish the production of autoantibodies.

Thus, oral prednisolone at a dose of 1-2 mg/kg twice daily is the mainstay treatment. Alternatively, oral or parenteral dexamethasone at an equipotent dose of 0.6 mg/kg daily can be used, but is likely not more beneficial.

There is no evidence that other immunosuppressive agents are effective. They should not be used initially as they are associated with severe side effects. Additional immunosuppressive therapy is warranted when prednisone fails, only controls the disease at persistently high doses, or when it causes unacceptable side effects. They are generally used together with prednisolone, but may eventually be used independently. Historically, cytotoxic drugs such as cyclophosphamide were added, however a small randomized study and several retrospective surveys failed to show any beneficial effects, but may be associated with greater morbidity and mortality in the acute management of IMHA. Retrospective studies and anecdotal reports with azathioprine, cyclosporine, danazol, mycophenylate, and human intravenous immunoglobulin suggest some efficacy, but controlled prospective clinical trials that document their efficacy are lacking. For instance, there is no evidence that azathioprine is effective and from a mechanistic view point it only inhibits antibody production and as it is an antimetabolite, it is only effective after a few weeks. Furthermore, the side effects of acute pancreatitis and agranulocytosis to aplasia makes this in most cases unsuitable. Cyclosporine at 5-10 mg/kg is likely the best and safest second agent but blood drug levels have to be determined in order to avoid toxicity and underdosing. Highly immunosuppressive agents from transplantation medicine such as mycophenylate and leflunamide are other agents which have been tried but no definitive beneficial effects have been reported. Finally, human intravenous immunoglobulin at 2 x 1 g/kg may rescue a non-responding IMHA patient but relapses are common.

One other agent is melatonin which has been added as immunotherapy which is begin but it is unclear if this has any beneficial effects. Splenectomy may be considered particularly in refractory cases with large spleen, but even a normal spleen may excessively clear antibody-coated red blood cells. Furthermore, splenic histopathology, toxicology and infectious disease screens may offer a diagnosis of an underlying disease. Finally, because of the apparently severe agglutination and the inflammatory and necrotic process, plasma exchange therapy has been used in a few cases and appeared to be helpful in expediting response and avoiding serious complications.

It should be noted that an apparent therapeutic response to immunosuppressive therapy is insufficient evidence for the diagnosis of IMHA. Response to therapy may be indicated by a hematocrit that rises or stabilizes, an appropriate reticulocytosis, diminished autoagglutination,

and fewer spherocytes; this response can be expected to be seen within days to weeks. The subsiding of autoagglutination would allow the performance of a direct Coombs' test and thereby permit the direct documentation of anti-erythrocytic antibodies. As glucocorticosteroid therapy is associated with well-known side effects, the initial dose will be tapered by reducing the amount by one-third every 7-14 days and moving toward every other day therapy. In secondary IMHA with appropriate control of the underlying disease, the tapering can be accomplished more rapidly.

Thromboembolic and Other Complications with IMHA

Because of the potential of gastrointestinal ulceration by glucocorticosteroids and other immunosuppressives, gastrointestinal protectants such as sucralfate may be considered. Because dogs with IMHA suffer from an immune deregulation which may have been triggered by an infection and are treated with immunosuppressive agents, these patients are prone to experience infections; it is, therefore, prudent to administer preventative as well as therapeutic antibiotics to these dogs with IMHA on immunosuppressive therapy.

Thromboemboli and DIC are unique serious complications that greatly contribute to the morbidity and mortality of dogs with IMHA which are not typically seen in humans and cats with IMHA. Although the pathogenesis remains unknown, venipuncture, catheters, confinement, and glucocorticosteroids as well as other immunosuppressive agents may be contributing factors. Thus far, no study has definitively documented any successful prevention and/or management protocol for these life-threatening hemostatic problems in canine IMHA. Predisposing factors should, whenever possible, be limited, and adequate perfusion and tissue oxygenation should be provided with fluids and transfusions or Oxyglobin. Generally, anticoagulation therapy is instituted after there is some evidence or suspicion of thromboemboli. Unfractionated Heparin (dose of 50-300 IU/kg subcutaneously every 6 hours or by continuous intravenous infusion) or Low Molecular Weight Heparin (LMWH; Dalteperin 150 IU/kg sc every 12 hours) are the most commonly used drugs and is used. The replacement of coagulation factors and antithrombin III has not been proven to be beneficial. Antiplatelet agents may also be used and for instance an ultralow dose of aspirin (1 mg kg once daily) has been advocated by a couple of groups, but other studies question its efficacy. Other antithrombotic agents such as modern antithrombotic agents have been used occasionally, but their efficacy and safety remain also unproven.

In conclusion, the successful management of IMHA remains a challenge; immunosuppressive therapies beyond glucocorticosteroids have not been proven to be effective but can be associated with serious side effects. Furthermore, the tendency to inflammation, necrosis and thromboembolism of dogs with IMHA contributes greatly to the morbidity and mortality of dogs with IMHA and effective preventative and therapeutic interventions have not yet been established.

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UROLOGY

URETERAL OBSTRUCTION IN CATS: DIAGNOSIS AND MANAGEMEN

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Ureteral obstruction in cats: diagnosis and management

Ureteral obstructions in cats may be luminal, intramural or extramural. Causes for intraluminal obstructions are stones, blood clots or debris (Adams 2017). Intramural causes are strictures, edema of the mucosa or neoplasia. Extramural causes are compression because of circumcaval ureters, masses compressing the ureters or trauma. The most common cause of ureteral obstruction in cats is urolithiasis. Most of these stones contain calcium oxalate and therefore dissolution by dietary interventions or medications is not possible. A ten-fold increase in ureteral stones has been reported in cats between 1980 and 1999 (Lekcharoensuk et al 2005).

Ureteral obstruction can lead to severe kidney damage.

Common clinical signs of ureteral obstruction are not specific. Inappetence, lethargy, signs of lower urinary tract disease, vomiting, and weight loss were the most common signs in one study (Wormser et al 2016). Eight percent of the cats showed no clinical signs.

Radiographs and ultrasound are used to identify the causes and the severity of the obstruction. In one study, sensitivity of these two modalities to identify ureteral calculi was 90% (Kyles et al 2005). Computer tomography can provide additional information

Treatment options for ureteral obstruction are surgical removal of the intraluminal obstruction, placing a ureteral stent placing a subcutaneous bypass system.

Traditional surgery was considered not very successful as 19% of the patients died or were euthanized during or shortly after the procedure (Kyles et al 2005). In another study, 21% of the cats did not survive to discharge (Roberts et al 2011). However in a more recent study where ureteral surgery was compared with ureteral stents, perioperative mortality of traditional surgery was 8% (Wormser et al 2016).

Because traditional surgery was unsatisfactory, other options for treatment were developed. Ureteral stents provided an option to treat the cats without opening the ureter. Ureteral stenting seems to be more successful than traditional surgery. In one study of 69 cats, stent placement was successful in 75 of 79 ureters (95%) (Berent et al 2014). The perioperative mortality rate was

7%. The median survival time was 498 days. Long term complications included re-obstruction of the ureters and were seen in 19% of the ureters. Over all, stents had to be replaced in 27% of the ureters.

Subcutaneous ureteral bypass devices are considered easier to place. In a study presented at the ACVIM forum 2016, in 137 cats, the device was successfully placed in all 174 ureters (Berent et al 2016). 94% of the cats survived to discharge. Median survival time was 827 days. 13% of the SUBs had to be replaced due to obstruction.

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ISOLATED COMMUNITIES AND ANIMAL WELFARE MANAGEMENT

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ISOLATED COMMUNITIES AND ANIMAL WELFARE EMERGENCY MANAGEMENT

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Resilience implies the ability to recover or 'bounce back' from threats or disasters. In planning for natural or man-made disasters, resilience has tended to focus at the level of a population or community. In particular, building the resilience of geographically isolated communities.

However, isolation can also exist within urban communities. Through lack of connectedness with others, vulnerable individuals within a community can become socially isolated. Vulnerable individuals may include; the elderly, people affected by a mental illness or drug dependencies, refugees, indigenous people, women, single parents, people with disabilities, and the homeless. Though socially isolated and vulnerable individuals may possess the skills to function on a day-to-day basis, they may lack the resilience to cope with a crisis situation. Where connectedness to others within a community may be lacking, the support people feel from pets can strengthen the emotional resilience of socially isolated individuals[1].

Current evacuation and emergency protocols focus predominantly on the preservation of human life and critical infrastructure. The impact that emergencies have on the psychological well-being of people is a secondary concern, and the complex social and psychological needs of vulnerable individuals are often overlooked. Existing protocols and procedures are designed for people who have connections within their community and can respond to instruction – applying these protocols to vulnerable or socially isolated people might not be successful, and there is a need to make protocols more flexible and inclusive to enhance cooperation and reduce trauma. For example, in an emergency setting, owners may be encouraged, or forced to leave pets behind. For socially isolated and vulnerable individuals, this may perpetuate psychological trauma and overwhelm already fragile coping abilities. Allowing people to evacuate alongside their pets can reduce distress and provide an alternative focus, thus acting as

a distractor or 'normalizer' during a crisis situation. This may promote a transition from reactionary behaviour to a more considered response to the situation.

Vulnerable individuals may be clients of veterinary practices due to pet ownership. Therefore, veterinary professionals have a unique opportunity to use a shared love of animals to connect with and advocate for the needs of vulnerable and socially isolated individuals during crisis situations. Additionally, as vulnerable people cannot always express their emotional needs, innovative engagement strategies are required to ensure these people retain a sense of connection.[2]

Breaking down the barriers

Pets can provide more than direct companionship for their owner. Pets can act as 'icebreakers' in a social setting. This is an incredibly valuable tool to help vulnerable individuals establish social connections within their community. Importantly, for socially isolated individuals, this 'pet-related' connection can translate into new sources of social support.

It is this safe target of conversation that can also be used to bond with socially isolated people during disaster events. Veterinary professionals can make a significant contribution to inclusiveness of socially isolated people. Ways that veterinary professionals can help during a crisis include; 'checking-in' on vulnerable clients, offering the veterinary practice as a 'safe-haven' where possible, and providing support in human welfare centres as a friendly face to talk to.

A shifting paradigm

In a recent paper outlining the roles of animal attachment on disaster resilience of vulnerable groups, Thompson and colleagues explained: "An emerging body of evidence recognises that animal attachment can pose a risk to human safety in disasters and that, conversely, animal attachment can be leveraged within community engagement strategies to increase disaster preparedness."[2] To put this more simply, pets can play a positive or negative role during disaster management. As policy influencers and first responders, we could swing the balance from negative to positive. This is a paradigm shift for emergency management and veterinary professionals. Re-working the 'status quo' will require considerable fore-sight and a multi-discipline approach to develop innovative ways on how to motivate animal owners to be prepared for disasters.

Many human welfare centres will not accept pets during an emergency situation. Unfortunately, ill-preparedness by the general public for a disaster event will lead to an unnecessarily large proportion of the community seeking refuge in welfare centres. This could mean leaving pets behind, resulting in pet owner distress and significant animal welfare issues. For example, In New

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Zealand, a country which has various hazards such as volcanoes, earthquakes, wildfires and severe weather events, only 14% of the population are fully prepared.[3] The best-case scenario is to keep people with their pets. This is achievable through greater education and preparedness of communities. There are on-going efforts in some countries to create pet-friendly emergency welfare centres. However, further work is required for this to become a standard consideration during disaster planning. The veterinary profession needs to take a proactive approach to ensure local council and government bodies recognize the importance of preparing pet-owners for emergencies, and in establishing more consistent supply of pet-friendly welfare shelters.

The recovery phase – an additional challenge

During the recovery phase communities may become fragmented and social support networks may be lost. The number of socially isolated or vulnerable people may rise. During this time, people who have lost their homes will be transitioning from emergency accommodation arrangements to temporary accommodation. In many instances due to housing shortages, the stock of available accommodation is insufficient, and finding accommodation that allows for pets may be even more of a challenge.

It is during this time that displaced people need as much psychosocial assistance as possible due to secondary trauma caused by the accumulative effect of additional stressors during the recovery efforts.[4] Dealing with insurance companies, banks, councils and builders can cause a significant amount of stress even more so than the actual event, hence why it is called the secondary trauma. Additionally, the event has long gone from media attention, and this can further compound the isolation felt due to still being in the disaster, and everyone else has moved on.

There are several ways that veterinary professionals can help during the recovery phase. Firstly, continuing to advocate for pets to stay with their owners where possible. Secondly, accepting donations (pay-it-forward) to assist with care of patients whose clients are no longer able to afford veterinary care. Thirdly prevention of transmissible diseases following a disaster (e.g. leptospirosis vaccination of dogs following flooding) and finally, discounted microchipping of unchipped pets to enable identification of pets should they go missing in the disruption following a natural disaster. In addition, veterinarians should observe clients for signs of mental health issues or distress, and know what services are should there be concerns about client well-being.

Following a disaster, local government or support agencies may hand out welcome home packs to homeowners when they return to rebuilt or repaired homes. This is an opportunity for the veterinary profession to show community support through contribution to such packs. The most helpful addition to such a pack would be advice regarding pet care following a disaster event (e.g. dealing with pet behavioural issues following a disaster).

Conclusion

The veterinary profession has a unique opportunity for communicating and motivating vulnerable people to engage in resilience building behaviors that promote survival and facilitate recovery from a disaster. Veterinary professionals can offer a safe-haven for animal owners during a disaster event, and can advocate for keeping people with their pets. Additionally, veterinarians can participate in outreach programmes during the recovery phase, and advise on animal-welfare impacts following a disaster.

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WELLNESS

PROFESSIONAL WELLNESS FOR VETERINARIANS: IS IT AN ISSUE

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Personal and professional wellness of veterinarians and veterinary staff are receiving increased attention in veterinary publications & conferences, social media and the non-veterinary press. Recent suicides of high-profile, socially-harassed or victimized veterinarians have caused consternation within the profession. There is an increased recognition that stress and compassion fatigue coupled with a demanding workplace environment are adversely affecting the mental well-being and physical health of veterinarians.

Several studies have estimated that the incidence of suicide in the veterinary profession in countries such as USA, UK, Australia & Norway to be double of the other health care professionals, and four times that of the general population (Stoewen, 2016; Bartam and Baldwin, 2008). A number of influencing factors may been postulated as contributing to this increased risk: personality factors, undergraduate training, professional isolation, work-related stressors, attitudes to death and euthanasia, access and technical knowledge, psychiatric conditions, stigma around mental illness, and suicide contagion (Bartram and Baldwin, 2008, 2010).

Also a heavy workload, insufficient rest and prolonged, intense contact with animals and their owners can result in occupational stresses and burnout. Veterinarians who neglect their physical, emotional and psychological needs can find themselves suffering from "compassion fatigue", and it has been estimated that between 15-67% of veterinarians are at high risk of burnout (Brannick et al, 2015).

However, the research done comes mainly for the developed world. And even there scientific evidence on topics like for instance compassion fatique, is lacking. Furthermore it seems that this professional wellness issues are not seen in for instance Asia. The question is whether or not this is true, and if so why?

In this lecture ways to optimize your professional wellness as a veterinarian will be discussed.

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INTRODUCTION TO PHYSICAL MODALITIES USED IN REHABILITATION

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INTRODUCTION TO PHYSICAL MODALITIES USED IN REHABILITATION

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Canine rehabilitation is the application of physiotherapeutic techniques to evaluate and treat musculoskeletal impairments in our canine patients. It incorporates the use of objective outcome measures (goniometers, girthometers, etc.), manual assessments (including palpation, joint glides, and neurological assessment), gait analysis, and special tests brought from the field of human physiotherapy. This allows the therapist to tease out the specific structure and tissue type causing the impairments.The therapist evaluates the presenting complaint, subjective information from the owner, and objective assessment carried out during the examination to create a problem list. Each item on the problem list is addressed in the plan of care.Therapeutic plans generally involve a combination of manual therapies (joint mobilizations and soft tissue mobilizations), physical modalities (laser, therapeutic ultrasound, e-stim, shockwave), and therapeutic exercises. The modalities are generally used to prepare the tissues for the manual therapies and therapeutic exercises. Physical modalities should never be the sole therapeutic method applied to any patient.Therapeutic parameters for each modality are chosen based upon the acuity of the injury, so the therapist must be well versed on the definitions of the acute, subacute, and chronic phases of healing. In the acute phase of healing, the initial damage is present along with an inflammatory response. During this phase, modalities are chosen to treat the acute pain, prevent exacerbation of the pain, and to prevent compensatory dysfunction that occurs secondary to pain. In the subacute phase of healing the inflammatory response has ended, but the tissues remain fragile and at high risk of slipping back into inflammation. During this phase, modalities are chosen to enhance tissue healing, resolve compensatory pain, and prevent exacerbation of the underlying injury. In the chronic phase of healing, tissue healing is underway but incomplete. During this phase, modalities are chosen to help gain complete resolution of the underlying injury, reconditioning of injured tissues, and whole-body reconditioning.The physical modalities fall into four categories: thermal, electrical, sound, and electromagnetic. Choosing which modalities to use should be based upon evidence-based medicine principles. According to the Evidence Based Veterinary Medicine Association (EBVMA), "Evidence-based medicine is the effort to place all medical decisions on the strongest scientific proof (evidence) available." A physical modality is a device or application that delivers a physical agent to the body for therapeutic purposes. In other words, this is the transfer of energy for therapeutic purposes.The most commonly used physical modalities in veterinary practice include cryotherapy, neuromuscular electrical stimulation, therapeutic ultrasound, lasers, and extracorporeal shock wave. Cryotherapy can be applied through ice baths, ice massage, ice packs, vapocoolant gel, or ice/compression units. Cryotherapy causes vasoconstriction, reduced cell metabolism, decreased nerve conduction velocity, analgesia, reduced edema, and decreased muscle spasm. The mechanism of action of cryotherapy in decreasing inflammation is vasoconstriction, decreased histamine release, decreased edema and decreased protease release. The mechanism of action for decreasing pain is via decreased release of prostaglandins, decreased muscle spasm, and decreased nerve conduction velocity. Cryotherapy also lowers the metabolic rate of cells, protecting cellular viability through decreased demand for oxygen and nutrients. This reduces hypoxia, preventing further cellular destruction. Swelling is diminished, leading to less secondary hypoxic injury. However, cryotherapy alone does not eliminate swelling. Some form of compression is essential to accomplish this. Intermittent compression mirrors muscle contractions to force tissue debris into the lymphatic system, thus lowering oncotic pressure and promoting fluid reabsorption. Cryotherapy has been shown to improve joint range of motion through suppression of excitatory muscle spindle afference. Studies have shown that intermittent pneumatic compression with cryotherapy prevents edema and optimizes lymphatic drainage.Many veterinarians still prefer to use bandages after orthopedic surgery. Studies have shown that cold compression with or without a bandage decreased swelling better than a bandage alone, and that cold compression significantly decreased pain, lameness, and swelling, and improved ROM in TPLO patients treated for 24 hours post operatively. Pneumatic cryotherapy devices are efficacious in reducing inflammation, combine dry cold therapy with intermittent compression, keep the skin surface dry, reducing the chance for infection, and allow for deeper penetrating cold than traditional ice packs.

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SVA DERMATOLOGY (SIMULTANEOUS TRANSLATION INTO MANDARIN CHINESE)

OTITIS EXTERNA - REFRACTORY AND RECURRENT CASES

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INTRODUCTION

Cases of chronic or recurrent otitis are challenging. Successful treatment requires that all the underlying factors leading to persistence or recurrence of the otitis are identified and managed. In particular, it is important to recognise the role of inflammation in otitis. Most owners and clinicians recognise ear infections, which are then successfully managed. However, the ongoing inflammation is often missed. This leads to a cycle of recurrent infection and chronic inflammation leading to progressive pathological changes and end-stage otitis that requires surgical intervention. The chronic inflammation makes each bout of infection harder to treat and repeated antimicrobial use may select for resistance.

In recurrent otitis, the treatment that is prescribed is effective but the condition recurs after therapy is withdrawn. In refractory otitis, the infection persists even when treatment is being administered.

THE REFRACTORY OTITIS EXTERNA

Where to start?

Some cases of refractory otitis externa develop infection with Gram negative organisms such as **Pseudomonas aeruginosa.** These most commonly follows treatment of a pre-existing ear infections with various antimicrobial agents. These organisms can be resistant to many commonly used antibiotics and can be difficult to treat.

1. Perform cytology and culture and sensitivity

There is substantial controversy about whether to perform bacterial culture and sensitivity of infected ears. **In my opinion, when there is chronic disease where rods or mixed populations are found on cytology, culture is useful.**

Detecting rods on cytology is consistent with Gram negative bacteria such as **Pseudomonas** species, **Proteus** species, **Escherichia coli. Pseudomonas** are the most common. Bacterial culture and sensitivity testing definitively identifies the bacteria involved in the infection. This can be useful for less common organisms that are hard to differentiate on cytology, eg, streptococci, enterococci, **Escherichia coli**, **Klebsiella**, **Proteus** and coryneforms.

The susceptibility pattern of these bacteria is hard to predict, although most first-time infections are susceptible to topical aminoglycosides, polymyxin B, silver sulfadiazine and fluoroquinolones. However, **Pseudomonas** species readily acquire resistance and most isolates from recurrent infections will be multi-drug resistant. Knowledge of their likely sensitivity patterns can then help guide treatment choices.

Antimicrobial susceptibility data is less useful for topical otic drugs because concentrations in the ear canal are much higher than those used with in vitro tests predict. The response to treatment is best assessed using clinical criteria and cytology. Antimicrobial sensitivity data can be used to predict the efficacy of systemic drugs, although the concentration in the ear tissues is often low and high doses are needed. For example, enrofloxacin would need to be given at 20 mg/kg to treat **Pseudomonas** isolates with an MIC of 0.5 µg/ml (middle of the susceptible range) in chronic otitis.

2. Systemic corticosteroids

As otitis progresses, the inflammation created by primary factors leads to hyperplasia of the stratified squamous epithelial lining of the canal, resulting in narrowing of the lumen and glandular hyperplasia, leading in turn to an increased production of cerumen and hidradenitis. These chronic changes, often in tandem with recurrent courses of topical antibiotics, lead to the development of a more resistant population of bacteria, especially Gram-negative bacteria, such as **Pseudomonas** species. Such cases require careful management as the canal is often ulcerated and painful and otitis media is a common sequel. Where the disease process becomes chronically irreversible, the lumen may be completely obliterated and in some of these cases ossification of the soft tissue may also take place. Once the damage to the ear has become this extensive, medical therapy is rarely effective. The most important perpetuating factors are listed in Table 1.

Table 1: Perpetuating Factors	Specific change
Pathological changes in the external ear canal	**Changes in canal wall:** Inflammation causing failure of epithelial migration. Chronic epidermal hyperplasia, canal stenosis, calcification of pericartilaginous fibrous tissue
	Changes in glandular tissue: hyperplasia of ceruminous and sebaceous glands, hidradenitis
	Changes in the tympanum: dilation, rupture, diverticulum (false middle ear – cholesteatoma)
Otitis media: chronic	Biofilm formation, granulation material, bony change in the bulla

Reducing pruritus, swelling, exudation and tissue proliferation is a key goal of therapy. Glucocorticoids (particularly dexamethasone) also reverse the ototoxic effect of **Pseudomonas** infections. Prednisolone (1 to 2 mg/kg every 12 to 24 hours) is sufficient to control inflammation and stenosis in most cases. Patients with severe fibrosis and stenosis, however, may respond better to dexamethasone (10 times as potent

as prednisolone). Oclacitinib and lokivetmab are not effective. Ciclosporin has also been shown to be effective in some cases but is only useful for chronic otitis.

3. Remove debris and discharge

Ear flushing is imperative to remove discharge from the external ear canal and/or the middle ear. Removal of debris and purulent material greatly improves the efficacy of topical antimicrobials, especially aminoglycosides and polymixin B.

Excess hair should be clipped from the medial aspect of the pinna and excess exudate removed from the pinna and around the entrance to the external ear canal. One method of ear flushing is performed with a three-way tap or stopcock connected to a 10ml syringe, warmed saline in a fluid bag and a 5-French feeding tube or 8-French polypropylene urinary catheter. It is often better to insert the feeding tube or catheter to a deeper part of the canal and backflush the material. A pulsing action can assist with dislodging trapped exudate. The external ear canal is alternately flushed and aspirated, removing the remaining exudate and debris allowing visualization of the tympanic membrane with an otoscope or video otoscope. For suppurative otitis externa, **2% acetic acid or 2% acetic acid and 2% malic acid** can be used for flushing ears instead of saline for infections with **Pseudomonas** otitis. Two and five minutes contact with 2% acetic acid is lethal to **Pseudomonas and Staphylococcus** respectively. Acidic cleaners may inactive some antimicrobials (especially aminoglycosides and fluoroquinolones), although ear canals have good buffering capacity and the pH rapidly returns to normal.

Biofilms

Biofilms can be physically broken up and removed by thorough flushing and aspiration. Topical trizEDTA and 2% n-acetylcysteine can disrupt biofilms, facilitating their removal and enhancing penetration of antimicrobials. Systemic administration of n-acetylcysteine is well tolerated and can help dissolve biofilms in the middle ear and other mucous surfaces.

Table 2: Ear Cleaning Regime
Flush and aspirate with warm saline
Instil 2% acetylcysteine solution
Flush out with 2% acetic acid or 2% malic/ 2% acetic acid and leave in ear for 2 mins (Pseudomonas) to 5 mins (Staphylococcus)
Flush out with 0.12% EDTA with 0.2% biguanadine hydrochloride 0.2% (Otoflush®)
Instil topical antibiotic/glucocorticoid of choice before going home

4. Treat otitis media

Inflammation in the ear canal will also affect the tympanic membrane, which may become oedematous, thickened or dilated. Rupture of the tympanum is common. Infection within the tympanic bulla leads to inflammation of the delicate mucoperiosteum lining of the bulla and the production of mucus, which traps infection within the bulla cavity, making it inaccessible to topical drugs. Biofilms are common within the middle ear of children.

Appropriate measures to resolve infection in the middle ear include: flushing of the bulla and the instillation of appropriate drugs into the site, which may include biofilm busting agents, anti-inflammatory drugs and antimicrobials. Where disease within the bulla is not managed, granulation tissue and bony change lead to irreversible damage that may only be successfully resolved by surgical intervention.

If the tympanic membrane is ruptured, repeated flushing of the middle ear should be performed. The tip of the feeding tube is placed adjacent to the tympanic membrane or, if the tympanic membrane is ruptured, into the middle ear under visualization through an operating otoscope. It is important to angle the tip ventrally to avoid the sensitive structures in the dorsal part of the middle ear cavity. The ear canals and middle ear are alternately flushed and aspirated until completely clean. Retrograde flushing using this technique is very effective at removing deep material and is the only effective way to clean the middle ear.

Opinion is divided on the systemic treatment of otitis media; some referral clinicians always use systemic treatment, others instil antimicrobials directly into the middle ear every three to 10 days (enrofloxacin, marbofloxacin, gentamicin appear to be safe used in this way), some use topical therapy and some a combination of approaches. It is likely that antimicrobials persist for several days following direct application into the middle ear, because this is effectively a blind-ending sac with limited drainage into the pharynx.

If the tympanic membrane is intact but appears abnormal and otitis media is suspected a myringotomy (the deliberate rupture of the tympanic membrane) should be performed to obtain samples for cytology, culture and sensitivity and to flush the middle ear cavity. An open-ended 3.5-French tomcat catheter is used to make the incision. Experimentally ruptured normal tympanic membranes heal in three to five weeks. If the ear is kept free from infection following myringotomy the tympanic membrane should heal.

5. Topical and systemic antimicrobial therapy

Topical therapy should be used. This results in high concentrations in the ear canals. Systemic treatment is very useful in suppurative otitis externa and/or otitis media where there is an active inflammatory discharge with concurrent infection in the deep ear canal tissues and middle ear. **Pseudomonas** are

resistant to many antimicrobials through low cell wall permeability, β-lactamases, clavulanate-resistance and efflux pumps. They readily develop further resistance if treatment is ineffective as they have a large genome to express resistance genes and mutations, and are capable of plasmid, transposon and bacteriophage transfer. **Fluoroquinolones and gentamicin are usually effective against first time Pseudomonas infections.** Fluoroquinolones, which have a high volume of distribution and penetrate well into most tissues, may have better efficacy in infections otherwise susceptible to other antimicrobials. Once fluoroquinolone resistance is established other anti-**Pseudomonas** antimicrobials are indicated; these are often expensive, not licensed for animals and have to been given intravenously if used systemically. (Table 3)

Table 3: Antimicrobial agents	
Ciprofloxacin	Systemic: 15mg/kg PO q24h Topical: Cipro HC ® 0.2-0.5 ml/ear q12h
Enrofloxacin	Systemic: 15-20 mg/kg PO q24h Injectable: 5% injectable solution diluted 1:4 with saline or Epiotic ® topically q24h; 22.7 mg/ml solution 1 ml/ear q24h
Marbofloxacin	Systemic: 5mg/kg PO q24h Topical: Aurizon ® 0.2-0.5 ml/ear q12h Injectable: 1% injectable solution diluted 1:4 with saline topically q24h; 20 mg/ml solution 1 ml/ear q24h
Ofloxacin	Topical: Ofloxacin 0.3% 0.15-0.3 ml/ear q24h
Carbenicillin	Systemic: 10-20 mg/kg IV q8h Topical: no proprietary product
Ceftazidime†	Systemic: 25-50 mg/kg IV q8h; Topical: no proprietary product Injectable: 100 mg/ml 1 ml/ear q12-24h
Silver sulfadiazine#	Topical: dilute 0.1-0.5% in saline or trizEDTA) apply 1 ml q24h
Amikacin	Systemic: 10-15 mg/kg SC q24h Topical: no proprietary product Injectable: 50 mg/ml 1 ml/ear q24h
Gentamicin	Systemic: 5-10 mg/kg SC q24h Topical: Otomax ®, Mometamax ® or Easotic ® Injectable: 50mg/ml
Tobramycin	Systemic: not used: nephrotoxic Topical: eye drops Injectable: 8 mg/ml injectable solution 0.15-0.3 ml/ear q24h

- † Reconstituted solution stable for up to seven days at 4°C or one month if frozen
- # Silver sulfadiazine shows additive activity with gentamicin and fluoroquinolones (although synergy has not been proven)

Potential Toxicity of Antimicrobial Agents

Tobramycin and amikacin are potentially ototoxic and should be used with care if the tympanic membrane is ruptured. **Enrofloxacin, marbofloxacin, ceftazidime and silver sulfadiazine** appear to be safe in the middle ear. There is potential for systemic toxicity with silver sulfadiazine and aminoglycosides in extensively ulcerated ears, although this is unlikely in practice as the total body dose will be low except in very small animals. The ototoxicity of gentamicin appears to depend on the preparation, and **topical application of injectable solutions of gentamicin appears to be safe.** Systemic aminoglycosides can be nephrotoxic and renal function should be monitored. Fluoroquinolones can cause cartilage damage in dogs under 12 months old (18 months in giant breeds) and neurotoxicity at high doses.

Tris EDTA

TrizEDTA damages bacterial cell walls and increases antimicrobial efficacy which can overcome partial resistance. It is best given 20 to 30 minutes before the antimicrobial but can be co-administered. It is well tolerated and non-ototoxic. TrizEDTA shows additive activity with chlorhexidine, gentamicin and fluoroquinolones at concentrations of 35.6/9.4 mg/ml, but there is no evidence of synergy and efficacy at lower concentrations. Solutions of 0.6 per cent enrofloxacin, 0.2 per cent marbofloxacin, 0.3 per cent gentamicin, 0.1 per cent amikacin and 1.7 per cent ceftazidime in trizEDTA are effective against many multi-drug-resistant bacteria including **Pseudomonas**.

Otitis media may need three to four weeks (and possibly longer) systemic treatment, which is a problem if parenteral drugs are used. **Pseudomonas** infections, however, usually clear quickly once effective cleansing, antimicrobial treatment and control of the primary cause are established.

5. Topical glucocorticoids

Mild inflammation responds rapidly to low potency topical glucocorticoids, but progressively more severe inflammation requires longer courses of more potent products (Table 4). Very potent products should be avoided in severe bacterial infections, particularly **Pseudomonas**, as they may suppress neutrophil activity.

Once the infection has resolved, topical glucocorticoids should be used at the lowest frequency that controls the inflammation. Systemic treatment is necessary if there is stenosis, severe fibrosis or mineralisation, or if topical therapy cannot be safely administered. It is usually possible to switch to topical therapy once the ear canals have opened. Dogs better tolerate topical therapy once the pain and inflammation has decreased.

Table 4: Relative potency	Glucocorticoid
Very potent glucocorticoids (up to 100 x hydrocortisone)	Fluocinolone
Potent glucocorticoids (25-100 x hydrocortisone)	Betamethasone Dexamethasone hydrocortisone aceponate Mometasone furoate
Moderate glucocorticoids (2-25 x hydrocortisone)	Prednisolone Triamcinolone
Mild glucocorticoids	Hydrocortisone

This table should be used for guidance only, as the relative potency of topical glucocorticoids also varies with the concentration, formulation and preparation. Topical therapy is safer than systemic therapy but adverse effects can be seen, for example, the hypothalamic-pituitary-adrenal (HPA) axis can be affected for up to two to four weeks after otic administration of dexamethasone. Hydrocortisone aceponate and mometasone furoate show less local atrophy and systemic absorption than other glucocorticoids. Atrophic effects can be useful in reversing fibrosis and stenosis early in treatment, but may later interfere with epidermal migration allowing debris and desquamated cells to accumulate in the ear canals.

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THE MOST IMPORTANT FELINE DENTAL DISEASES FOR YOUR PRACTICE

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Feline Tooth resorption (TR)

TRs are a very common malady. Reports vary as to their incidence, but approximately 60% of cats over 6 years of age have at least one, and those that have one typically have more. These lesions are caused by odontoclasts which are cells that are responsible for the normal remodelling of tooth structure. These cells are activated and do not down regulate, resulting in tooth destruction.

There are currently two recognized forms of resorptive lesions, type 1 and type 2. Clinically, they appear very similar, as dental defects that are first noted at the gingival margin. However, advanced cases will show significant tooth destruction and may appear to be a fractured tooth. The best diagnostic tool for differentiating between types is dental radiology. With type 1 lesions, there is no replacement of the lost root structure by bone, whereas with type 2 there is generally marked replacement of the lost tooth structure.

Type 1 TRs are typically associated with inflammation such as caudal stomatitis or periodontal disease. In these cases, it is thought that the soft tissue inflammation has activated the odontoclasts. The inciting cause of class 1 lesions is a cemental defect. Odontoclasts move in and destroy the dentin, leading to secondary enamel loss and a resorption lacuna. The weakened crown will eventually fracture, and in these cases the root canal system stays intact resulting in continued pain and infection for the patient.

Type 2 lesions are generally seen in otherwise healthy mouths; however the lesions will create local gingivitis. The etiology of type 2 TRs remains unproven. The two major current theories are abfraction injuries from eating hard food and excess vitamin D in the diet. Type 2 TRs show histological evidence of simultaneous repair of the defect by osteoblasts at the same time that tooth is being resorbed by odontoclasts.

Historically, restoration was a recommended therapy, however due to the progressive nature of the disease; extraction is now the treatment of choice. Extractions can be very difficult in these cases due to tooth weakening and ankylosis. Additionally, in some cases, there is little to no tooth structure remaining. In cases with significant weakening and or ankylosis, performing the extractions via a surgical approach is recommended to speed the procedure and decrease the incidence of fractured and retained roots.

Recently, crown amputation has been suggested as an acceptable treatment option for advanced type 2 lesions as it results in significantly less trauma and faster healing than complete extraction. This procedure, although widely accepted, is still controversial. Most veterinary dentists employ this technique, however in widely varying frequency. Veterinary dentists typically employ this treatment option only when there is significant or complete root replacement by bone. Unfortunately, the majority of general practitioners use this technique far too often. Crown amputation should only be performed on teeth with radiographically confirmed advanced type 2 TRs which show no peri-apical or periodontal bone loss. Crown amputation should not be performed on teeth with: type 1 TRs, radiographic or clinical evidence of endodontic or periodontal pathology, inflammation, or infection; or in patients with L/P stomatitis. Those practitioners without dental radiology capability SHOULD NOT perform crown amputation. In these cases, the teeth should either be fully extracted or the patient referred to a facility with dental radiology.

Fractured teeth

This is very similar to canine endodontics. However, true fractures are rare in any teeth except the canines. Teeth with direct pulp exposure are painful and/or infected and require root canal therapy or extraction. Root canal therapy is always recommended if possible as extraction of the maxillary canines is challenging and maxillary canine extraction carries a high risk of lip catching. (see below).

There are two main differences between dogs and cats with regard to endodontic disease. First, the pulp chamber of the canine teeth extends very close to the cussp tip. This means that any fracture (no matter how small) is suspect for endodontic disease. Anesthesia for careful probing and dental radiographs should be performed for any fracture. Secondly, cats will tend to resorb the root apex when a tooth has a chronic infection.

Lip trauma following maxillary canine extraction

In my experience, approximately 1/3 of cats that have maxillary cuspids extracted surgically will develop lip trauma from the mandibular canine. For this reason, we try to avoid extracting maxillary canines when possible preferring to perform root canal therapy or periodontal surgery if indicated.

Many of these cats will show no clinical signs, however if the lip is examined, ulcers will be present. These cats are painful and are in need of therapy. Other patients may show mild to severe evidence of discomfort.

The options for therapy include coronal amputation and vital pulp therapy or extraction of the offending mandibular canine.

Case report: "Sid" had both maxillary canines extracted elsewhere due to periodontal disease. He presented with

a complaint of having difficulty eating and the owner was concerned about a TMJ problem because of the way he moved his jaw after eating. Oral exam revealed significant trauma to the lips secondary to the mandibular canines.

The treatment was to perform coronal amputation and vital pulp therapy on the mandibular canines. This is much less painful for the patient and will maintain the strength in the rostral mandible.

Eosinophillic Granuloma Complex

The true etiology of these conditions is unknown; however a local accumulation of eosinophils is thought to initiate the inflammation and necrosis. The accumulation may result from a local (food) or systemic allergies; although these lesions have been seen in cases where allergic disease has been ruled out. Additional causes include a response to irritation, such as chronic grooming or traumatic malocclusion. There may also be a genetic predisposition.

Indolent Ulcers are the most common oral manifestation, and they will present as brownish-red lesions on the upper lip or around the maxillary canine teeth.

Linear granulomas can be single or multiple; the most common sites are the lips, gingiva, palate and tongue. They are generally non-painful, but can become secondarily infected. The typical presentation is a raised, lobulated yellow-pink mass; however, they can also appear ulcerative causing severe damage to the oral mucosa and underlying bone. This may lead to severe periodontal loss, pathologic fractures, or oronasal fistulas.

Histopathology should be performed to confirm the diagnosis. Following confirmation of the diagnosis, a thorough allergy evaluation should be conducted including food trial, flea treatment, +/- allergy testing.

The acute disease process is best treated with systemic corticosteroids; however corticosteroids should NOT be used for long term disease control due to the significant systemic side effects. The typical initial protocol is prednisone 2 mg/kg q 12 hours for 3-4 weeks. Additional options include intralesional triamcinalone (3 mg weekly) or methyl prednisone injections. Antibiotic therapy is required occasionally to induce remission and/or treat secondary infection. There are also cases that appear to respond to antibiotic therapy alone. Therefore, we initially treat mild cases with antibiotics alone and more severe cases with a combination of antibiotics and corticosteroids.

Many cases remain idiopathic, requiring lifelong therapy; options for this include antibiotics and cyclosporine. Fewer side effects may be expected with cyclosporine in comparison to steroids, but there are reports of opportunistic fungal and fatal protozoal infections associated with its chronic use. Use the lowest effective dose,and perform regular therapeutic levels and routine blood testing.

Caudal Stomatitis

This is another frustrating oral inflammatory disease. The best description is a severe immune mediated reaction to dental tissues. Some feel that this may actually be a group of disease processes that look the same clinically which is why they can be very frustrating to treat.

The history will generally include anorexia, drooling, gagging, and pain during mastication. Physical exam will typically include a thin pet with unkempt fur. The oral exam will reveal severe stomatitis usually over all teeth. The inflammation will most commonly be worse on cheek teeth than canines and incisors. However, faucitis is the key clinical finding. Severe hyperplastic inflammation to the gingiva can result from periodontal disease, however faucitis will not be present.

A pre-operative blood panel will generally show a marked elevation in globulins (Polyclonal gammopathy) and total protein.

Medical Therapy: Most medical therapies will work for a while, however in general resistance will start within a year or less. In addition, most therapies have side effects worse than the disease process in and of itself. In general, medical therapy is very frustrating to the practitioner and client.

Corticosteroids are the mainstay of most medical therapy today. It is generally very effective at first and is relatively inexpensive for the client. In my experience, injectable (depomedrol 10 mg IM) is much more effective than oral preparations in my experience. However, they will typically loose effectiveness after a year or so requiring higher and higher doses at shorter increments. This generally results in significant deleterious effects. About 10% of stomatitis cases we treat are already diabetic!!!

Antibiotics are safer than steroids but much less effective, especially in long term therapy. They are generally disappointing in their success. Metronidazole and clindamycin are the mainstays of therapy; however Clavamox and amoxicillin can be used as well. Metronidazole may be the antibiotic of choice due to its anti-inflammatory effect.

Other immune suppressive such as Imuran, Cytoxan, Gold Salts, Cyclosporine have been used. However, they are all very expensive with numerous adverse side effects (mylosuppression). Cyclosporine is currently the most commonly prescribed immune modulatory drug (other than steroids) for this disease process. However, its chronic use is somewhat expensive and has been implicated in severe fungal and protozoal infections. Starting dose is 5-10 mg/kg. Look for a trough level of about 500 ng/ml on regular basis. In most dentists opinion it is only really effective AFTER teeth are removed. However, it has shown promise in resistant cases.

Laser therapy is not proven at all, most clients and

RDVM's are very unhappy with the long term results. It is very expensive and short term relief only.

Surgical Therapy: Extraction is currently the ONLY effective long term treatment for this disease process in cats. In our experience, the sooner this is done, the better that cats do both post-operatively as well as long term.

For extractions to be successful, the teeth must be COMPLETELY removed. Therefore post-operative radiographic confirmation of complete extraction of the tooth roots is recommended. Following the insurance of complete removal of the teeth, perform aveloplasty to remove the periodontal ligament and smooth rough bony edges. This is typically performed do this with a rough diamond bur. Studies report a 60% success rate when all teeth caudal to the canines are extracted, however our experience has not been as good. However, whole mouth extractions have a success rate of approximately **90-95%** for clinical remission. Slight faucitis may remain, but pets are comfortable. In addition, the rare cases that don't completely respond are generally much more responsive to medical therapy. If there is NO inflammation to the canines or incisors (which is rare), then the owner is given the option of leaving the canines. However, if these are inflamed, all teeth should be extracted.

Resistant Cases

In the rare cases where the teeth have been fully extracted but inflammation and pain continues, other therapies are needed. The current treatment of choice in the USA is cyclosporine. Another option, which appears to work better in Europe is feline interferon. Finally, UC Davis has had some success with Stem Cell Therapy.

Feline Juvenile (puberty) gingivitis/periodontitis

Definition: Juvenile periodontal disease is inflammation which occurs soon after permanent tooth eruption. This syndrome can be described in two categories, **feline hyperplastic gingivitis and juvenile onset periodontitis**.

Etiology: The etiology of this condition is unknown. However, in humans there is a period of increased susceptibility to gingivitis during the pubertal period. A genetic predisposition towards feline juvenile onset periodontitis has been reported in Siamese, Somali, and Maine Coon cats.

Clinical Features: Hyperplastic gingivitis appears as gingival enlargement and significant inflammation which is confined to the gingiva and begins during the eruptive period of the permanent dentition. Bleeding during mastication and on oral exam are common findings. While occasionally seen in dogs, this condition has a much higher incidence in cats. It is generally a non-painful condition for the patient, and halitosis is a common complaint. If left untreated, it typically proceeds quickly to periodontal disease, which may result in early exfoliation of the teeth. This disease is commonly mistaken for caudal stomatitis. The distinguishing clinical sign is the lack of caudal inflammation in this disease process. As the patient matures, susceptibility appears to subside at approximately two years of age.

In contrast, **juvenile periodontitis** does not involve enlargement of the gingiva and usually leads to the rapid proliferation of plaque and calculus and subsequent inflammation. This in turn results in significant early bone loss, periodontal pocket formation, and furcation exposure. This is generally the worst in around the mandibular first molars. Treatment and effective management of these cases is often exceedingly difficult.

Diagnostics:

Histopathology (via incisional biopsy) should be considered to rule out other causes of gingival inflammation. Culture and sensitivity testing is generally unrewarding, but may be of value in non-responsive cases. Dental radiographs should be performed to evaluate the quality of the alveolar bone and also for early tooth resorption. Finally, **Bartonella** testing may be beneficial in some cases, especially in patients who do not respond to traditional management practices.

Management:

In the management of both of these conditions, early (9 months of age) and frequent (q 6-9 months) dental prophylaxis (even if only minimal plaque is present) along with strict homecare is critical to decrease inflammation. Ideally, homecare consists of daily brushing, as it is the gold standard of plaque control. Other homecare alternatives include chlorhexadine rinses as well as plaque control diets and treats. In cases where gingival hyperplasia is present, early gingivectomy is recommended to remove psuedopockets, decrease inflammation, and facilitate plaque control (both professional and homecare). Finally, extraction of any significantly diseased teeth is warranted to decrease the degree of inflammation.

Key Points:

- The diagnostic key between caudal stomatitis and periodontal disease is the presence of inflammation in the caudal area.
- All fractured teeth in cats are suspect for endodontic disease
- Extraction is the treatment of choice for caudal stomatitis and tooth resorption.
- Dental extractions are critical for proper therapy

Further Reading:

Niemiec BA: Dental, Oral, and maxillofacial pathology, a color handbook. (Manson)

Niemiec BA: Dental Applications in Emergency Medicine (Practical Veterinary Publishing) www.practicalvetpublishing.com

Bellows, J: Feline Dentistry (Wiley Blackwell)

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TIPS FROM THE EXPERTS FOR THE MANAGEMENT OF...

CHRONIC DIARRHEA IN DOGS

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TIPS FROM THE EXPERTS FOR THE APPROACH AND MANAGEMENT OF

CHRONIC DIARRHOEA IN DOGS

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Definition

Chronic diarrhoea is used in dogs with diarrhoea lasting for longer than a three-week duration. It is a clinical sign that may result from primary gastrointestinal or extra-gastrointestinal diseases but is usually associated with chronic enteropathies.

Causes

In a recent publication by Volkman et al., primary gastrointestinal disease was found in 90% of the 136 dogs with chronic diarrhoea.[1] Of these, inflammatory diseases were the most prevalent (71%; FRD 66% dietary responsive, 23% SRD, 11% ARD), with infectious and neoplastic diseases less frequently characterised.[1] 10% of cases had metabolic aetiologies, including exocrine pancreatic insufficiency, endocrine, hepatic, renal and cardiac disease.

Approach

I) History and Clinical Examination

Useful information includes establishing the nature of diarrhoea, the demeanour of patient, the presence of weight loss, the presence of vomiting and associated frequency, appetite, and presence of puritis. Dietary history should include treats, access to table scraps, and previous diet trials.

Physical examination is often unremarkable, but attention should be paid to assess for pale mucous membranes, lymphadenopathy, oedema, and joint effusion. Also thickened loops of intestine, masses or a fluid wave on abdominal palpation. The examination should also include a digital rectal exam to assess for intra- or extraluminal masses, mucosal abnormalities or blood.

II) Laboratory

Routine haematology, serum biochemistry and urinalysis are often normal but may reveal evidence of hypoalbuminemia or panhypoproteinemia, hypocholesterolemia, increased urea, anaemia or the absence of a stress leukogram. A faecal examination is recommended to screen for parasites.

Serum trypsin-like immunoreactivity and an ACTH stimulation test may be used to investigate exocrine pancreatic insufficiency and hypoadrenocorticism respectively. Determination of serum cobalamin levels may be beneficial in patient management.

III) Imaging

Abdominal ultrasound is often performed as part of the investigation in chronic diarrhoea. The overall diagnostic utility, however, was found to be low in a study, with ultrasound not making a difference in 66% of cases, providing additional benefit in only 17%.[2] Sonography was shown to be most useful when abnormalities were already identified on physical examination (abdominal and rectal masses).[2] Even with gastrointestinal lymphoma, 26.7% of dogs had normal sonographic findings, highlighting limitations of the modality.[3]

Alternatively, evaluation of the GI tract via computed tomography has also been described. Helical acquisition of 2-5 mm slices in anaesthetised dogs allowed assessment of intestinal diameter and wall thickenss.[4]

IV) Dietary and Antibiotic trials

Although histopathology is perceived to be important, it cannot distinguish inflammatory bowel disease from food- and antibiotic-responsive diseases. Significant weight loss, poor body condition, anorexia, hypoalbuminemia or panhypoproteinemia, or sonographic evidence of significant infiltrative disease would indicate that endoscopy is appropriate earlier rather than proceeding with a therapeutic trial.[5]

Options for a dietary elimination trial include home-prepared or commercial novel protein diets, as well as hydrolysed diets. Food trials should be conducted for at least two weeks. Controversy exists between these choices as some dogs can tolerate home-prepared, single protein diets but not their commercially prepared versions. However, home-cooked diets can be unbalanced and inadequate. Choosing a novel protein may also be difficult depending on patient dietary history, and cross-reactivity between food antigens.[6-8] Hydrolysed diets were found to be superior to highly digestible ones in the management of chronic enteropathy, with significantly more dogs remaining in remission at three years.[9] However, concerns have been raised regarding hydrolysate diet being tolerated by most, but not all, of the dogs, sensitized

to the intact compounds.[10] Finally, many medications, especially OTC medications, may contain unwanted/hidden proteins.[11] These should, therefore, be taken into consideration in performing an elimination diet.

Antibiotic responsive diarrhoea is hypothesised to be a result of the following factors or a combination of them: defects in the mucosal barrier, altered mucosal immune response, and dysbiosis.[12] Choices of antibiotics that may be trialled include oxytetracycline, metronidazole and tylosin. Concurrent cobalamin deficiency should be managed with parenteral or oral cobalamin.

V) Biopsy

There are three means of obtaining representative tissue samples for histology: flexible endoscopy, laparoscopy, and surgery. Current recommendations are to perform upper and lower GI endoscopy as the preferred method of sampling.[5] Acquisition of 6–7 adequate or 10–15 marginal tissue samples is advised for histopathological evaluation.[5]

Advantages of endoscopy include visualisation and biopsy of mucosal changes that cannot be seen by a serosal approach during surgery; the collection of multiple tissue biopsies per site; minimal risk of perforation and septic peritonitis, compared to surgical biopsy; and decreased morbidity to the patient. Disadvantages include limited access to the entire GI tract and the inadequacy of tissue samples that may not permit diagnosis. Recent studies have highlighted the importance of concurrent ileal biopsies in providing valuable information not always found in duodenal or colonic biopsies.[13]

Monitoring Management

The canine IBD activity index (CIBDAI) and the canine chronic enteropathy clinical activity index (CCECAI) help assess response to therapy. The latter consists of both objective and subjective measures. A CCECAI of >12 has been associated with negative outcomes, along with hypocobalaminemia, hypoalbuminemia and duodenal endoscopic abnormalities.[14]

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CANINE KERATOCONJUNCTIVITIS SICCA – WHAT DO I DO WHEN CYCLOSPORINE DOES NOT WORK?

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Introductory Philosophy

Prior to and especially since the introduction of cyclosporine as a treatment for canine dry eye, I think we have had a strong tendency to consider keratoconjunctivitis sicca (KCS) as a simple deficiency of aqueous tear production. To be sure, this is justified (and reinforced on a daily basis in our clinics) because the vast majority of our canine patients respond so remarkably to topical administration of cyclosporine. Therefore, we sometimes need to remind ourselves that the nasolacrimal system consists of complex secretory, distributional, and drainage components all of which must act in superb harmony to effectively protect the corneal and conjunctival surfaces. In some ways, the fact that 0.2% cyclosporine ointment (Optimmune®) is so effective in the majority of KCS patients has made treating tear film disease truly fascinating. I enjoy the challenge of thinking about and better managing those less common cases that are unresponsive or only partially responsive to immunomodulatory stimulation of aqueous tear production. A complete but brief review of nasolacrimal anatomy and physiology is a necessary first step.

A CLINICIAN'S APPROACH TO THE LACRIMAL UNIT

Secretion

- Orbital and third eyelid lacrimal glands that produce the aqueous component of tears
- Tarsal (or meibomian) glands, which are modified sebaceous glands that secrete an oily fluid similar to sebum and responsible for reducing evaporation of the aqueous component of the tears.
- Conjunctival goblet cells, especially those in the ventral fornix, that produce mucin, which improves retention of the aqueous tears by the hydrophobic corneal epithelium.

Distribution and loss

The composite preocular tear film **produced** by lacrimal, tarsal and conjunctival glands is critical for corneal, conjunctival, and general ocular health. It is **distributed** by normal blinking and movements of the third eyelid and globe before pooling in a space referred to as the "lacrimal lake" between the anterior faces of the cornea and third eyelid and the posterior margin of the lower eyelid. A percentage of tears determined in large part by tearfilm stability (and therefore composition), ocular surface topography, and blink rate (determined in part by skull shape, corneal sensitivity, emotional state) then

evaporates or is lost over the eyelid margins. Excess tears **drain** via upper or lower nasolacrimal puncta into the nasolacrimal system.

Qualitative vs. Quantitative Tear Film Deficiencies

Classically, **qualitative** deficiencies describe biochemical alteration of a component of the tear film, while **quantitative** deficiencies describe decreased volume of a tear component. Because the aqueous component of tears comprises the major volume of the tear film, qualitative tear film disturbance and KCS are sometimes considered synonymous. The majority of canine KCS is believed to be due to an idiopathic, but immune mediated dacryoadenitis involving the lacrimal glands. These cases are most likely to respond to therapy with topical cyclosporine A (CsA). This treatment is so reliably successful in immune-mediated dacryoadenitis, that I wonder if failure to consider other causes of KCS is possibly the most common cause of poor response to this standard therapy.

using the "DAMNIT" list to direct examination and testing

As the internists have taught us, a logical approach to apparently idiopathic or disease or cases unresponsive to "best guess" therapy is very wise. I think this is particularly true for canine dry eye patients unresponsive to topical administration of CsA. Here's a few causes I consider (for completeness, I have included feline as well as canine causes):

Developmental KCS (acinar hypoplasia) is reasonably common in Yorkshire terriers and other toy breeds and is often associated with absolute sicca (STT = 0). Curiously, this can be unilateral. As might be expected, this form of KCS is unlikely to respond to topical CsA and is one of the more challenging forms to treat, usually requiring parotid duct transposition.

Autoimmune disease with mononuclear cell infiltration and fibrosis of the lacrimal gland is the most common etiology for KCS in dogs. The stimulus for this disease is unknown, however the observation that it occurs more commonly in some breeds suggests that a familial predisposition may exist. Commonly-affected breeds are West Highland White Terriers, Bulldogs, and Cocker Spaniels. These patients seem the most likely to respond to CsA.

Metabolic causes of KCS are limited. Although some studies suggest an association between KCS and certain endocrine diseases such as diabetes and hypothyroidism, this is not universally proven. Regardless, concurrent treatment of any endocrinopathy and topical application of CsA would appear wise and may improve prognosis.

Neoplasia of the lacrimal glands is rare; however glandular dysfunction can be seen in association with

any form of orbital disease, particularly cellulitis or space-occupying masses. Exophthalmos or strabismus with reduced globe retropulsion should arouse suspicion of such a cause and prompt orbital imaging. Hypovitaminosis A has been associated with **nutritional** KCS; however this is most common in food animals.

Infectious etiologies of reduced aqueous production include distemper virus in dogs and feline herpesvirus (FHV-1) in cats. In these diseases, signs of KCS are usually overshadowed by more overt ocular or systemic lesions. However, assessment of tear production and supplementation of the tear film when necessary should be a routine part of management of these diseases. Unlike other causes of KCS, tear production usually resumes if the primary infectious etiology is resolved. Perhaps of more relevance is the way in which infectious diseases may affect tear quality through destruction or dysfunction of the conjunctival goblet cells and meibomian glands. For example, conjunctivitis of any cause, is often associated with reduction in goblet cell density, an unstable tear film and worsening conjunctival (and sometimes corneal) disease – thus setting up a "vicious cycle". Likewise, bacterial blepharoconjunctivitis or orbital cellulitis may also extend to the tarsal and orbital lacrimal glands respectively. Surgical removal of the third eyelid gland following third eyelid gland prolapse (or cherry eye) can be an **iatrogenic** cause of KCS.

Traumatic disruption of the lacrimal gland, its blood supply, or innervation (CN V or VII) is a known cause of KCS. Trauma may be anatomically distant from the gland if the nerve or vascular supply is involved. Possibly one of the most common causes of neurologic KCS is injury to the facial nerve, particularly in association with middle ear disease. Neurogenic reduction or failure of blinking due to facial nerve dysfunction and/or dysfunction of the sensory fibers of the trigeminal nerve can exacerbate KCS in these cases. Concurrent desiccation and crusting of the ipsilateral nostril (xeromycteria) strongly suggests neurogenic dysfunction. The most commonly incriminated **toxic** causes of KCS are sulphur drugs and atropine; however etodolac (Etogesic®) appears to be associated with a rapid onset of usually absolute sicca poorly responsive to cessation of the drug and/or administration of cyclosporine. General anesthesia and sedation can also cause a temporary depression of STT values.

Regardless of cause, the pathogenesis and end result of deficient aqueous production is multifactorial. Surface dehydration, hypoxia and necrosis of surface tissues, accumulation of exudates, and secondary infections are important mechanisms.

Clinical signs

Clinical signs will always be the initial alert that tear film dysfunction should be considered. The classic signs of aqueous tear film deficiency are familiar, with the hallmark clinical sign being accumulation of tenacious, adherent mucopurulent discharge over the corneal surface, conjunctiva, and eyelids. The mechanisms underlying this are likely over-production of mucins to compensate for aqueous deficiency as well as reduced hydration and flushing of those mucins secreted. It should come as no surprise to us therefore that analogous mechanisms are at play in mucin or lipid deficiency. For example, reductions in the quantity or quality of ocular surface mucins should be expected to cause a compensatory increase in aqueous production (and likely a less visible increase in lipid production) as well as a tear film that is less well "anchored" to the ocular surface. In other words one of the signs of qualitative tear film deficiency may well be... epiphora!!

Somewhat irrespective of which tear component is deficient, the secondary corneal changes are relatively non-specific and reflect the chronic, irritating nature of the disease. These include a lackluster corneal surface (especially with aqueous deficiency), superficial corneal vascularization and pigmentation, and sometimes (if the onset of dry eye is acute) corneal ulceration. Ocular discomfort and conjunctival thickening due to squamous metaplasia are also common.

I am always careful to thoroughly assess all visible conjunctival regions (especially the deep fornicial regions) using both diffuse and a slit beam. In particular, I look for evidence of thickening/cellular infiltrate, chemosis, hyperemia, follicles, papillary conjunctivitis, or excessive folding. I also pay particular attention to the meibomian gland profiles visible through the palpebral conjunctiva and any glandular secretions naturally occurring or forcibly expressed from the gland orifices. Look particularly for secretions that are more difficult to express, more opaque than translucent, thicker or "waxier" than normal, or those that form a small inspissated "bubble" from the orifice (so-called "choked" meibomian glands)

Diagnostic testing

The "workhorse" of dry eye testing in veterinary medicine is of course is **Schirmer's tear test type 1 (STT-1)**. In dogs, I consider STT values less than 15 mm/minute in conjunction with consistent clinical signs diagnostic. However, it is important to recall that the STT-1 result merely reflects the volume of tear film in the **lacrimal lake** plus the volume of **reflex tears stimulated** to be **produced** and **released** by the STT strip gently abrading the cornea. It is interesting to ponder, therefore, the effects of lid conformation, emotional state, corneal sensitivity, placement of the STT strip (medially, centrally or laterally in the ventral conjunctival fornix), lacrimal gland function, and patency of the lacrimal gland ductules. I am confident that a patient cold have

normal tear production but a sufficiently dysfunctional tear delivery system (due to conjunctivitis-induced compression of the lacrimal gland ductules) to (likely reversibly) reduce his STT-1 result. I do not routinely use **STT-2** (STT following topical anesthesia) or **STT-3** (STT following or during a noxious stimuli) in canine patients but am beginning to appreciate their value in cats.

Despite the utility of the STT, there are many other potentially underused tests that are of value – particularly in those patients who are unresponsive to topically applied CsA. I like to use an **assessment of blink rate and effectiveness**. It amazes me – especially in many brachycephalic breeds how poorly and infrequently they blink. Unless they have a remarkably increased tearfilm stability to compensate for this, one must assume that they have greater evaporative losses than dolichocephalic dogs. Perhaps these are patients who would benefit from a medial canthoplasty. Our best clinical test of tear film stability in vivo appears to be the **tear film breakup time (TFBUT).** Although patient compliance sometimes makes this test difficult, I believe that it provides highly valuable information in select patients. As we learn more about this test, we would do well to pay attention to what the physicians have known for some time about performing this test very consistently especially with regards timing relative to the rest of the exam, amount of fluorescein applied. I think that the specially prepared Dry Eye Test (DET) strips by Amcon Labs (www.dryeyetest.com) are worth considering. The normal range has not been established using sufficiently large population of normal dogs of various skull shapes, but most manuscripts to date report mean ± SD values of around 20 ± 5 seconds.

In patients where the clinical exam suggests it may be informative, I consider **culture and sensitivity and cytology of expressed mebum** and/or an **eyelid (meibomian gland) and/or conjunctival biopsy**. If I am interested primarily in the conjunctiva (and especially the goblet cells) I simply do a snip biopsy of the fornicial conjunctiva under topical anesthesia. In patients where I am more interested in the entire qualitative tear film unit I do a full-thickness punch biopsy from dermis to conjunctiva through an affected area of the eyelid. If there is marginal disease, I consider a wedge biopsy. In all cases, I work with our in-house ocular pathologist to ensure **goblet cell density (GCD)** is reported. These are typically calculated (and reported) as a percentage of the non-goblet conjunctival epithelial cells. Like TFBUT, the number of normal dogs which have been sampled and assessed in a uniform manner is insufficient to permit the statement yet of a true reference range, and there is much variation in GCD according to site sampled; however the GCD of the palpebral/fornicial sites (which are the most readily sampled) are typically reported to be around 20-30%. The periodic acid Schiff (PAS) staining technique can greatly facilitate counts.

An often overlooked but critical component of the exam of some dry-eye patients is **assessment of corneal sensitivity (or corneal touch threshold – CTT)** using the Cochet-Bonnet aesthesiometer. If we recall the critical role of the trigeminal nerve in sensing ocular surface dryness, reflex and basal tearing, reflex and basal blinking, and carriage of the parasympathetic fibers of lacrimation as well as trophic factors for the ocular surface, it is difficult to underrate the importance of normal function of this nerve to the lacrimal unit. It is involved in the afferent and efferent arm of tear production and delivery, and in tear distribution and retention via normal lid position and blinking.

In all cases, the ocular surface should be stained with **vital dyes**. It is critical to recall that these stain the corneal epithelium (rose bengal or lissamine green) or subepithelial collage n (fluorescein) of both conjunctiva and cornea and the entire visible ocular surface should be examined following stain application.

Recalling the DAMNIT list facilitates an efficient but directed examination of the body systems and signs sometimes associated with those less common causes of KCS are essential. I include a thorough history directed at the known causes, followed by examination for associated systemic diseases, a thorough assessment of cranial nerve function, especially palpebral and corneal reflexes, and careful evaluation of upper, lower, and third eyelids. This must include assessment of their position in relationship to the cornea, and appearance of eyelid margins, cilia, and the meibomian glands and orifices. Globe retropulsion and jaw opening, "slipping" the oral mucous membranes, and assessment of the nares for dryness is also essential – sometimes in association with an otic exam. Culture and sensitivity, along with cytology is unnecessary as microbial overgrowth is secondary and typically responds as soon as tear production is improved.

TREATMENT

My five main treatment goals:

- Always diagnose and treat the underlying cause if possible. (This is <u>especially</u> important in patients unresponsive to CsA)
- Minimize further tear loss and maximize tear distribution
- Stimulate of tear production (CsA irrespective of cause)
- Supplement the tear film in a manner that considers which of the components is inadequate
- Treat or prevent secondary infection

<u>Underlying causes</u>

Thorough attention to the "DAMNIT" list, a careful assessment of history and clinical signs, and appropriate diagnostic testing will facilitate recognition of any underlying cause, expedite appropriate treatment, and improve prognosis for full return of secretory function.

43RD WORLD SMALL ANIMAL VETERINARY ASSOCIATION CONGRESS AND 9TH FASAVA CONGRESS

Minimization of tear loss and maximization of tear distribution

Minimization of tear loss and maximization of tear distribution relies on a thorough assessment of lid anatomy and function. Many dogs with only marginal tear production can be made more comfortable with correction of mild ectropion or entropion, removal of distichia, and/or reduction of palpebral fissure size.

Stimulation of normal tear production remains the main goal of medical therapy

Tear replacement products are no substitute for improved production of endogenous tears with their multitude of immunologic and nutritive factors, and appropriate pH and osmolarity. **Cyclosporine** remains the most effective drug for this purpose in my opinion. In addition to its ability to reduce immune-mediated infiltration of the lacrimal gland, this compound has a direct lacrimogenic function, and it promotes mucin production from conjunctival goblet cells. Its direct lacrimogenic function appears to rely on frequent application, while immunosuppression and remodeling of glandular tissue presumably require more chronic use. Therefore, in most cases this drug should be instituted twice daily and the patient rechecked in approximately 2 weeks. It is important that the client be instructed to apply CsA as scheduled right up until the time of recheck examination. Omitting the morning treatment because the dog was going to be examined later that day may cause an artificial depression in STT values. Clients should also be advised that initial response to therapy is best judged by change in STT values, mucoid discharge, and ocular comfort, rather than decrease in pigmentation or corneal vascularization. Improvement in these corneal changes occurs at a similar rate to that which they occurred – slowly. Tapering of dose frequency or product concentration is typically not possible and should be based on clinical and measured (STT) responses. Failure to respond to 0.2% CsA BID is a reason to trial a higher concentration such as 1% or 2%. In my experience, increased frequency beyond BID does not have a satisfactory effect.

Information regarding **tacrolimus** is encouraging. This drug acts by a similar mechanism to CsA but is more potent and operates via a different cellular receptor. Reports confirm that it is effective in some cases that are unresponsive to CsA. It is compounded in various ways by many pharmacies. To date I am aware of data for a 0.02% aqueous and a 0.03% suspension in olive oil only. Although its safety and efficacy as an ophthalmic drug in dogs have been preliminarily tested, an FDA alert in the USA suggests that topical application of this drug as a dermatologic preparation in humans, especially children, may be associated with development of lymphoma and squamous cell carcinoma. The FDA currently recommends that tacrolimus be used only when other

drugs have failed or not been tolerated, and then with caution. I follow this guideline for our veterinary patients too. Consider recommending that clients wear gloves when handling this product and that children do not administer the drug to their pets.

Some advocate use of **topical corticosteroids** to further reduce dacryoadenitis. This has some rationale but requires caution in an eye that is already more prone to ulceration. Addition of a topical, penetrating steroid such as dexamethasone or prednisolone after initial treatment with CsA has successfully promoted some tear production and improved corneal health may be justified.

Cholinergic agents such as **pilocarpine** may be used to provide parasympathetic stimulation of the lacrimal gland. This alternative mechanism might be expected to be more physiologic and therefore likely to succeed in cases of neurogenic KCS than more common cases of immune-mediated dacryoadenitis. Topical use of this drug is very irritating, produces a noticeable uveitis, and may not provide adequate drug concentrations at neurologic synapses. This has led to the suggestion that oral dosing on an empirical but individualized basis is necessary. This requires that the dose be titrated to just below systemic toxicity in each animal. Signs of toxicity include vomiting, diarrhea, and bradycardia. Ophthalmic pilocarpine is used orally usually via a doctored food bolus. One dosage recommendation (credit Dr. Randy Scagliotti) is that 1% pilocarpine is used for dogs $\leq$ 4 kg, 2% for dogs weighing 4-20 kg, and 4% pilocarpine for dogs $\geq$ 20 kg. The initial dose is one drop PO twice daily for three days. This dose is increased by one drop every three days until the earliest signs of toxicity (usually vomiting or anorexia without diarrhea) are observed. The drug is discontinued for 24 hours or until GI signs abate and then re-instituted at the highest dose which did not produce signs of toxicity. Because of the different mechanism by which CsA acts and because of its additional desirable effects, the two drugs are expected to be synergistic. There is a case report supporting the addition of a topical sympathomimetic eye drop to this regimen, and my personal experience supports this. I use 2.5% phenylephrine. While this seems counter-intuitive at first, it appears that there are smooth muscle fibers associated with the lacrimal glands that act via contraction to express tears over the eyes. Thus the initial use of pilocarpine to stimulate tear production followed by the **addition** of phenylephrine to stimulate tear secretion has been advocated by some. We have tried this with remarkable results in a small number of dogs.

Artificial tears

Supplementation of tears has traditionally been provided in one of three forms: aqueous ("artificial tear") solutions, more viscous polymers or methylcellulose solutions, and ointments in a petrolatum base. However, no

product currently available adequately replaces all of the functions served by tears. As such, application of tear supplements can have a dilutional effect on those tears being naturally produced. In addition, any product (and especially the preservatives most contain) can cause surface irritation. Finally, tear supplement solutions may require extremely frequent application to be effective. These factors have made this a problematic area in veterinary medicine. Commercial introduction of hyaluronan tear replacement products has provided an important adjunctive therapy for most dogs with KCS. These products have mucinomimetic properties and some are available in a preservative-free formulation. They are extremely well tolerated in dogs and cats. I typically use hyaluronans early in the treatment schedule while CsA is being introduced but often continue them even if adequate tear production returns.

Secondary infection

Secondary infection is common when tear quality or quantity declines. This is best treated with a well-tolerated, reasonably broad-spectrum antibiotic with the major goal being control of normal Gram-positive flora overgrowth. Triple antibiotic (neomycin-polymyxin-bacitracin) ophthalmic ointment is an excellent choice. This can be discontinued as soon as STT values improve and mucopurulent discharge declines since chronic topical antibiotic therapy is contraindicated for maximal ocular surface health.

Parotid duct transposition

It is my opinion that parotid duct transposition (PDT) is associated with significant complications in some patients and does not obviate the need for ongoing medical management. Therefore, medical management is the preferred method of treatment and should always be attempted first. I reserve PDT for those cases in which a thorough clinical examination has failed to reveal a cause and which have not responded to protracted and multiple medical therapies – typically patients with congenital glandular aplasia/hypoplasia.

SVA REHABILITATION

NEUROMUSCULAR ELECTRIC STIMULATION

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NEUROMUSCULAR ELECTRIC STIMULATION

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Therapeutic plans in veterinary rehabilitation generally involve a combination of manual therapies (joint mobilizations and soft tissue mobilizations), physical modalities (laser, therapeutic ultrasound, e-stim, shockwave), and therapeutic exercises. The modalities are generally used to prepare the tissues for the manual therapies and therapeutic exercises. Physical modalities should never be the sole therapeutic method applied to any patient.Therapeutic parameters for each modality are chosen based upon the acuity of the injury, so the therapist must be well versed on the definitions of the acute, subacute, and chronic phases of healing. The mechanism of action for neuromuscular electrical stimulation (NMES) is electrical stimulation causing muscle contraction. The mechanism of action for transcutaneous electrical nerve stimulation is electrical stimulation at a frequency that does not cause muscle contraction but that blocks afferent input leading to pain relief.

NMES has many applications in veterinary rehabilitation including reduction of edema, muscle reeducation, reversal of atrophy, and wound healing. At the cellular level, NMES causes excitation of nerve cells leading to changes in cell membrane permeability, stimulation of protein synthesis and stimulation of fibroblast and osteoblast activity. Wound healing is enhanced through increased blood flow to the area and stimulated cell turnover.TENS is used for pain control. In very simplistic terms, TENS works along the lines of the Gate Theory introduced by Wall and Melczak in the 1960's: the severity of pain sensation is a result of the balance between the excitatory and inhibitory inputs to the T cells in the spinal cord. By stimulating peripheral nerves at proper frequencies, the excitatory nociceptive inputs can be blocked from traveling to the brain where pain is recognized. TENS has a short-lived effect, lasting perhaps up to an hour after the application is stopped. It is used in veterinary rehabilitation as immediate

post-operative pain control and during therapy sessions to allow pain free movement during exercises. Electrotherapy variables include intensity, mode, ramp duration, on:off time, pulse width, frequency, treatment duration, and treatment frequency. Intensity is measured in milliamps (mA), and is generally between 50 and 400mA. Intensity determines how many nerve fibers are stimulated. Mode can be set to constant, synchronous, or alternate. In constant, there is a constant flow of current through both channels. In synchronous, both channels turn on and off simultaneously. In alternate, the one channel turns on as the other turns off. Ramp durations is the time that the stimulus takes to reach peak intensity and is measured in seconds. On:Off time is the ratio of time during which the intermittent current is on and is measured in seconds. Pulse width is the length of time that pulses of electricity are on and is measured in microseconds. Pulse width determines which nerve fibers are stimulated, and widths of greater than 150 microseconds are more comfortable for the patient.Frequency, measured in Hertz (Hz) or pulses per second, is the speed at which pulses enter the body, and determines the amount of muscle tension that is developed. Higher frequency is more comfortable. Frequencies of 1 to 10Hz are used for pain management, while frequencies of 25-50Hz generate strong tetanic muscle contractions. Treatment duration is generally between 15 and 20 minutes, and treatment frequency is generally at least two to three times per week. Depth of penetration is determined by the distance between the electrodes.Electrodes must be flexible to conform to the patient's muscles, low resistance (less than 100 ohms), highly conductive, reusable, inexpensive, and appropriately sized for the patient. A medium, usually gel, is necessary to transmit the current between the electrode and the patient's skin.

WSV18-0180

POLYCYTHEMIA - DIAGNOSTICS AND MANAGEMENT OF A HIGH HEMATOCRIT

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Although strictly speaking polycythemia implies a rise of all blood cell counts above normal, the occurrence of leukocytosis and thrombocytosis along with erythrocytosis is exceptionally rare in companion animals. Hence, polycythemia would be more appropriately called erythrocytosis and clinically refers to an increase to an above normal red blood cell count, hematocrit or packed cell volume (PCV), and hemoglobin (Hb) concentration. Polycythemic animals will not show clinical signs until the PCV reaches >60% with some of the highest PCVs exceeding 85%. Because of an underappreciation of the normal upper limit of the PCV in cats (48%) versus dogs (56%), polycythemia is generally underdiagnosed in cats. Based upon blood volume and red cell mass, polycythemia can be divided into relative and absolute polycythemias, which represent completely different conditions, both requiring immediate but opposing therapeutic interventions.

Relative polycythemia is characterized by an elevated PCV in the presence of a normal (or even decreased) total red blood cell mass. This is usually due to a decrease in plasma volume associated with severe dehydration or increased serum total proteins, e.g. profound vomiting and diarrhea, or severe burns. The hematocrit is generally only mildly increased, therefore relative polycythemia is rarely associated with signs of hyperviscosity, and the clinical features of the underlying disorder prevail. Because of the obvious signs of dehydration, relative polycythemia is usually easily recognized and simply corrected with aggressive fluid therapy.

Absolute or true polycythemia is characterized by an expanded red blood cell mass. Splenic contraction is an unlikely cause in dogs and cats, as it only marginally increases the PCV. The blood volume and red cell mass could be determined by labeling red cells radioactively or with biotin. This is, however, rarely if ever needed in clinical practice, as dehydration can be readily excluded as cause of relative polycythemia. More difficult is the differentiation of absolute polycythemia into **primary or secondary polycythemia**, which depends on whether the condition is erythropoietin independent or dependent.

Some clinicians readily equate absolute erythrocytosis

with polycythemia vera (P. vera) without considering other differential diagnoses. Clinical experience, however, suggests that P. vera may occur rarely compared to other forms of polycythemia. New insight into the causes of primary and secondary polycythemias has been gained in human and veterinary medicine. However, if the underlying condition cannot be corrected, lowering the PCV into a safe range in patients with absolute polycythemia may be successfully accomplished with repeated phlebotomy and, if needed, chemotherapy.

PRIMARY POLYCYTHEMIA

Primary polycythemia appears erythropoietin independent and has often been considered synonymous with P. vera in small animals, although additional forms of primary polycythemia need to be considered. In fact, animals with a presumptive diagnosis of primary polycythemia as well as cases with an early presentation in life that continues over a chronic course of many years, point strongly to the existence of other forms of primary polycythemia in dogs and cats. More research is needed in small animals to determine, if such processes are occurring in these polycythemic patients who do not clearly have P. vera.

Polycythemia vera is a myeloproliferative clonal disease that arises from a multipotent hematopoietic progenitor cell in the bone marrow. A single transformed stem cell gains a selective growth advantage and becomes the predominant source of marrow precursors, and the clonality of the bone marrow cells of human patients with P. vera has been documented. P. vera, therefore, results in the accumulation of morphologically normal red blood cells, and less commonly white cells and platelets, and their progenitor cells in the absence of a definable stimulus. Granulocyte and platelet counts in the blood would be expected to be increased, but are usually normal. The bone marrow aspirate is consistent with erythroid hyperplasia, but is not diagnostic for a myeloproliferative disease in humans. There are no cytologically characteristic features of the bone marrow cells in P. vera as hematopoietic cells appear to fully mature. Classically, human patients have serum erythropoietin levels in the low to normal range and erythroid progenitor cells that proliferate and mature independent of erythropoietin, but these culture assays are not robust and readily available. In humans with P. vera, Jak-2 mutations have been documented and in a couple of dogs Jak-2 mutations have been found but the test for Jak-2 mutation is not clinically available in veterinary medicine. Hence, the diagnosis of P. vera is still based on the exclusion of other causes of erythrocytosis.

SECONDARY POLYCYTHEMIA

Secondary polycythemia refers to a group of diseases triggered by an exaggerated erythropoietin dependent stimulation of red cell production. This may be considered an **appropriate** response in which the erythron is responding normally to generalized tissue hypoxia or **inappropriate** in which the erythropoiesis is being stimulated by an aberrant production of erythropoietin or due to local renal hypoxia.

Appropriate PCV rises are seen in high altitudes and with cardiopulmonary disease such as congenital heart defects with right to left shunts (ventricular septal defects, reversed PDA, Tetralogy of Fallot) and rarely chronic obstructive pulmonary diseases. Cats with cardiac shunting usually die before they can develop signs of polycythemia. Furthermore, an appropriate secondary polycythemia has been documented in several breeds of dogs and domestic shorthair cats with hereditary methemoglobin reductase deficiency, which results in the erythrocytes' inability to carry oxygen. Similarly, chronic carbon monoxide intoxication can cause polycythemia with pink mucous membranes.

Inappropriate absolute polycythemia includes renal diseases, as well as tumors producing erythropoietin, and is typically associated with typically high serum erythropoietin levels. Various renal tumors, including nephroblastomas and carcinomas, may result in renal hypoxia and thereby cause elevations in serum erythropoietin and, consequently, inappropriate secondary polycythemia; whereas erythropoietin-producing tumors in other tissues have rarely been documented. Inappropriate secondary polycythemia of renal origin may also be rarely caused by amyloidosis, polycystic kidney disease, glomeronephritis, and renal fibrosarcoma and lymphoma. In most cases increased serum erythropoietin concentrations were documented or an association was established based upon the resolution of the polycythemia following the resection of the mass in animals and humans.

CLINICAL SIGNS

The clinical signs of relative polycythemia are easily recognized and will not be further discussed here. Clinical signs of **absolute** polycythemia are characterized by manifestations of the underlying disease process and are associated with hyperviscosity and the expanded blood volume. They include hyperemic or cyanotic mucous membranes (due to cardiopulmonary disorders and methemoglobinemia), hemorrhage (epistaxis and hyphema), and neurologic disturbances such as lethargy and seizures. Neurologic signs are the most common presenting complaints, but with the advent of more frequent health screens including complete blood cell counts, the erythrocytosis may be discovered earlier as an incidental finding. Cyanosis or renal size abnormalities may suggest a particular organ failure as well as mechanism, and define the type of the polycythemia. However, secondary cardiac and renal changes due to erythrocytosis may also be observed and confound the

interpretation. Polydipsia and -uria and splenomegaly has been seen in animals assumed to have P. vera and rarely with other polycythemias.

Diagnostic Tests

As **relative** polycythemias are readily recognized, confirmation of a normal blood volume may not be required in clinical practice. In fact, patients with absolute polycythemia often have an expanded blood volume. Diagnostic tests for **absolute** polycythemia include a complete blood cell count, absolute reticulocyte count, chemistry screen, urine analysis, blood gases and pulse oxymetry, chest and abdominal radiographs, cardiac examination and an ultrasound to evaluate the kidneys and liver. Cyanosis may only be noted caudily. Dark blood may be exposed to air to determine if it is deoxyhemoglobin or methemoglobin. Serum erythropoietin values, determined by a species-validated assay, may be elevated in cases of secondary polycythemia, but a normal to low erythropoietin level does not rule out secondary polycythemia. An increased absolute reticulocyte count in light of a polycythemia supports the exaggerated hematopoietic response and documents the presence of an absolute polycythemia. However, a bone marrow aspirate for cytologic examination adds no new information, since it fails to differentiate between primary and secondary polycythemias: the myeloproliferative disease resulting in P. vera has no characteristic cytologic or pathologic features of malignancy

Treatment

The treatment of relative and absolute polycythemia is clearly different. In emergency situations, patients with relative polycythemia will respond to fluid therapy, whereas patients with absolute polycythemia may need to be treated by phlebotomy. If possible, renal and other tumors/masses should be removed or treated and cardiopulmonary distress should be corrected. Animals with methemoglobin reductase deficiency may not need to be treated except during severe stress situations with methylene blue (1 mg/kg IV once). In severely polycythemic patients repeated phlebotomies at no more 20 ml/kg (10 ml in cats) per session with or without simultaneous fluid replacement is the initial approach to lower the PCV to <60%. Phlebotomies can be repeated on a daily basis until the target PCV has been reached. In animals with absolute polycythemia, fluid administration beyond replacement may be associated with cardiopulmonary failure as these animals are completely volume expanded. In cases that require multiple phlebotomies in a short period, replacement of coagulation factors and albumin with plasma may need to be considered. The target hematocrit is higher in dogs than in cats, as well as in cases with cardiopulmonary disease, but generally is greater than 50%.

Longterm control of absolute polycythemia may be achieved by periodic phlebotomies, radioactive phosphorus, and/or chemotherapy. Chemotherapy with hydroxyurea at 10-25 mg/kg twice daily to once every other day is commonly used. Animals treated with chemotherapy need to be monitored, not only by PCV measurements as for phlebotomized patients, but also by complete blood cell counts to identify drug induced cytopenias. Hydroxurea has also caused nail sloughing. Furthermore, antithrombotic doses of aspirin (1 mg/kg per day) may be considered, but there is no proof that it reduces the risk of thrombosis, and higher doses may lead to increased bleeding tendencies. Polycythemic animals may remain asymptomatic for weeks to years and, in some cases, can be successfully managed for years. Longterm follow up has not been reported in animals, although the author has observed persistent polycythemia for more than a decade in certain animals.

WSV18-0156

UPDATE ON THE USE OF SUBCUTANEOUS URETERAL BYPASS (SUB) IN CATS

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THE SUBCUTANEOUS URETERAL BYPASS (SUB) IN CATS

FOR NON-MALIGNANT URETERAL OBSTRUCTION

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Learning Objective: To understand the indications for placement of a SUB device and the technical procedural aspects of SUB placement.

Ureteral obstructions in cats are a frustrating condition to diagnose, manage and treat effectively. The onset of clinical signs are often vague and non-specific, especially if partial or unilateral obstruction is present.

Diagnosis is made by abdominal radiography and ultrasonography. Ultrasonography is the preferred imaging modality as it allows assessment of the kidney parenchyma and renal pelves, ureters and bladder. Importantly it allows for detection of radiolucent uroliths as well as obstructions or non-urolith obstructions of the ureters.

Cats with a diagnosis of ureteral obstruction should initially be treated medically with intravenous fluid therapy, analgesia and correction of any electrolyte opr acid-base disturbances, especially hyperkalemia.

Ureteral obstruction is most commonly due to urolithiasis with calcium oxalate being the most common form of urolith identified. Other causes of ureteral obstruction include tumours, strictures and blood clots and circumcaval ureteral anatomy. The normal feline ureter is only 2-3mm in diameter.

Surgical options to treat urethral uroliths include; ureterotomy or retrograde flushing the urolith to the renal pelvis and then urolith removal by pyelotomy or nephrotomy. Both of these surgical options had a high risk of urine leakage or ureteral stenosis. If the stone is relatively distal in the ureter, ureteral transection and reimplantation into the bladder is a possibility.

The Subcutaneous Ureteral Bypass (SUB) device was developed to provide an alternate pathway for urine to flow from the renal pelvis to the bladder. SUBs are placed during an open abdominal surgery using fluoroscopy. The SUB device consists of a pigtail catheter that is placed through the renal parenchyma into the renal pelvis with the aid of a guidewire and intraoperative fluoroscopy. This catheter exits the abdominal cavity through the body wall and is connected to a flushing port located in a paramedian subcutaneous position. The other end of the port is connected to another catheter that enters the abdomen and is placed in the bladder, thus providing an 'artificial ureter' bypass pathway for urine to flow from the renal pelvis to the bladder.

Potential complications with the SUB device include: blockage of the catheters with urolith or calcium oxalate encrustation recurrent urinary tract infections and kinking of the SUB tubes. The SUB device should be flushed via the subcutaneous port at the end of surgery and within the first month after surgery and then every 3-6 months for life. A recently special flushing mix and kit is now available. The flushing is normally done with any light sedation and the renal pelvis is imaged with ultrasonography to document renal pelvis dilation during flushing patency which indicates patency of the nephrostomy catheter.

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WSV18-0184

THE ROLE OF ANIMAL SHELTERS IN DISASTER MANAGEMENT

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In case of natural disasters, and other emergencies, members of the public will often be offered shelter by organizations like the Red Cross and other humanitarian organizations or government institutions. Such shelters are generally set up to deal with human health and safety, but rarely will consider or permit companion animals. Animal shelters can play a vital role in these circumstances and be a life-saving solution for both animals and humans, as we have seen several examples of pet owners refusing to evacuate a dangerous area and leave the pets behind, thereby endangering their own lives to keep their pets safe.

The purpose for most animal shelters is to provide refuge for animals that are abandoned, lost or otherwise homeless until they can be reunited with their owners or re-homed. In case of disasters a sheltering organization may be faced with the request to quickly house large numbers of animals. This often requires additional emergency housing to be set up in already existing facilities, or assembly of temporary structures for sheltering until the animals can be returned to their owners, moved to more permanent facilities, adopted into new homes, or, if warranted, euthanized. Emergency sheltering equipment is then typically disassembled, and supplies stored until the next crisis arises.

While many shelter medicine principles apply to both emergency shelters and traditional brick-and-mortar facilities, there are significant differences that must be recognized to ensure humane animal care in a disaster situation. This lecture aims to provide practical recommendations on how to best maximize available resources to properly care for animals during and following a disaster. Some the topics to be considered are suitable spaces for a emergency shelters, housing unit requirements, sanitation, staffing, and how to provide humane animal care and medical care.

WSV18-0054

RIDING THE WAVES- COMMUNICATION AND SUPPORT IN THE VETERINARY

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The Riptide Project is a global and multidimensional non-profit initiative aimed at improving veterinary mental health and wellbeing in veterinary professionals. Veterinary professionals are 2-4 times more likely than members of the general public to commit suicide. The risk factors related to veterinary suicide are varied, but range from character predispositions, financial stresses, inadequate support, and ready access to means[2]. Furthermore, vets and vet nurses self-identify very strongly with their careers, and often struggle to leave the profession, even if they have reached dire straits[3].

Excluding client factors, about 47% of respondents from a DVM360 survey on job satisfaction reported that workplace relationships and time management issues contributed the most to their on-the-job stress[4]. Veterinary professionals all enter the industry with the same desire to work with and help animals. It's time we helped each other.

The Riptide Project aims to promote conversations between veterinary individuals and organisations. We do this in two main ways – posting stories of veterinary professionals from all over the world, and connecting veterinary professionals through the "Riptide Cuppas" system.

Through face-to-face and online support, veterinary professionals from varied cultural and work backgrounds will be able to connect with likeminded people, both on home ground and abroad. The networking opportunities also open avenues for employment, and allow the diversification of veterinary careers outside of clinical practice.

The Riptide Project was launched to a global audience at the 2017 WSAVA/FECAVA Congress in Copenhagen, Denmark. We look forward to engaging, learning from, and working with, other veterinary professionals who are passionate about improving mental health and wellbeing in the industry in Singapore.

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WSV18-0048

SVA REHABILITATION

THERAPEUTIC ULTRASOUND: INDICATIONS AND CONTRAINDICATIONS

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THERAPEUTIC ULTRASOUND: INDICATIONS AND CONTRAINDICATIONS

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Therapeutic plans in veterinary rehabilitation generally involve a combination of manual therapies (joint mobilizations and soft tissue mobilizations), physical modalities (laser, therapeutic ultrasound, e-stim, shockwave), and therapeutic exercises. The modalities are generally used to prepare the tissues for the manual therapies and therapeutic exercises. Physical modalities should never be the sole therapeutic method applied to any patient.Therapeutic parameters for each modality are chosen based upon the acuity of the injury, so the therapist must be well versed on the definitions of the acute, subacute, and chronic phases of healing. Therapeutic ultrasound works on a frequency of 1MHz (megahertz) or 3MHz via a reverse piezoelectric effect. This is the conversion of electricity to sound waves and takes place within the piezoelectric crystal housed in the transducer (sound head). Therapeutic ultrasound can create thermal and non-thermal effects, with the latter causing tissue modulation instead of heat.Tissue heating caused by therapeutic ultrasound causes increased collagen extensibility and blood flow, decreased muscle spasm, and creates a mild inflammatory effect. Non-thermal effects of therapeutic ultrasound cause enhanced tissue repair via stimulation of fibroblasts, increased protein synthesis, blood flow and glycosaminoglycans synthesis, facilitated inflammation, and improved cartilage healing.Indications for therapeutic ultrasound include muscle spasm, muscle pain secondary to IVDD, tendinopathy, delayed wound healing, and muscle strain injuries. Setting variables for therapeutic ultrasound include duty cycle, frequency, intensity, and treatment duration. Duty cycle refers to the ratio of on time to off time and can be either continuous or pulsed. Continuous treatment causes tissue heating. Pulsed duty cycle can range from 10% to 90%, with increased potential for tissue heating at higher levels. Frequency, measured in megahertz (MHz), determines the depth of penetration of the sound waves. 1MHz penetrates to 5 centimeters and 3MHz penetrates to 1 to 2 centimeters.Intensity is measured in watts per square centimeter and is generally set between 0.1 and 2.0 W/cm^2. Treatment duration is determined by the size of the transducer head and the size of the area to be treated. Transducer heads come in sizes from 1cm^2 to 10cm^2, and the general rule is to treat for 2.5 minutes per treatment area equal to the head diameter. This means that if the patient has a treatment area of 20cm^2 and the therapist has a 5cm^2 treatment head, the treatment will take 10 minutes.

There are precautions that the therapist must take when using a therapeutic ultrasound device. The crystal in the transducer head is very fragile. The head must maintain contact with fluid at all times when the unit is turned on. If the head is allowed to remain in dry air while turned on, the crystal will be damaged. As the potential for tissue heating exists, the transducer head must be kept in motion at all times during the therapy session. Fur can attenuate the sound waves preventing transmission to the deeper tissues and creating heat at the surface. To prevent this, the fur must be clipped or at least soaked in water or gel. Contraindications for use of therapeutic ultrasound include therapy over open physes, over fractures, or over a pregnant uterus.There are few clinical studies looking at the use of therapeutic ultrasound in veterinary practice. One study demonstrated healing of partial gastrocnemius muscle avulsions in dogs using therapeutic ultrasound, with follow up of 6 to 12 months.

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WSV18-0085

SVA DERMATOLOGY (SIMULTANEOUS TRANSLATION INTO MANDARIN CHINESE)

NON-PRURITIC ALOPECIA IN THE DOG: IS IT ALWAYS HORMONAL?

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Introduction

Allergen immunotherapy (AIT) in human patients is a safe and effective treatment for allergic rhinitis, asthma and venom hypersensitivity. It has also been used for atopic dermatitis and food allergy. AIT may also be effective in preventing sensitisation to new allergens and halting the "atopic march".

In dogs, AIT is an effective treatment for canine atopic dermatitis. It is the only current treatment that can modify the allergic disease and potentially improve the allergic clinical signs and reduce the amount of anti-pruritic medications required long term. Recently, food specific sublingual immunotherapy has been shown to be well tolerated and safe in healthy dogs and effective in a small number of dogs with adverse food reactions. AIT has also been used to manage feline atopic dermatitis and feline asthma.

Mechanisms of AIT

Briefly, allergy is characterised by a dominance of T-helper 2 cells and their cytokines resulting in production of allergen-specific IgE. A summary of the immunological changes in allergic disease and with AIT is provided by Mueller **et al** 2018,and shown in Table 1.

The main aims for AIT are to induce tolerance to these allergens and stimulate T regulatory responses. A summary of the mechanisms of AIT in human patients is provided by Jutel **et al**, 2016 and shown in Figure 1. Generally, similar findings are found in dogs.

Table 1: Immunological data about allergy and allergen immunotherapy in humans and dogs (Mueller et al 2018)

Figure 1: Mechanisms of Allergen immunotherapy (Jutel et al, 2016)

Subcutaneous immunotherapy (SCIT)

AIT in humans and dogs has traditionally been administered subcutaneously. For dogs, different protocols using different adjuvants are used but generally they consist of an induction phase with increasing allergen doses followed by a maintenance phase. SCIT should be continued for at least 12mths before efficacy is determined. If effective, SCIT is recommended for at least 3-5 years. Adverse reactions can occur at any stage of SCIT but more commonly during induction phase. They range from localised (e.g. erythema, pruritus and swelling) to serious systemic anaphylaxis.

Sublingual immunotherapy (SLIT)

In this route, allergen extract is administered (usually as a tablet or liquid formulation for human patients) under and around the tongue. The formulation contains ingredients to stabilise allergen and promote mucosal absorption. This route has the additional benefit of having the allergen taken up by dendritic cells (antigen presenting cells) in the oral mucosa and presented to T cells in draining lymph nodes. Neither food nor drink is allowed for at least 5 minutes. The optimal duration of SLIT is not standardised but at least 3 years is recommended.

Systemic adverse reactions to SLIT are much lower compared to SCIT due to lower number of mast cells within the oral and sublingual mucosa. Localised adverse reactions include oropharyngeal pruritus and/or swelling. Most importantly, the risk for systemic adverse reactions is significantly lower in SLIT compared to SCIT. For example, 0.1 to 3.5% of SCIT result in a systemic allergic reaction compared to 0.056% for SLIT.

Studies on SLIT in dogs for allergic disease are few. There are different suppliers providing different formulations, administration protocols, storage conditions with different reported efficacy and potential adverse reactions. SLIT is usually administered using a metered pump dispenser.

A small placebo controlled study of 13 house dust mite sensitive dogs where 7 of the 13 dogs received daily doses of house dust mite culture mixed with cream cheese applied to inside of cheeks and hard palate everyday for 7 months did not show any clinical improvement nor changes in allergen specific IgE levels. Another small study in 18 atopic dogs sensitised to dust mites, timothy grass and ragweed that received one year of SLIT showed a slight improvement in clinical signs but significant increases in TGF-beta and IL 10. Another small study of 10 dust mite sensitive dogs showed clinical improvement with supportive serological changes (decreased mite specific IgE and increased IgG levels) after 6 months of SLIT. Another multicentre open trial where dogs received twice daily SLIT for at least 6mths, reported 55% (66/124) of dogs had a good to excellent

response. Interestingly, 49% (23/47) of dogs that had a good to excellent response to SLIT had previously failed SCIT.

SCIT vs SLIT in dogs (Table 2)

In dogs, until more studies are performed investigating the ideal dose, protocol and efficacy, SLIT is best suited for dogs where either owner or dog is needle-phobic, if the dog developed serious adverse reactions to SCIT or has failed SCIT.

Table 2: Pros and cons of SCIT and SLIT in dogs

	SCIT	SLIT
Pros	Extensive experience and established success rate~52-77%	No injections required-easily given at home
	Low rate of adverse reactions	Lower rate of serious adverse reactions with no anaphylaxis reported
	Safe long term therapy	May be effective in patients that failed SCIT (49%)
		Allows mixing of mold extracts in same vial
Cons	Rare but possible anaphylactic reactions	Limited experience
	Injections required	Unclear success rates~40 to 60%
		Patient adherence-daily to twice daily administration
		Usually more expensive vs SCIT
		Food and water needs to be withheld before and after SLIT
		May not be palatable-salty in humans

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PECULIARITIES OF FELINE ANEMIAS: BLOOD LOSS, HEMOLYSIS, MARROW FAILURE

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Anemia is an extremely common clinical problem in cats and is associated with many different conditions. Despite severe anemia many cats may only show mild clinical signs particularly when chronic. In order to recognize the type, degree, and regeneration of anemia in cats, it is important to appropriately appreciate the hematological peculiarities of cats. When compared to dogs, the normal packed cell volume in cats is lower (Packed Cell Volume [PCV] 0.30-0.48/L; 30-48%), feline red blood cells are considerably smaller (MCV 38-50 fl), central red cell pallor is small (cannot see spherocytes), bone marrow iron stores are visiually lacking, and there are mostly mild regenerative responses observed. There are aggregate reticulocytes (also reflected as polychromasia) which are short-lived in circulation (like in dogs), but cats also have punctate reticulocytes, which may linger around in circulation for a couple of weeks. The best parameter to assess a regenerative bone marrow response is the absolute reticulocyte count (normally <60,000/µl), which refers to the presence of aggregate reticulocytes and is equal to the degree of polychromasia. Note nucleated red blood cells may be proportionally increased with reticulocytes or may occur independently due to bone marrow endothelial damage as in lead poisoning, sepsis and myelodysplasia. While the evaluation of PCV, total protein, and blood smear are most valuable, a complete blood cell count and specific tests are generally required to reach classify the anemia, for a definitive diagnosis and to monitor the response to therapy. A complete blood cell count with reticulocyte count of modern hematology analyzers is ideal to best assess hematological disorders, but other tests are likely needed. These may include for instance chemistry screen, urinalysis, bone marrow cytology or core biopsy, hemostatic tests, iron parameters, infectious disease screen by serology and PCR, toxicological analysis, Coombs', and genetic/DNA tests.

Although kidney failure and some infections (flea infestation, FeLV infection and hemobartonellosis) are likely the most common causes of anemia, there are many other differential diagnoses to consider, such as bleeding disorders, toxicity, metabolic disturbances, hereditary defects, and immune-mediated hemolytic anemias. It is therefore crucial to carefully assess the feline patient by history taking, physical exam and routine laboratory tests in order to determine the cause and offer the most appropriate treatment.

In contrast to dogs, **blood loss anemias** are less commonly observed in cats, albeit they happen with trauma and surgery. In fact, many cats drop their PCV during and shortly after surgery which may in part be blood loss, but also unexplained lysis and sequestration. Moreover, external blood loss can rapidly result in iron deficiency particularly in the very young kitten (even with repeat phlebotomies for diagnostic purposes), however, the classic microcytosis and hypochromasia in iron deficiency may be very difficult to appreciate due to small feline red cells. The most common reason for blood loss is flea infestation, while maggots, ticks, and hookworms are less likely leading to major blood loss. Skin and other tumors may also cause local bleeding. Blood loss anemias are generally regenerative after 3-4 days and remain regenerative even when iron deficient. While in the above cases hemorrhage was caused by vascular injury, there are also a variety of bleeding disorders to consider.

Thrombocytopenia is rare in cats but may be induced by drugs (methimazol) and rarely infection and cancer; immune-mediated thrombocytopenia also seems to occur rarely. Accurate platelet counts can be difficult to obtain due to their large size platelets and tendency to aggregate. Thus, any platelet count needs to be confirmed with an estimate from a blood smear examination (20,000 platelets/µl equals 1 platelet seen on a high power microscopic field). Thrombopathia – impaired platelet function - may be triggered by aspirin or similar drugs (cats appear particularly sensitive to platelet injury but less likely to aspirin or steroidal ulceration). Hereditary thrombopathias are extremely rare. Compared to dogs, anticoagulant rodenticide poisoning is less commonly observed in cats. However, coagulopathies due to hepatic failure are much more severe in cats than dogs; hence diagnostic liver biopsies are frequently associated with serious hemorrhage. Furthermore, there are several hereditary coagulopathies such as hemophilia A and B in domestic and Himalayan cats as well as a vitamin K-dependent coagulopathy in Devon Rex and Sphinx cats. Interestingly, domestic and exotic shorthair cats often have a coagulation factor XII deficiency; while this causes a markedly prolonged partial thromboplastin time, this is not associated with a bleeding tendency. Generally, the prothrombin and partial thromboplastin times provide sufficient information to differentiate the coagulopathies, although specific factor analyses may be needed.

Hemolytic anemias in cats are often hard to recognize as the degree of bone marrow regeneration and the evidence of bilirbinuria and hyperbilirubinemia are often mild (any bilirubinuria is important in a cat). In fact, icterus in cats is much more likely due to hepatic failure. While the normal feline spleen is very small, it can get fairly enlarged in cases of hemolytic anemia. Furthermore there is a syndrome of increased erythrocytic osmotic fragility seen in Abyssinian, Somali and other domestic house and purebred cats with massive splenomegaly. Pyruvate kinase deficiency is a common hereditary disease causing intermittent hemolytic anemia in Abbyssinnian, Somali and other purebred and even domestic house cats. There is also porphyria, a heme synthesis defect, which causes hemolysis but most remarkably erythrodontia with fluorescing teeth. Porphyria may dominantly or recessively inherited and cats may live for years with this condition requiring no specific treatment.

In contrast to dogs, primary (auto-) immune-mediated hemolytic anemia seems rare in cats, but may be seen with other triggers such as infections, drugs, and cancer (secondary IMHA). Their species-specific Coombs' test is positive. In addition, some show autoagglutination that may break up when adding saline (Rouleaux) and after washing with saline when caused by unspecific agglutination such as by EDTA. More important than primary IMHA is alloantibody associated hemolysis. Neonatal type A and AB kittens nursing from a type B queen will frequently develop acute hemolysis of the newborn during the first hours to days of life. Classic signs are acute death, massive pigmenturia due to hemoglobinuria and occasionally they may develop icterus and a tail tip necrosis and survive. Similarly important are A-B mismatched (first transfusion due to preformed antibodies and rarely others like Mik and only after prior sensitizing) acute hemolytic transfusion reactions. Thus AB typing is critical prior to breeding and transfusing cats. Transfusing canine blood to cats (xenotransfusion) causes always severe hemolytic reactions and is not recommended.

Feline hemolytic anemias
- **Infections**
- Mycoplasma hemofelis, (also hemominutum, turicesis)
- Cytauxzoon felis
- Feline Leukemia Virus infection (A type)
- Feline Infectious Peritonitis
- **Immune**
- Alloimmune – neonatal isoerythrolysis and acute transfusion reactions
- Autoimmune or primary hemolytic anemia (rare compared to dogs)
- Secondary (drugs [methimazol], infection, cancer)
- **Toxic**
- Drugs - acetaminophen, lidocaine spray, propofol, etc.
- Onions
- **Metabolic**
- -Hypophosphatemia (D. mellitus, hepatopathy, hyperalimentation)

- **Hereditary**
- Pyruvate kinase deficiency (Abyssinian, Somali, other purebred and DSH cats)
- Increased osmotic fragility (Abyssinian, Somali, other purebred and DSH cats)
- Porphyria (Siamese and DSH cats)

There are several important differential diagnoses for hemolytic anemias and thus treatment options depend on the cause of hemolysis. Various triggers such as drugs and chemicals can be rapidly removed. However, other diagnoses may require tests at reference laboratories, such as for serology and real-time PCR for infectious diseases and genetic tests for hereditary diseases. It is therefore not unusual to start with a combination of prednisolone (fortunately even at high doses well tolerated) and doxycycline as initial treatment of hemolytic anemias to cover the bases until test results are back and specific and proper therapy can be instituted. For hereditary hemolytic anemias it is most important to avoid harmful treatments and offer a safe (indoor) environment.

Lastly, **non-regenerative anemias** due to decreased erythro- or overall hematopoiesis can be associated with a variety of disorders. Indeed, mild non-regenerative normochromic normocytic anemia is commonly seen with many organ diseases and is mostly well tolerated. However, many middle-aged to older cats with chronic renal failure develop a moderate to severe anemia. The main cause is a lack of renal production of erythroproietin, but uremic toxins affecting red cell stability and bone marrow production as well as blood loss from ulcers also play a role. Transfusion or human recombinant erythropoietin (darbepoetin) can reverse the anemia and associated clinical signs. However, repeat transfusions are generally needed and cats may become refractory, as they develop alloantibodies against the transfused red cells. Moreover, cats can develop antibodies against the recombinant human erythropoietin, which leads to a severe and hardly reversible pure red cell aplasia. Renal transplantation from a carefully selected donor cat has effectively reversed not only the anemia but also restored kidney function. While rarely truly deficient, iron, folate, and cobalamin may be replenished as needed. FeLV infections may end in a pure red cell aplasia (C type) or myelodysplasia to aplasia, while FIV exhibits less effects on the bone marrow. Cancer associated anemias may have many causes but may result in aplastic or myelophistic bone marrows.

Finally, many anemic cats need transfusion support during the initial management. There is no specific trigger PCV, but rather the overall clinical picture with a PCV of <20% is used. It should be noted that cats need to be AB blood typed before the first and crossmatched before the second transfusion (>4 days from first transfusion).

TIPS FROM THE EXPERTS FOR THE MANAGEMENT OF…

DIABETES MELLITUS IN DOGS

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TIPS FROM THE EXPERTS FOR THE APPROACH AND MANAGEMENT OF DIABETES MELLITUS IN DOGS

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Diabetes mellitus is a common endocrinopathy in dogs in cats. Dogs usually get type 1 or the insulin-dependent form. This occurs from loss of pancreatic beta cells such that adequate amounts of insulin are not produced and secreted. Cats usually get type 2 or the non-insulin dependent form of diabetes mellitus. This occurs as a result of insulin-resistance, often from obesity. The pancreatic beta cells are forced to produce increasing amounts of insulin to over come the resistance, resulting in ultimate loss of function in the cells. Additional loss results from direct glucose toxicity of the beta cells. Cats can go into diabetic remission a syndrome in which insulin requirements diminish or cease to be necessary after initial treatment. This is probably due to correction of a condition causing insulin resistance.

DIAGNOSIS.

Diabetes mellitus is relatively easy to diagnose. Clinical signs include polyuria/polydipsia, weight loss, persistent or recurrent urinary tract infections, weakness and muscle wasting, cataracts (usually dogs), and peripheral neuropathies (usually cats). Diagnosis can be made by recognition of appropriate clinical signs, and demonstration of persistent hyperglycemia and glucosuria. One confounding factor to this diagnosis is stress. Stress, alone, can cause hyperglycemia, that can be high enough to be cause spill over into the urine and glucosuria. Should the clinician have any doubt of whether hyperglycemia and glucosuria are due to diabetes mellitus, s/he should check a serum fructosamine level. This value gives the average of the blood glucose over the preceding 2-3 weeks. If elevated, then diabetes mellitus can be diagnosed. If not elevated, then the hyperglycemia/glucosuria was probably due to stress.

Initial evaluation will decide how intensively the patient should be managed as diabetic treatment is begun. If the animal is eating and drinking normally and is well hydrated, there is no reason to hospitalize him/her while insulin therapy is initiated. If the animal is ketotic, acidotic, hyperosmolar, or dehydrated, s/he should be admitted

to the hospital and stabilized before long term insulin therapy is instituted. The most common concurrent diseases seen with diabetes mellitus include: urinary tract infections, concurrent endocrinopathies such as Cushing's disease, hypothyroidism, and hyperthyroidism, pancreatitis, infections, and pregnancy.Initial evaluation of the diabetic animal should include a complete physical examination, CBC, chemistry profile, urinalysis, and T4 (cats). A urine culture should be considered even if urinalysis and sediment parameters are normal since up to 35% of urinary tract infections can be clinically silent in animals with dilute urine. Abdominal imaging may be pursued if clinically indicated. Concurrent medical conditions should be addressed aggressively so that the diabetes mellitus can be more easily controlled.

TREATMENT.

Diabetes mellitus can be frustrating, expensive, and time-consuming for owners to treat. Our goals of therapy should be correction of clinical signs, control of concurrent diseases, and avoidance of emergency situations such as hypoglycemia, ketosis, and hyperosmolality. It is beneficial to have an in depth discussion with the owner as to the time and effort s/he can realistically commit for diabetic control for the pet. It is important to establish a good rapport with diabetic owners since they will be asked to provide invasive (injections) and time-consuming (glucose monitoring) care for their pets. Stable cats with blood glucoses less than 400 mg/dl may be treated initially with a diet change. Cats are very carbohydrate intolerant, and a low carbohydrate diet can result in euglycemia in some cats. Prescription diets are recommended; however, if the owners can't or won't commit to these diets, commercial diets can be used. Canned diets tend to be lower in carbohydrates and some websites include carbohydrate data on specific diets. Because of the deleterious effects of a hyperglycemic environment on the cat pancreas, diet change alone should not be tried for more than 2 weeks. If the cat has not become euglycemic over that time period, then insulin therapy should be instituted. Insulins recommended for cats include: ProZinc ^{tm,} glargine (Lantus ^{tm}), and Vetsulin ^{tm}. ProZinc insulin has an advantage in that it is labeled for veterinary use. Determir tm may also be used, although there is less experience with this insulin in cats. NPH insulin may be used but it does not have the duration of action in cats to allow bid treatment. Optimally NPH needs to be given tid-qid in cats. The starting dose of insulin (regardless of type) in cats is: 0.25 U/kg bid. The optimal diet for diabetic dogs is one high in insoluble fiber. This diet slows glucose absorption from the gut and postprandial hyperglycemia. Unlike cats, dogs will not usually revert to euglycemia as a result of a food change. Newly diagnosed diabetic dogs should be started on insulin therapy. Appropriate insulins for use in the dog are: Vetsulin ^{tm}, NPH, Detemir ^{tm}, ProZinc ^{tm}, and glargine (Lantus ^{tm}). Dogs are initially

dosed at 0.5 U/kg bid except for Determir that should be started at 0.25 U/kg.

Dogs and cats should be fed twice a day when insulin is given. Many owners give insulin while the animal is eating. This makes the insulin injection a pleasant experience for the pets and easier for owners to treat the animal. Some cats prefer to nibble food throughout the day. These grazers can often be well managed by allowing them free choice eating with insulin injections twice per day. Care should be taken to ensure that the cats are not receiving more than their caloric needs since extra weight should be avoided.

Exercise is beneficial to diabetics and serves to lower insulin requirements and provide better glycemic control. Daily walking for dogs and cat play can be effective ancillary treatments for diabetes mellitus. Average time for initial diabetic control is 4-6 weeks.

MONITORING.

After insulin is started, the veterinarian should wait 7-14 days to monitor any effects since it takes this long for the animal to adjust to insulin therapy. During that time period owners may measure urine glucose and ketones. They should call if there are more than 2 negative urine glucose readings or if the ketones are positive. If urine glucose is negative, one doesn't know if the blood glucose is 40 or 200 mg/dl. Alternatively, the owner can use a portable glucometer and measure blood glucose directly. Insulin doses should not be changes as a result of readings, but owners should call if the animal is ketotic or hypoglycemic.

At the initial recheck, the veterinarian should question the owner about resolution of clinical signs. A physical exam and weight measurement should be completed. In this way the veterinarian can evaluate the clinical response to insulin.Serum fructosamine levels have been advocated for use in monitoring insulin response. For fructosamine to be interpretable, the pet should have been on a stable insulin dose for at least 3 weeks before the fructosamine is taken. Fructosamine levels can be useful; however, it is inappropriate to use in unstable animals or those in which a hypoglycemic-hyperglycemic (Somogyi) response is suspected. For these patients, a glucose curve must be completed.

GLUCOSE CURVES.

A glucose curve is the only way to truly evaluate the body's response to insulin. Important information obtained from glucose curves includes the onset of action of the insulin, the duration of action of the insulin, the time of peak activity of the insulin, and how low the glucose goes (nadir). The first three parameters indicate whether the right type of insulin is being used; the last parameter gives information about the dose of insulin used.

The traditional glucose curve has many limitations, including disruption of the patient's normal activity and eating routine, the introduction of stress-related hyperglycemia, and labor intensiveness of the procedure. Furthermore, both diabetic dogs and cats have been shown to have significant variations in their day-to-day glycemic control. Intermittent blood sampling over only a 12-hour period may grossly over or underestimate a patient's glycemic control, and glucose peaks and nadirs may be missed if they occur between samplings. Continuous glucose monitoring systems (CGMS) provide a minimally invasive method for continuously evaluating glycemic control for up to 72 hours. Interstitial glucose has been shown to correlate well with plasma glucose levels. They are comprised of an external sensor with a flexible electrode that reacts with glucose when it is inserted into the subcutaneous tissue. This sensor then communicates to a small monitor that records the glucose data. MiniMed iPro2 and Abbott Freestyle Libre systems have been used successfully in veterinary medicine and can be sent home with animals.

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OPHTHALMOLOGY

UVEITIS – IT'S JUST INTRAOCULAR LYMPHADENOPATHY

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Introductory Philosophy

The uvea contains familiar tissues and cell types (lymphocytes, smooth muscle, and blood vessels, for example), is inflamed by familiar antigens (infectious agents, neoplasia, auto-antigens) and reacts with the 5 cardinal signs of inflammation seen elsewhere (heat, pain, swelling, etc.). This review aims to aid diagnosis and therapy of uveitis by likening it to inflammation elsewhere (because it is more similar than it is different) while highlighting differences (because these are helpful).

Clinical Signs

Active (acute) uveitis

Uveitis has few pathognomonic signs and these are notably more subtle in cats than they are in dogs. Therefore, uveitis in cats often goes undetected by owners and untreated by veterinarians until potentially blinding sequelae such as glaucoma, cataracts, and retinal detachment or degeneration occur. Therefore, clinicians must maintain a high index of suspicion regarding uveitis in all cats with ocular disease and even those with nonspecific signs such as lethargy, "hiding", anorexia, or fever.

Uveitis, like inflammation elsewhere, is evident as one or a combination of the 5 cardinal signs of inflammation: heat, pain, swelling, redness, and loss of function. One just has to think about how these are best seen:

- **Intraocular pain**: blepharospasm or epiphora; however cats seem more likely to show subtle and less localizing signs such as lethargy or anorexia
- **Iridal swelling** requires that the eye is examined using a source of magnification (such as the Optivisor®) in association with a bright and focal light source (such as the Finoff transilluminator®) directed very obliquely across the globe. Look for a loss of the normal "texture"
- **Redness** evident as scleral injection is typically evident in dogs but can be particularly subtle in many cats. The tendency to diagnose any redness of this region as conjunctivitis must be avoided. Redness of the iris usually indicates neovascularization and not congestion and is discussed below under chronic changes.
- **Dysfunction**: Given the diverse range of critical functions of the uvea, loss of function produces an important series of clinical signs evident as breakdown of the blood aqueous barrier (BAB), miosis, corneal edema, and hypotony. Of these BAB breakdown is pathognomonic and so the anterior chamber is worthy of special attention since the aqueous humor

is equivalent to the interstitial space. In particular look for **hypopyon** (white blood cells), **hyphema** (red blood cells), and **fibrin**, **aqueous flare** (albumin and other small proteins) and **keratic precipitates** (white blood cells and inflammatory proteins clumped against the corneal endothelial surface).

Chronic uveitis and its sequelae are associated with scarring (fusion of one tissue to another and cicatrization), chronic dysfunction, and neovascularization evident as **glaucoma** (due to scarring or clogging of the iridocorneal angle), **anterior or posterior synechia** (adhesions between the iris and corneal endothelium or lens, respectively), **phthisis** (globe contracture) or **retinal detachment** (due to contraction of vitreous fibrin). Altered aqueous humor composition and circulation also causes a relative malnutrition of the lens (evident as **cataract**) and inner cornea (evident as corneal edema, vascularization, fibrosis etc.). **Lens luxation** may occur due to enzymatic lysis or phagocytosis of the lens zonules, secondary to cataract development, or as a sequela to buphthalmos due to secondary glaucoma. Neovascularization of the face of the iris (**rubeosis iridis**) is a pathognomonic sign of subacute or chronic active uveitis. It is one of the signs that is seen more easily in cats than in dogs due to the typically lighter iris color of cats.

Diagnosis

Box 1 highlights the important tests to diagnose whether uveitis is present. Confirming or eliminating all suspected **etiological diagnoses** is the essential next step. By conducting an excellent general physical and ophthalmic exam as well as gathering a focused history, the initial goal is to establish whether further diagnostic testing is strongly supported. I do this by categorizing the uveitis as present in a well or systemically ill patient, unilateral or bilateral; exogenous or endogenous; acute or chronic; and as involving the anterior uvea, choroid, or both.

Box 1. Essential diagnostic tests to detect evidence of uveitis (Tests should be conducted in this order) IOP = intraocular pressure; KPs = Keratic precipitates; AC = Anterior chamber; BAB = Blood-ocular barrier.

Ophthalmic test	Look for:
Retroillumination	• Anisocoria (especially due to miosis) • Dyscoria • Corectopia • Iridal atrophy • Opacities in the clear ocular media (KPs, hypopyon, hyphema, vitreous debris, posterior synechia, secondary cataract)
Oblique illumination of eye	• Corneal edema • Iridal swelling/nodules • Iridal thinning/atrophy • Rubeosis iridis • Posterior synechia • Iris bombé • Hypopyon • Hyphema • AC fibrin

Tonometry (IOP assessment)	• Decreased or low-normal in uncomplicated uveitis • Elevated or high-normal when complicated by glaucoma
Pupil dilation	• Resistance to dilation • Snow banking
Assessment of aqueous flare	• Serum proteins in the AC due to BAB breakdown
Fundic examination	• Signs of posterior uveitis (Retinal detachment or degeneration, chorioretinal granulomas, hemorrhage, edema) • Vitreous debris/infiltrates • Snow banking (cats)
Application of fluorescein stain	• Corneal ulceration suggests exogenous (axonal) uveitis • Corneal ulcers preclude use of topical corticosteroids

An etiologic diagnosis should then be pursued through a diagnostic workup identical to that employed for a cat with lymphadenopathy. Consider CBC, Biochemistry, urinalysis, serology, chest and abdominal imaging, etc. as appropriate for the following agents.

Etiology

The known causes of endogenous anterior uveitis in cats are expanding but still too few to explain the majority (~ 70%) of cases.

Infectious agents as a cause of uveitis

Viral	Bacterial	Fungal/Algal
• FIP • FeLV* • FIV • FHV • CDV • CAV	• *Bartonella* spp. • *Mycobacterium* spp. • *Ehrlichia* spp.¥ • *Borrelia burgdorferi*¥ • *Brucella* • *Leptospira*	• *Cryptococcus neoformans*† • *Histoplasma capsulatum*† • *Blastomyces dermatitidis*† • Candida albicans • Coccidioides immitis† • Aspergillus spp.
Parasitic	Protozoal	
Cuterebra	• *Toxoplasma gondii*	
Larval migrans	• *Leishmania* spp.	

*Via immunosuppression or oncogenesis

† Chorioretinitis predominates

¥ Seroprevalence data only; no clinical evidence

Neoplasia as a cause of uveitis

The most common primary intraocular neoplasm is melanoma; however this typically causes little or no uveitis. By sharp contrast, the most common metastatic ocular neoplasm – lymphoma – tends to be associated with marked breakdown of the BAB with hypopyon formation, fibrin exudation into the anterior chamber, and hyphema. One of the curious observations with this disease is that the degree of apparent pain often seems less than might be expected from the severity of other signs of intraocular inflammation. The exception to this is when secondary glaucoma occurs, which can be quite frequent due to the highly cellular and fibrinous nature of the anterior chamber exudate

Treatment

Treatment of anterior uveitis must be tailored to the individual case based on proven or suspected cause, severity, anatomical location, chronicity, and presence of systemic or other intraocular disease. Regardless, some general therapeutic guidelines are possible.

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Optimal treatment relies upon identification and removal or reduction of the causative antigen; however this is rarely possible. Additionally, all patients with uveitis need their intraocular inflammation controlled rapidly and completely, since it is painful and produces vision-threatening sequelae. Thus, immunomodulating drugs form the mainstay of therapy for uveitis. The major decisions are therefore which immunomodulating drugs should be given, via what route and, at what dose.

Immunomodulatory therapy

Corticosteroids are commonly used for uveitis. Their systemic use should be reserved until a definitive cause responsive to corticosteroids has been found or, failing this, until causes known to be worsened by glucocorticoids have been adequately eliminated. In particular, the systemic mycoses must be adequately eliminated as potential causes. Likewise, patients in which lymphoma is possible and which would benefit from a multidrug chemotherapeutic regimen should not be treated with systemic corticosteroids alone. By contrast, topical administration of corticosteroids may be employed safely even when an infectious or neoplastic cause might prevent systemic administration of the same drugs. Prednisolone acetate (1% or 0.125%) and dexamethasone (0.1%) will penetrate intact corneal epithelium and reach the anterior uveal tract. Hydrocortisone (as found in many combined antibiotic–corticosteroid ophthalmic preparations) does not penetrate intraocularly and should not be used. The frequency of application should be tailored to the severity of the uveitis; starting as frequently as q 2 hours and tapering as a clinical response is noted. When safe, corticosteroids should be administered systemically for posterior uveitis and when more significant immunomodulation is necessary, or when corneal ulceration prohibits their topical use. Typical doses of prednisolone range from 1 mg/kg q 12 hours when notable inflammation is present to 0.5 mg/kg once daily when a more moderate anti-inflammatory effect is desired. As with topical corticosteroids, dose and dose frequency of systemically administered glucocorticoids should be carefully reduced based entirely upon clinical evidence of waning disease.

Compared with corticosteroids, **non-steroidal anti-inflammatory drugs (NSAIDs)** are not immunosuppressive, and may be more expensive, sold in smaller volumes (as topical ophthalmic solutions), and sometimes unavailable in ointment form. These limitations must be borne in mind for dogs and cats with uveitis; however they may be preferred over corticosteroids in patients with diabetes or other endocrinopathies in which corticosteroid use may not be wise. They can also be administered systemically instead of corticosteroids when systemic infectious disease is suspected or proven, or until lymphosarcoma is eliminated as a differential consideration. And they may be given in conjunction with a topical steroid. As such, they may make an excellent choice for initial control of inflammation while likely causes are being ruled in or out. The same general comments regarding dose frequency and route made for corticosteroids apply equally to NSAIDs.

Iridocycloplegic agents

Atropine has multiple favorable actions in eyes with uveitis and form a critical component of treatment. It paralyzes the iris sphincter and ciliary body muscles causing mydriasis and cycloplegia, respectively. Pupil dilation reduces leakage of vascular elements into the aqueous humor by causing radial blood vessels within the iris stroma to "concertina" upon themselves (thus providing a physiological tamponade); decreasing iris surface area (from which inflammatory mediators and vascular components originate); reducing uveal vascular endothelial permeability; and by reducing chances and consequences of posterior synechiation. However "bunching" of the iris in the periphery does increase the chance of anterior synechia and potentially obstruction of the iridocorneal angle. Cycloplegia reduces ocular pain but also increases resistance to aqueous outflow. Therefore, pupil dilation and cycloplegia are desirable in all cases of uveitis except those where secondary glaucoma is present or likely. This is more likely in some dg breeds predisposed to primary glaucoma but can also occur in cats and non-predisposed dogs The effect of mydriasis upon IOP can be tested by a single application of the short acting drug tropicamide followed by tonometry when the pupil is fully dilated. If IOP is increased by tropicamide, atropine should not be administered. If atropine is initiated, IOP should be rechecked regularly and application discontinued if IOP increases above normal. In cats, atropine should be applied as an ophthalmic ointment rather than a solution because it is bitter and passage down the nasolacrimal duct can cause violent salivation and frothing that is harmless but disturbing to the cat and its owner.

Monitoring and Sequelae

Prompt specific treatment of uveitis with tapering of therapy based upon reduction of clinical signs may result in some sequelae but these are usually mild and should be static. If they are not, this suggests chronic or recurrent uveitis and further investigations and treatment are necessary. Classic sequelae include corneal fibrosis, cataract, or posterior synechia. None of these changes should result in pain or, unless severe, vision disturbance. By contrast, more severe, unrecognized, persistent, or recurrent uveitis frequently results in a blind and sometimes painful globe. The most common sequelae (and their prevalence in cats) include cataracts in 20-36% of eyes, lens luxation in 11-18%, glaucoma in 16-46% and enucleation in 29%. Many patients experience more than

one of these sequelae. For these reasons, frequent and careful monitoring of a patient with uveitis is essential. This should be performed as for patients with immune-mediated disease elsewhere with gradual tapering of medications and re-examination at doubling intervals presuming there is improvement; more often if there is not. Re-examination and tapering of medications should be continued until there is **complete** resolution of **every** clinical sign of active uveitis. I believe that tonometry is the most sensitive test with which to monitor uveitis during treatment because subtle hypotony (sometimes only relative to the contralateral eye) can continue long after other more overt signs have normalized. Continued treatment of these patients may prevent or delay development of sight-threatening ocular complications.

WSV18-0049

SVA REHABILITATION
LASER: CLASS 3 VS CLASS 4 - WHERE DO I BEGIN

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LASER: CLASS 3 VS CLASS 4 - WHERE DO I BEGIN?

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Laser therapy, also called photobiomodulation, uses electromagnetic energy to stimulate tissues in the body. Laser is an acronym for **L**ight **A**mplification by **S**timulated **E**mission of **R**adiation, and by definition is collimated and monochromatic. The depth of penetration of laser energy is determined by the wavelength of the light energy.Lasers a classified as Class 1, Class 2, Class 3a, Class 3b, and Class 4. These classes are determined by the milliwatts (mW) of power and were created to describe levels of danger associated with the use of each. Class 1 lasers have less than 0.5mW of power. A typical example would be a garage door opener or television remote control. Class 2 lasers are between 0.5 and 1.0mW. Most laser pointers are in this class. Class 3 lasers have between 1 and 500mW. This group is referred to as therapeutic 'cold' lasers and come with 'eye caution to eye danger'. Class 4 lasers have more than 500mW and are referred to as 'hot' lasers. Surgical and industrial lasers are in this class, which has the precautions of 'fire hazard and fire danger'.Terminology related to laser usage can be quite confusing. "Low level laser therapy", "LLLT", "Cold Laser", "Class 4 laser therapy", and "High intensity laser therapy" are often used incorrectly and are generally misleading. It is safer to use the terms **photobiomodulation** or **therapeutic laser** or **laser therapy.**The physiological effects of laser include accelerated cell division via mitochondrial stimulation, increased leukocyte phagocytosis, stimulation of fibroblasts and collagen stimulation, enhanced synthesis of ATP, and angiogenesis. These effects lead to vasodilation, decreased inflammation, decreased edema, slowed nerve conduction and inhibited peripheral nociception.Indications for laser therapy include wound healing, pain relief (both acute and chronic), muscle spasms (including, perhaps, delayed onset muscle soreness or 'DOMS'), and edema reduction. Laser is used in pain management practice to speed wound healing and control inflammation. Contraindications include use near the eye, over neoplasia, over open growth plates, pregnant uterus or

the thyroid.Laser application requires attention to probe positioning. The probe must be held perpendicular to the target tissue. The use of contact vs. non-contact techniques is determined by the power of the laser. High powered lasers require a non-contact technique due to the potential for burning tissues. Patients with dark skin will experience discomfort from skin heating so power must be turned down, if possible, or treatment times per area must be shortened. Heat generating lasers can cause pain if staples are in the field of treatment. Use of lasers to treat intraarticular areas of joints requires that the joint be in the open packed position.Laser dosing is measured in units of power being transmitted over time. Energy is measured in Joules. One Joule is the equivalent of one watt delivered for one second. To deliver one Joule of energy from a 500mW (Class 3) laser, the treatment must be continued for 2 seconds. Most therapeutic regimens used in veterinary practice and clinical trials call for 1 to 8 Joules per square centimeter. The equates to 2 to 16 seconds of therapy time per square centimeter of treatment area. Laser takes considerably less time to use than therapeutic ultrasound where treatment times are often 5 to 20 minutes.The key to dosing is remembering that the depth of penetration is determined by the wavelength of the laser being used. The power does not alter depth of penetration. It affects only the time required to deliver the desired dose.Safety measures for use of lasers are primarily aimed at eye protection. Goggles are laser-specific and have varying optical densities. Sunglasses do not block laser light. In fact, as they cause pupil dilation, sunglasses increase the risk of laser damage to the retina. Patients should wear 'doggles' as well. Laser therapy should be done in areas with minimal reflective services to minimize accidental eye injury. Keeping the laser probe in contact with the patient can lower the chances for accidental laser exposure, however some lasers have the potential to generate heat and could burn tissues if kept in contact with the tissues. Any practice using laser therapy should have a laser safety officer (LSO). The LSO needs to be aware of the Nominal Hazard Zone which is the space in which the level of direct or indirect exposure to laser light could be hazardous. This is 20 feet (6.5 meters) for Class 4 lasers. A "Laser in Use" warning sign must be placed on the door to the treatment area, and ideally, there will be an automatic locking mechanism on the treatment door that prevents accidental entry to the room whenever the laser is in use.Many studies have been published related to laser therapy. One such study looked at wound healing in a rat skin model. This study showed that laser therapy at 4 Joules/cm^2 every other day caused decreased inflammation and increased collagen deposition and gave the best results in increasing the burst strength of skin incisions. Another mouse study showed that paw edema could be prevented and treated using laser therapy. A canine clinical study showed wound healing

in 3 weeks in what had been a very delayed (7 months) wound closure in a canine limb. Laser therapy reduced the time to ambulation in dogs after hemilaminectomy in one preliminary study. Human studies show significant improvement in pain and flexion measurements in knee osteoarthritis patients. Meta-analyses regarding laser use in tissue healing concluded that "phototherapy is highly effective for tissue repair" and that there is 'strong support from experimental animal studies'. Meta-analyses regarding laser use for pain relief concluded that "phototherapy relieves pain of various etiologies".

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ADRENAL GLAND DISEASE - BEYOND HYPERADRENOCORTICISM

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Adrenal gland disease – beyond hyperadrenocorticism

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Hyperadrenocorticism is a relatively common endocrine disorder in dogs. It is typically associated with inappropriate secretion of adrenocorticotropic hormone (ACTH) by the pituitary gland (pituitary dependent hyperadrenocorticism (PDH)) and less commonly with a primary adrenal disorder (adrenal dependent hyperadrenocorticism (ADH)). By now it has been well described both pathophysiologically and clinically. There are numerous tests that are recommended for diagnosis and differentiation of the site of the lesion and the limitations of test performance in both these areas is well recognised. More recently there has been increasing interest in atypical forms of hyperadrenocorticism and other forms of adrenal hyperfunction. Additionally, with the increasing use of abdominal ultrasound and CT, adrenal masses are often identified without clinical suspicion of hormone dysfunction.Atypical hyperadrenocorticism was a term first used to describe dogs with clinical and clinicopathological signs of hyperadrenocorticism in which both the traditional ACTH stimulation and low dose dexamethasone suppression tests were negative for the disease[1]. Others have suggested that the term atypical be replaced by occult. However, this term may be less appropriate as, at least in humans, it is reserved for patients without obvious or typical signs of hyperadrenocorticism[2]. Whatever term is used it has been suggested that in such cases there is diversion of the normal adrenocortical pathways for cortisol and aldosterone synthesis into overproduction of cortisol precursors and sex hormones. In support of this, many studies have demonstrated that dogs with both typical and atypical hyperadrenocorticism have elevated concentrations of 17 hydroxyprogesterone, progesterone, corticosterone, androstenedione, oestradiol, testosterone, 11-deoxycortisol and 21-deoxycortisol following ACTH administration. This is not surprising as ACTH stimulates all cortisol precursors in a linear manner and does not imply a role of these hormones in the development of clinical signs. Indeed, elevated concentrations of these hormones are also found in dogs with non-adrenal illness. Generally their increase parallels that seen with cortisol. Their measurement may increase the sensitivity of the ACTH stimulation test for diagnosing hyperadrenocorticism but this is associated with a reduction in specificity. Caution is therefore advised in assessing such hormones for diagnosing hyperadrenocorticism. It is notable that atypical hyperadrenocorticism is rare and only a small number of dogs with hyperadrenocorticism have been reported with both normal ACTH stimulation and low dose dexamethasone suppression test results. If clinical signs are mild, retesting if they progress is recommended. Alternatively other diagnostic tests such as advanced imaging and the urine cortisol:creatinine ratio (UCCR) should be considered.

There are other forms of hyperadrenocorticism that better fit the term atypical disease and include combined PDH and ADH, bilateral adrenal tumours, non-cortisol producing adrenal tumours, food induced hyperadrenocorticism and ectopic ACTH production. Functional pituitary and adrenal tumours have been diagnosed in a small number of dogs[3]. Whilst clinical signs of hyperadrenocorticism are typical, the results of tests used to distinguish between PDH and ADH may be confounding. Bilateral adrenal tumours, whilst described in dogs are rare.

It is well known that dogs with classical clinical signs of hyperadrenocorticism caused by adrenal tumours may have unexpectedly low basal and/or minimally stimulated cortisol after ACTH administration occasionally with values at or below the cut-offs traditionally used in the low dose dexamethasone suppression test[4,5]. It is in these dogs that measurement of cortisol precursors is most valuable as they remain elevated after ACTH administration in the face of minimal change in cortisol. It is presumed that the cortisol pathway in these animals is no longer intact because of partial or complete deficiencies in the enzymes necessary for cortisol production. Why such dogs maintain clinical signs of cortisol excess is controversial. Most cortisol precursors have limited glucocorticoid effect and other disorders or physiological states associated with elevated concentrations do not give rise to the same clinical signs. It is possible in some animals that total daily production of cortisol is inappropriately high despite a lack of cortisol stimulation after ACTH administration. It is also possible that high circulating concentrations of these hormones displace cortisol from its binding proteins elevating the free and active portion.

Ectopic production of ACTH by a non-pituitary tumour is a well-recognised phenomenon in humans and accounts for up to 15 % of all cases of hyperadrenocorticism. It is generally associated with small cell carcinoma of the lungs and less frequently other tumours such as

neuroendocrine tumours of pancreatic or gastrointestinal origin and C cell carcinomas of the thyroid gland. Often associated with extreme elevations in cortisol and pronounced clinical signs, administration of corticotropin releasing hormone (CRH) generally does not increase ACTH concentrations. The tumours may be very small and advanced imaging techniques may be necessary for their location. There has been a recent report of probable ectopic ACTH production in a dog[6]. Aberrant expression of functional hormone receptors in the adrenal gland may also give rise to hyperadrenocorticism. A recent case report described a dog with clinical signs of hyperadrenocorticism and potential food induced hyperadrenocorticism[7]. Based on these two case reports it has now been suggested that hyperadrenocorticism be viewed as being ACTH dependent (PDH and ectopic ACTH production) and ACTH independent (adrenal tumours and food induced).

Hyperfunction of the adrenal gland may also involve mineralocorticoids or catecholamines. Hyperaldosteronism associated with adrenal neoplasia (Conn's syndrome) in humans is usually associated with hypokalaemia, hypertension and metabolic alkalosis. Conn's syndrome is more commonly recognised in cats but has occasionally been diagnosed in dogs. It may be related to excess aldosterone production or its precursors and diagnosis requires depiction of their elevation together with reduced renin activity.

Phaeochromocytomas are increasingly recognised in dogs. The majority are unilateral and they are commonly malignant. Clinical signs relate to catecholamine excess but as catecholamine secretion is variable, these signs may be sporadic, unpredictable, intermittent or paroxysmal. Signs typically involve the cardiovascular and neuromuscular systems. Whilst hypertension is considered the cardinal sign, it is only present in approximately 50 % of cases. There may be many non-specific signs such as polyuria/polydipsia or signs related to tumour growth. Diagnosis is best made using measurement of urinary free normetanephrine concentration or urinary normetanephrine:creatinine ratio[8]. Many case reports have suggested that dogs with phaeochromocytoma potentially have disorders akin to multiple endocrine neoplasia syndromes in humans. However, few have included all the typical manifestations noted in humans and the concurrent involvement of tumours in several endocrine glands simultaneously may be purely coincidental in dogs.Increasingly adrenal masses are found during either abdominal ultrasonography or CT where other non-endocrine diseases are being investigated. Overall the prevalence is between 4 and 9 % and they are typically found in older animals[9,10]. Tumours > 2 cm in size are more likely to be malignant. When found diagnostic investigation should centre on determining whether functional or not.

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43RD WORLD SMALL ANIMAL VETERINARY ASSOCIATION CONGRESS AND 9TH FASAVA CONGRESS

WSV18-0309

CURRENT TIPS ON THE MANAGEMENT OF CANINE URINARY TRACT INFECTIONS

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Current tips on the management of canine urinary tract infections

Urinary tract infection (UTI) refers to microbial colonization of any portion of the urinary system that is normally sterile. The distal urethra is not sterile; it has a normal flora (1). UTIs are often caused by bacterial organisms that are part of the microflora of the intestinal tract (2). Bacterial UTI is said to occur in about 14% of all dogs at some time during their life (3) and the infection rate was higher in females than in males as shown at necropsy. The infection rate was highest in dogs younger than 2 years and older than 6 years reaching 50% in females older than 10 years (4).

Infection can occur either in the upper or the lower urinary tract or in both sites at once. It might sometimes be difficult to identify the location of an infection. Furthermore, an infection in one part of the urinary tract increases the likelihood of another part of the urinary tract becoming infected as well (1). Most UTIs are the result of ascending migration of pathogens from the distal urogenital tract to the sterile part (5). UTI develops when the host's defenses are overwhelmed by microbes. Normal defenses include washout of pathogens by normal micturition with complete emptying of the bladder, mucosal layer with glycosaminoglycanes, epithelial desquamation, functional properties like ureteral peristalsis and local and systemic immune competence. Furthermore urine itself has antimicrobial properties that may play a role in limiting bacterial growth and include high osmolality, urine constituents with antimicrobial effect (e.g. high concentration of urea, organic acids, Tamm-Horsfall mucoproteins or low-molecular weight carbohydrates) and extreme values of urine pH (6, 7). Not all microbes are pathogenic; bacteria need special virulence factors to initiate UTI. UTI is usually caused by one single bacterial species. Predominant bacterial species in a multicenter study from Europe were one study were E. coli (60%), Proteus spp. (12%), Staphylococcus spp. (11%), Enterococcus spp. (5%), Streptococcus spp. (4%), Klebsiella spp. (2%), and Enterobacter spp. (1%) (8).

Typical clinical signs of lower UTI are stranguria, pollakiuria, and hematuria. As the bladder and the proximal urethra are so close together, infection in one is very likely to affect the other. Asymptomatic bacteriuria is also common in animals and it is difficult to localize it in either the upper or the lower urinary tract (9). It is often seen in animals with compromised host defense, such as those with glucocorticoid excess or diabetes mellitus.

The gold standard for diagnosis of UTI is urine culture. Examination of the urine sediment provides some help in the identification of UTI. More than four white blood cells per 400X field in unstained sediment under a cover slip together with bacteria identified during the same examination are indicatives. However the presence of pyuria represents any inflammation and is not synonymous with UTI and the absence of pyuria does not rule out UTI.

Cystocentesis is the preferred method of urine collection for culture, because lower genitourinary tract contamination is avoided.Antimicrobial treatment for urinary tract infections is subject to discussions. Antimicrobial resistance of bacteria causing urinary tract infection increase and the use of antibiotics in animals is or might be restricted in some countries because of the resistance situation. Administration of antimicrobial agents should be based on susceptibility testing. Empirical treatment should be avoided if possible. Overtreatment of urinary tract infection was found to be common (10). Current guidelines suggest amoxicillin and trimethoprim-sulfonamide as first line antibiotics (11). However, local susceptibility patterns should be considered if empirical treatment is necessary.

Uncomplicated UTIs are those in which no underlying problem is found and they should be treated for about 7 days. However, no studies have been done determining the exact length of treatment. Complicated UTIs are infections with an underlying anatomic, functional or metabolic condition preventing the clearance of an infection (9). The occurrence of three or more UTIs within 12 months is also considered a complicated UTI (11). In these animals, treatment for a longer period than the routine 7 days may be indicated (up to 4 weeks). In these cases, it may also be indicated to test the urine after the first week of treatment to evaluate the response to therapy, and after the end of treatment to make sure that no more bacteria are present.

Treatment of UTI is usually successful and nearly 75% of infections remain single episodes (12). Recurrent UTI might be caused either by the same organism which was isolated before treatment (refractory UTI or relapsing UTI) or by a different organism (reinfection). In both cases, further work up is required to identify the underlying causes. If predisposing disorders are not addressed, control of UTI will be poor.

Specifically for recurrent UTIs but also for uncomplicated UTIs alternative therapies are discussed. Cranberry and D-Mannose can inhibit the adhesion of bacteria to the bladder wall. Their efficacy has not been proven

in dogs. Probiotics are considered to reestablish a normal vaginal microbiome, but again proof is lacking. Immunomodulation by killed uropathogen bacteria essential oils or acupuncture are other treatment options discussed. In addition, colonization of the bladder with a human asymptomatic bacteriuria E. coli strain is considered as possible treatment option (13).

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WELLNESS

THE HAPPY, HEALTHY VETERINARIAN. HOW TO MAINTAIN YOUR MENTAL AND PHYSICAL WELL-BEING

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Aims of workshop

- Exploring the scope of veterinary wellbeing at the present moment
- Situation of professional welfare in different regions of the world (North America, South America, Europe, Africa, Asia)
- Creating a conversation about mental wellbeing
- Tips and tricks how to identify chronic stress, burn-out and what to do about it
- Tips and tricks how to communicate problematic situations with colleagues and what to do about it (Social networks & at work)
- Toolkits

Format of workshop

3 brief presentations from each of us then ask for audience participation?

- Ie. the main things they struggle with and we can discuss these topics, and give
- Practical tips what to do

Workshop Technique

Warm- up (5 minutes)

Nienke posts a brief assignment that may be done independently. This may be a brief reading, writing, editing, or problem solving activity to ready them for learning.

Mini-Lesson (10-15 minutes)

Nienke, Vicki & Martin provide whole class direct and explicit instruction in one of the following ways: conduct a shared reading demonstrating a reading strategy read and think aloud for a specific purpose teach a key concept demonstrate a writing strategy direct assistants in a hands-activity Nienke, Vicki & Martin outline the work to be done including the expectations as to how to apply the content learned in the minilesson to the work the expectation of completed work

Independent Work Time (30-40 minutes)

Assistants work independently, in pairs, or small groups. Nienke, Vicki & Martin circulate for 2 or 3 minutes to ensure all assistants are on task, and then confers with individuals for a few minutes, taking anecdotal notes work with a small group in direct instruction

Share Session (5 minutes)

Nienke reconvene class to focus on the work of one or two assistants that use what was taught in the mini-lesson recap key learning of the day (1 minute) check for understanding (with short reflective writing or exit slips) give homework feedback.

WSV18-0133

SVA DERMATOLOGY (SIMULTANEOUS TRANSLATION INTO MANDARIN CHINESE)

NEW CONCEPTS OF STAPHYLOCOCCAL INFECTIONS: MICROBIOME AND BIOFILMS

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MICROBIOMES

The skin is a complex ecosystem and is colonized by a wide variety of microorganisms, including bacteria, fungi, and viruses. The microbiome encompasses the full complement of microorganisms, their genes, and their metabolites. The normal skin microbiota is necessary for optimal skin fitness, modulating the innate immune response and preventing colonization of potentially pathogenic microorganisms.

Studies using sequencing of 16 S rRNA genes have revealed that the skin surfaces of humans and companion animals are inhabited by a highly diverse microbiota that was previously not appreciated by culture-based methods. Furthermore, there are topographic differences in the various skin surfaces, with the microbiota from similar skin locations of different people being more closely related than different skin locations from the same individual. Temperature, pH, moisture, environmental contact, and contact with mucous membranes are some of the factors that may influence the variability of bacterial abundance and distribution on the skin. In humans, **Propionibacterium** predominantly colonize the sebaceous areas, **Staphylococcus** and **Corynebacterium** are commonly found in moist areas, and gram-negative organisms are more likely to colonize dry skin areas such as the forearm or leg. The skin microbiota also changes with age, with infants having significantly different microbial populations than adults. Human skin also harbours a diverse fungal microbiome. The genus **Malassezia** is most abundant in all skin regions, with 11 of the 14 known **Malassezia** species being identified among skin sites. The plantar heel is the most diverse site with higher representation of different fungal genera, including **Malassezia**, **Aspergillus**, **Cryptococcus**, **Rhodotorula**, and **Epicoccum**.

The Skin Microbiome in Healthy Dogs

The diversity of the skin microbiota in different cutaneous and mucocutaneous regions in healthy dogs has been demonstrated. Similar to humans, different skin sites from dogs are inhabited by a variable and unique microbiome, with significant individual variability between samples from different dogs and between different skin sites within the same dog. A large number of

previously uncultured or rarely isolated microbes have been identified, demonstrating that the skin of dogs is inhabited by diverse microbial communities. Higher microbial diversity was observed in the haired skin (axilla, groin, periocular, pinna, dorsal nose, interdigital, lumbar) compared to mucosal surfaces or mucocutaneous junctions (lips, nose, ear, and conjunctiva). The nostril and conjunctiva showed the lowest, while the axilla and dorsal aspect of the nose showed highest microbial diversity. On average, around 300 different bacterial genera were identified on the canine dorsal nose. The most abundant phyla across all surfaces were Proteobacteria, Firmicutes, Actinobacteria, and Bacteroidetes (Rodrigues Hoffmann 2014).

The Skin Microbiome in Cutaneous Disease

The normal skin microbiota is necessary for optimal skin function, modulating the innate immune response and preventing colonization with potentially pathogenic microorganisms. In many skin conditions, it remains unclear if changes in the microbiome play a causal role in skin diseases or are rather the result of the disease. In humans with atopic dermatitis (AD) and psoriasis, the changes in the cutaneous microbiome have been proposed to be the result of an altered epidermal barrier function, Toll-like receptor 2 defects, decreases in antimicrobial peptides, and/or increased expression of extracellular matrix proteins. (de Jongh 2005). These mechanisms are thought to be responsible for an increased abundance of **Staphylococcus aureus** and susceptibility to staphylococcal infections in AD patients.

Similar to humans, dogs develop AD with hypersensitivity to environmental allergens such as house dust mites and/or food allergens. Recurrent infections with **Staphylococcus** sp. are very common in AD dogs, and in some dogs bacterial products can also trigger lesions of AD, possibly due to an altered epidermal barrier function.

A marked reduction in microbial diversity is observed in children during AD flares, and it is proposed that these changes precede an increase in the severity of AD. The skin microbiome colonizing the haired skin of dogs with allergic skin disease also demonstrates a lower bacterial diversity when compared to the same skin sites (axilla, groin, and interdigital skin) of healthy dogs (Rodrigues Hoffmann 2014). Significant differences in bacterial taxa have been observed between allergic and healthy dogs, especially higher abundance of Betaproteobacteria in the skin of healthy dogs.

Using culture-based methods, the skin and nasal mucous membranes of atopic human patients and dogs are more often colonized with **S. aureus** and **S. pseudintermedius**, respectively, than healthy patients. Based on 16S rRNA pyrosequencing data, **S. aureus** markedly dominated affected skin regions, more commonly the antecubital and popliteal creases, in children with AD. Likewise,

baseline and post flare samples from children with AD also had more abundance of **S. aureus** compared to the skin of healthy children. Allergen challenge in experimentally sensitized atopic dogs leads to bacterial dysbiosis with increased abundance of **S. pseudintermedius** at the site of lesion induction (Pierezan 2016).

The skin microbiota of atopic dogs has been longitudinally evaluated with parallel assessment of skin barrier function at disease flare, during antimicrobial therapy, and post-therapy. Sequencing of the bacterial 16S ribosomal RNA gene showed decreased bacterial diversity and increased proportions of **Staphylococcus** (**S. pseudintermedius** in particular) and **Corynebacterium** species compared with a cohort of healthy control dogs. Treatment restored bacterial diversity with decreased proportions of **Staphylococcus** species, concurrent with decreased canine atopic dermatitis severity. Skin barrier function, as measured by corneometry, pH, and transepidermal water loss also normalized with treatment. Bacterial diversity correlated with transepidermal water loss and pH level but not with corneometry results (Bradley 2016).

In cAD there is a predisposition to the development of coagulase-positive **Staphylococcus** species colonization and dermatitis as in AD. **S. aureus** is the primary coagulase-positive **Staphylococcus** species of human skin and mucosal sites. **S. pseudintermedius** is a skin and mucosal commensal in the dog and the most frequent pathogen isolated from dogs with skin or ear canal infections. Human **S. pseudintermedius** colonization is rare and primarily restricted to those with regular contact with dogs and cats. **S. aureus** is infrequently isolated from infection and carriage sites of dogs in clinical practice and in epidemiological surveys, and it is considered a comparatively infrequent canine pathogen. The dog may act as a potential vector of **S. aureus,** which raises zoonotic and anthropozoonotic concerns for potential transfer of pathogens, drug resistance, and genetic elements.

Microbiomes are often shared between dogs and humans, with pet owners having a more diverse microbiome than non-pet owners. Dogs that cohabit are also likely to have similar microbiomes. These concepts become very important in the context of antimicrobial resistance, where resistance genes can be spread from one bacteria to another and become established within the commensal population.

BIOFILMS

What is a biofilm?

A bacterial biofilm is a complex, sessile community of bacteria embedded within a self-produced matrix of carbohydrates, proteins and DNA (extracellular polymeric substance, EPS). Within a biofilm, bacteria

have markedly altered metabolism, enhanced cell-to-cell communication, and are able to evade the host immune response and the effects of antimicrobials through their isolated metabolism along with physical and chemical protection of the biofilm matrix.

Biofilm formation is influenced by a number of factors including:

- Bacterial species:
- Environmental conditions: temperature, pH, oxygen concentration, iron availability
- Surface: rough versus smooth

The stages of biofilm formation progress from initial attachment to the surface, through a phase of maturation, to a final phase of dispersion. Once the biofilm has formed, it confers a number of survival advantages for the bacteria that can have significant effects on pathogenicity including:

- Creates a high density of bacteria
- Increased metabolic efficiency
- Evasion of host defences such as phagocytosis
- Exchange of genes resulting in more virulent strains
- Increased production of toxins
- Protection against microbial agents
- Provides a nidus of infection that permits detachment allowing spread to other sites

How are they relevant?

Biofilms have a major impact on treatment and antimicrobial resistance, particularly in canine otitis externa. They are common and under-diagnosed, although they can be easily identified on otoscopy or cytology. Clinically, they form an adherent, thick and slimy discharge that is often dark brown or black in both the external ear canal and middle ear cavity. On cytology they appear as variably thick veil-like material that may obscure bacteria and cells. Biofilms are clinically important as they inhibit cleaning, prevent penetration of antimicrobials and provide a protected reservoir of bacteria. Also, antimicrobials that require bacterial division will be less effective, as bacteria in biofilms are usually in a quiescent state. Biofilms may also enhance the development of antimicrobial resistance, especially in Gram-negative bacteria that acquire stepwise resistance mutations to concentration-dependent antimicrobials.

Biofilm production by canine otitis isolates of **P. aeruginosa** is common and may play a role in the pathogenesis of disease. The MICs for biofilm-embedded bacteria differ from their planktonic counterparts, potentially leading to a lack of response to treatment. In one study, 40% of canine otic isolates of **P. aeruginosa** were classified as biofilm producers. Biofilm MICs for polymyxin B, neomycin, gentamicin and enrofloxacin were significantly higher than for the planktonic form. If these medications are used for topical treatment of a **Pseudomonas** otitis, the concentration of the medication should be increased. (Pye 2013)

N-acetyl cysteine (NAC) is a mucolytic with antibacterial and antioxidant properties. It is also otoprotective and can prevent chemotherapy induced hearing loss and contributes to detachment of biofilms associated with **P.aeruginosa**. Recently it has been demonstrated that NAC has an inhibitory effect in vivo against common pathogens isolated from canine otitis externa; **S.pseudintermedius, Pseudomonas aeruginosa, Corynebacterium and haemolytic Streptococcus**. The product appears to be safe and well tolerated and has an inhibitory effect at a concentration of 1%; middle ear mucosal inflammation was reported at concentrations > 2% and conductive hearing loss at 4%. (May 2016). NAC could be incorporated as part of the ear cleaning routine if a biofilm is suspected.

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WSV18-0050

SVA REHABILITATION

EXTRACORPOREAL SHOCK WAVE THERAPY (ESWT): INDICATIONS AND SCIENTIFIC EVIDENCE

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EXTRACORPOREAL SHOCK WAVE THERAPY (ESWT): INDICATIONS AND SCIENTIFIC EVIDENCE Janet B. Van Dyke, DVM,

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Therapeutic plans generally involve a combination of manual therapies (joint mobilizations and soft tissue mobilizations), physical modalities (laser, therapeutic ultrasound, e-stim, shockwave), and therapeutic exercises. The modalities are generally used to prepare the tissues for the manual therapies and therapeutic exercises. Physical modalities should never be the sole therapeutic method applied to any patient.Therapeutic parameters for each modality are chosen based upon the acuity of the injury, so the therapist must be well versed on the definitions of the acute, subacute, and chronic phases of healing.Extracorporeal shock wave therapy is also known as high energy focused sound wave therapy. This is a high energy pressure wave, or impulse, produced by supersonic craft, explosions, lightning, or other extreme phenomena that create sudden, huge changes in pressure. This is essentially a controlled mini-explosion creating energy that we can focus for treatment. The device has a 'trode' head in which a spark is generated, the energy from this spark is reflected off of a surface and then focused through the trode head's bulb. The depth of penetration is determined by the thickness of this bulb.Shock waves are rapid, high energy sound waves characterized by an extremely rapid rise time or 'pressure front', followed by a slight negative pressure dip that causes cavitation, and resulting in a clearly defined focal area in which the energy is concentrated. There are four types of shock wave device: Electrohydraulic, electromagnetic, piezo-electric, and radial pressure wave. Electrohydraulic devices have peak pressures of 70Mpa, rise times of nanoseconds, and create true shockwaves at all settings. Electromagnetic devices have a peak pressure of 20MPa, rise times measured in microseconds, and create true shockwaves at high energy settings only. Piezo-electric devices have peak pressures of 15MPa, rise times in microseconds, and create true shockwaves at high energy settings only. This group is used for lithotripsy. Radial pressure wave devices have a peak pressure of 0.4MPa, rise times in milliseconds, and they do not produce a shock wave at any setting. The energy is completely dissipated at 0.5 to 1.0cm of depth. These devices are marketed as 'painless shock wave devices, requiring no sedation'.

Shock waves stimulate release of many cytokines including nitric oxide, VEGF, PCNA, TGF-b1, and BMP2, leading to a brief inflammatory phase, vasodilation, neovascularization, endothelial cell proliferation, tissue healing, and osteogenesis. ESWT causes decreased inflammation via downregulation of TNF-a and IL-10, increased bone and tissue healing via increased secretion of BMP2, TGF-b, VEGF, PCNA, and eNOS, and decreased bone via release of serotonin in the dorsal horn of the spinal cord, leading to descending inhibition. Additional medical effects of ESWT can be disruption of biological biofilm, resulting in a bactericidal effect, decreased cartilage degradation, and a temporary analgesic effect, lasting 3-4 days in one equine study. Studies have revealed beneficial effects of ESWT in treating delayed and non-union fractures, leading to 76% success in healing delayed unions (as compared to 79% success with re-operation). Used preemptively, a significant decrease was found in the incidence of non-unions in high risk fractures. In a fracture healing study in canine fracture gap models, the use of ESWT lead to significantly greater callus and significantly more cortical bone at 12 weeks post-op. In a clinical canine study reported in VCOT in 2002, four of 6 dogs with non-union fractures were treated with ESWT. Three of the 4 treated dogs healed.

ESWT is used to treat osteoarthritis as well as fractures. A study reported in VCOT in 2005 showed a trend over the 14 weeks of treatment toward improved range of motion with no change in peak vertical force (PVF) in the treated group while the control group showed a significant loss of PVF. Another study in 2010 reported a significant increase in PVF in elbow OA patients treated with ESWT. This magnitude of change in PVF was similar to that seen in patients treated with NSAIDs. A study reported in Vet Surgery in 2012 showed a significant decrease in patellar ligament thickening at 6 and 8 weeks post-surgery in TPLO patients treated with ESWT. Cauda equina cases treated with ESWT resulted in an 87.5% positive response with a median duration of 13.6 months. A study out of Tufts University in 2015 revealed excellent short-term results in treating dogs with chronic lameness due to shoulder disease. Another study published in Vet Record in 2016 looked at ESWT and therapeutic exercise for supraspinatus tendinopathies. 85% of these cases had good or excellent outcomes in both short term and long term follow up. Equine studies have shown decreased lameness and improved ROM 2 months post ESWT for carpal joint osteoarthritis.

Application of ESWT requires proper patient preparation. Sedation is recommended as the device creates noise and can cause some pain. It is recommended to shave the area and wipe with alcohol. Ultrasound gel is used as a medium to provide optimal sound wave transmission. The trode is moved around the treatment area, angling the head as needed to reach the target tissue. Generally, 500 to 1000 pulses are applied to each treatment area. The devices can generate 48- pulses per minute, so most treatments take approximately 2 minutes per site. The patient can be discharged as soon as recovered from sedation. The analgesic effect of this therapy necessitates limiting patient activity for three to 5 days post-treatment. NSAIDs may be used, but this may reduce the desired brief inflammatory healing effect. Patients are reevaluated every 2 weeks and retreated as needed. Most indications get the best results with two to three treatments.

WSV18-0135

SVA DERMATOLOGY (SIMULTANEOUS TRANSLATION INTO MANDARIN CHINESE)

OCLACITINIB VS LOKIVETMAB

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OCLACITINIB

Oclacitinib maleate (Apoquel®) is a novel targeted therapy that selectively inhibits key pathways involved in itch and inflammation associated with allergy. Oclacitinib selectively inhibits Janus kinase 1-dependent cytokines with minimal effects against Janus kinase 2-dependent cytokines involved in haematopoiesis. Janus kinase 1 enzyme activities play a central role in cytokine signalling and are involved in the signal transduction of many pro-inflammatory, pro-allergic and pruritogenic cytokines implicated in atopic dermatitis, including interleukin (IL)-2, IL-4, IL-6 and IL-13. Janus kinases are also involved in the signalling of IL-31, a recently identified cytokine that has been shown to play a key role in canine pruritus. Oclacitinib has been shown to inhibit IL31 cytokine function in dogs.

Oclacitinib is indicated for control of acute or chronic pruritus in dogs over 12 months of age. The drug is not approved for dogs less than 12 months of age. Oclacitinib administered at a dose of 0.4 to 0.6 mg/kg orally twice daily for up to 14 days and then once-daily thereafter for dogs is highly effective for the management of pruritus and skin lesions in dogs with allergic dermatitis. Oclacitinib has been shown to reduce pruritus and clinical signs as effectively as prednisolone (Gadeyne 2014) and ciclosporin (Little 2015). The drug has a very rapid onset of action with relief sometimes apparent within hours of oral administration. Some dogs experience an increase in pruritus when switched from twice to once daily, related to the short half-life of the drug (4 hours). Overall, it appears that at least 60–70% of allergic dogs receiving the drug have rapid, substantial, and prolonged relief of their clinical signs. Veterinary dermatologists often use this drug as part of a multimodal treatment approach.

Short term adverse effects of oclacitinib appear mild and include gastrointestinal side effects of vomiting and diarrhoea at an incidence of approximately 2%. The long-term administration of oclacitinib administered once-daily appears to be relatively safe. Results of a long-term compassionate use study support the safety of chronic use of oclacitinib and suggest an improved quality of life for the dog and the owner (Cosgrove 2015). Oclacitinib has been administered for as long as 3 years in some dogs; in longer-term studies, occasional patients have developed benign or malignant neoplasms, but no more often than would be expected for dogs in the studied age range.

The drug has been limited to use in dogs 12 months or older, predominantly because in one high-dose safety study, generalized demodicosis developed in some young laboratory dogs. Oclacitinib has not been evaluated in combination with other drugs such as systemic corticosteroids or ciclosporin; and concurrent use should be avoided with these drugs. It can be used safely in conjunction with antibiotics, antihistamines, antifungal drugs, NSAIDs, allergen-specific immunotherapy and vaccination of treated dogs is effective. Oclacitinib does not appear to interfere with serologic or intradermal allergy tests. As with other immunosuppressive treatments, oclacitinib modulates the immune system and may increase susceptibility to infection and infestation and exacerbate neoplastic conditions. It is contraindicated in dogs with severe infections, demodicosis, or with active malignancy.

LOKIVETMAB

Lokivetmab (Cytopoint®, Canine Atopic Dermatitis Immunotherapeutic®) is a caninized anti-canine interleukin (IL)-31 monoclonal antibody that binds to and neutralises circulating IL-31, thereby inhibiting its binding to the IL-31 receptor. The subcutaneous administration of lokivetmab results in a dose related reduction in canine IL-31-induced pruritus in dogs for up to eight weeks following a single dose. A blinded placebo controlled trial revealed a greater reduction in pruritus for at least one month compared to placebo and the level and duration of response was shown to increase with increased dose (Michels 2016). In a clinical trial involving client-owned dogs with atopic dermatitis, a single subcutaneous injection of lokivetmab at a dose of 2 mg/kg began to reduce pruritus within one day and was effective for a full month for 80% of affected dogs.

Lokivetmab has a good safety profile. Adverse effects are minimal and include vomiting, diarrhea, and lethargy. (Michels 2016). In a field safety study, lokivetmab was well tolerated in dogs after two subcutaneous monthly injections. A wide variety of concomitant medications were safely used, including parasiticides, antibiotics, antifungals, corticosteroids, vaccines, immunotherapy, antihistamines, and other antipruritics, such as oclacitinib and cyclosporin (Michels 2016). Lokivetmab has also been demonstrated to be well tolerated in a laboratory safety study in which seven consecutive monthly subcutaneous injections were administered to laboratory beagle dogs at doses of 3.3 mg/kg or 10 mg/kg body weight (12 dogs per group) (Zoetis data).

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43RD WORLD SMALL ANIMAL VETERINARY ASSOCIATION CONGRESS AND 9TH FASAVA CONGRESS

WSV18-0009

GETTING CALORIES INTO THE INAPPETANT OR ANORECTIC CAT

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Inappetence and anorexia are common problems in feline patients. Inadequate nutrient intake is, at best, detrimental and interferes with healing. At worst, it is life-threatening. Cats have only a limited ability to conserve body protein; this can result in negative nitrogen balance, protein: calorie malnutrition and deterioration of protective mechanisms impacting immunity, red cell hemoglobin content, muscle mass as well as the ability to repair tissues. Additionally, cats have limited storage of many other nutrients as well as a restricted ability to down-regulate numerous metabolic processes. Their design is best suited to eating multiple small meals per day, high in protein, and moderate in fat. Inappetence and anorexia should be dealt with promptly and adequately.

Meeting the patient's nutritional needs is not a substitute for localizing the cause for this inappetence. It is, however, necessary and allows time to identify the cause. Providing nutrients may be the most challenging part of any therapeutic regimen, and recovery or attaining the best possible QOL in cats may depend on our ability to ensure optimal nutrition.

The first question that must be answered is: why has this cat stopped eating? Is it because of a loss in appetite or some other reason? Nausea may be of neurologic origin (e.g., vestibular disease or irritation of the chemoreceptor trigger zone or the vomiting center by inflammation, neoplasia or chemicals including metabolites or drugs). It may be a result of dehydration or may originate with GI inflammation for any reason (e.g., ileus, colitis, upper intestinal or gastric disease). However, decreased food intake may be due to other factors, such as dysphagia, pain (e.g., oral, dental, GI, multisystemic, etc.), dislike of the diet (e.g., boredom, altered palatability, spoilage), aversion, fear (e.g., environmental changes including those in the social demographics).

Nutritional support should be considered for the severely malnourished cat (20% weight loss, body condition score 1-2/9) or moderately malnourished (a 10% weight loss, BCS 3-4/9) who also have catabolic disease. Some cats will benefit from early intervention even at normal weight and condition if they suffer from advanced renal disease, hepatopathy, protein losing GI or glomerular disease, pancreatitis or bile duct obstruction.

Inappetent cats, and those not ingesting adequate protein, shift into a catabolic state. They are at risk for hepatic lipidosis, especially if ill and possibly at a greater risk if previously obese. Lipidosis is a disease of dysfunctional lipoprotein metabolism; it is important to calculate the daily caloric and protein requirements as part of the therapeutic plan. [Calories: 50 kcal/kg ideal BW/day; 4.5 g protein/kg ideal BW/day]. The diet needs to be balanced for energy (protein, fat, +/- carbohydrates), vitamins and minerals. It needs to be palatable taking the following four factors into account: texture, aroma, taste, and consistency. Bowls should be wide and flat to avoid interfering with whiskers. The environment should be non-threatening, so a hospital setting is especially off-putting. Feline facial pheromone may be beneficial to reduce stress.

Rehydration and correction of electrolyte imbalances are important but oft overlooked goals in the correction of inappetence and anorexia. Anti-emetics have a place if the cat is vomiting. In gastric-origin nausea, agents such as H2 antagonists, gastroprotectants, proton pump inhibitors or prostaglandin E agonists may be beneficial depending on the cause of the gastric upset.

Appetite stimulants including cyproheptadine (1 mg/cat PO BID), mirtazapine (1-2mg/cat PO q48h) may help jump-start a cat's appetite, but keep track of total calories consumed. If a cat is eating but not enough, supportive feeding (assisted syringe feeding or tube feeding) must be considered. A cat eating small amounts of baby food will not meet his caloric needs until he eats 2-3 jars/day. Meat baby food is not balanced, but is sufficient for several weeks. There are several diets specifically designed for the assisted feeding of cats (Royal Canin Recovery, Hill's a/d, Purina PVD CN, Eukanuba Maximum Calorie), liquid balanced enteral diets for cats (Clinicare, Rebound) Additionally, we can make a slurry from any canned food; blend with a liquid feline diet rather than water to minimize loss of calories.

There are several options for assisted feeding each with advantages and disadvantages. In general, the author starts with syringe assisted feeding until the cat is stable enough to allow the brief anaesthetic required for the placement of an esophageal tube. With concurrent liver disease, give three doses of Vitamin K1 (1.0 mg/kg q12h SC) prior to tube placement, biopsies or any other procedure that might result in bleeding. Placement of esophageal tubes is discussed elsewhere. The instrumentation for this procedure is very basic requiring only the following: 14-16 Fr red rubber feeding tube/urinary catheter, Carmalt or other long curved forceps, a scalpel blade, suture and bandaging materials and a multiple use injection port (prn adaptor).

Calculating how much to feed requires that you know the patient's current weight as well as their healthy weight and the caloric densities (kcal/ml) of the diet you are intending to use (see Table 1). Use 50 kcal/kg as a rough guide to determine calories needed. Start by feeding 1/3-1/2 of the calories needed for the current, inappetant

weight. On day two, feed 2/3-3/4 of this number and on day three, feed the full calories needed for the current weight. For weight gain, gradually increase to the calories needed for the cat's healthy weight.

Example:

3.4 kg sick cat BCS 3/9, healthy weight 4.0 kg BCS 5/9

3.4 kg X 50 kcal/kg/day = 170 kcal by day 3

170 kcal = 81 ml Eukanuba Maximum Calorie

OR 131 ml of Hill's a/d or Royal Canin Recovery or PVD CN

Day 1 feed 30-40 ml of Max Cal or 44-65 ml of the other diets

Day 2 feed 54-61 ml of Max Cal or 87-98 ml of the other diets

Day 3 feed 81 ml Max Cal or 131 ml of the other diets.

Once stable, gradually increase to meet caloric requirements for 4 kg healthy weight.

4 kg X 50 kcal/kg/day = 200 kcal (95 ml Max Cal vs.154 ml of the other diets).

With surgically placed tubes there is a delay in how quickly one can start to use them; with an esophageal tube only a 2-3 hour delay is required to ensure full recovery from anaesthesia whereas gastrostomy and jejunostomy tubes require a longer wait of 10-12 hours. Cats can eat with any of these tubes in place. It is recommended to avoid offering food for a week to reduce the likelihood of them developing aversion to the food offered. Once a cat is eating well with tube in place the question becomes when one can remove the tube. Weigh the cat and, as long as he/she is eating well, avoid using the tube (for nutrients) for a week then reweigh the kitty. If the weight is stable (or increased), then it is safe to remove the tube. Because of stoma formation (except nasoesophageal tubes), removal does not require anaesthesia. Remove the suture (purse-string or stay sutures) and pull the tube out. In the case of a gastrostomy tube, its bulb must be straightened out the bulb/balloon by inserting a straight probe through the tube while concurrently pulling the tube out. Suturing is not required for any of the skin openings. Cleanse minimal serous discharge that may occur for 2-3 days.

Feeding frequency: the number of feedings per day, (and hence intervals), is determined based on the volume of food tolerated per feeding. Start with 6 ml and increase by 6 ml increments to about 36-48 for most cats. In the uncommon case of the patient who cannot tolerate even 6 ml boluses despite antiemetic therapy (see Pancreatitis notes in these Proceedings), trickle feeding may be instituted. Trickle feeding is a technique in which liquefied food is syringed into an empty fluid bag and administered gravitationally or by pump assistance via an

intravenous line attached to the large bore feeding tube or by use of a large syringe filled with food and syringe pump. Renew food and delivery tubing and syringe at 12-hour intervals to avoid bacterial contamination. A promotility agent may be warranted as well. A good client reference is the Animal Medical Center of Canberra's website: www.animalmedicalcentre.com.au => Pet Health => Articles => Cats => Tube feeding.

The success of assisted feeding is measured objectively by weight gain. Subjective measures will include improved coat quality, increased energy, muscle recovery and innumerable other effects that the client will appreciate. An improved quality of life is the goal whether recovery form the underlying problem is possible or not.

Table 1: Caloric densities of convalescent diets, for calculating feeding volumes:

ReboundTM: 1 kcal/ml

ClinicareTM: 1 kcal/ml

Royal Canin/MediCal RecoveryTM: 1.23 kcal/ml

Hill's a/dTM: 1.3 kcal/ml

Purina PVD CNTM: 1.23 kcal/ml

Eukanuba Maximum CalorieTM: 2.1 kcal/ml

Please email me if you would like the video of how to place an e-tube or for the using a feeding tube video.

SUGGESTED READING

Hodshon B, Tobias K. Esophagostomy Feeding Tubes Clinicians Brief February 2014 (www.cliniciansbrief.com/article/esophagostomy-feeding-tubes)

WSV18-0241

TIPS FROM THE EXPERTS FOR THE MANAGEMENT OF...

VOMITING CATS

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WSV18-0276

OPHTHALMOLOGY

TOP 10 HINTS FOR A GREAT EYE EXAM

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WSV18-0051

SVA REHABILITATION

REHABILITATION OF PEDIATRIC CANINE DISORDERS - HIP DYSPLASIA, ELBOW DYSPLASIA AND ANGULAR DEFORMITIES

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REHABILITATION OF PEDIATRIC CANINE DISORDERS – HIP DYSPLASIA, ELBOW DYSPLASIA AND ANGULAR DEFORMITIES

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Pediatric canine patients present to rehabilitation with many injuries and impairments. We will focus upon the three most commonly seen issues in patients presented to rehabilitation services: Canine hip dysplasia, canine elbow dysplasias, and angular deformities of the radius and ulna. Surgical intervention may or may not be indicated for this patient group. All will present with soft tissue impairments that can be addressed through rehabilitation techniques. Here the emphasis is upon obtaining a proper, thoroughly investigated soft tissue diagnosis, using good problem solving to create and meet goals that are functional, with a focus upon safety for this immature patient group.Canine rehabilitation is the application of physiotherapeutic techniques to evaluate and treat musculoskeletal impairments in our canine patients. It incorporates the use of objective outcome measures (goniometers, girthometers, etc.), manual assessments (including palpation, joint glides, and neurological assessment), gait analysis, and special tests brought from the field of human physiotherapy. This allows the therapist to tease out the specific structure and tissue type causing the impairments.The therapist evaluates the presenting complaint, subjective information from the owner, and objective assessment carried out during the examination to create a problem list. Each item on the problem list is addressed in the plan of care.Therapeutic plans generally involve a combination of manual therapies (joint mobilizations and soft tissue mobilizations/stretches), physical modalities (laser, therapeutic ultrasound, e-stim, shockwave), and therapeutic exercises. The modalities are generally used to prepare the tissues for the manual therapies and therapeutic exercises. Physical modalities should never be the sole therapeutic method applied to any patient. Therapeutic exercise plans are based upon the weight bearing status of the patient, with early interventions focusing upon functional weight bearing exercises, later progressing to functional strengthening exercises. All exercise plans incorporate, balance, strength, flexibility, and endurance. Exercise equipment includes physioballs (shaped as rolls, peanuts, eggs, donuts and balls), cavaletti poles, therapy band, rocker/wobble boards, and treadmills. Exercise programs are always started within the patient's comfort zone. This is especially important with puppies. The intensity of the exercise is gradually increased until the goals are met. Then, the exercise is changed. The key with keeping puppies engaged in the exercise program is remembering to keep each exercise fun, recognizing early signs of boredom or stress, and being ready to change the program to address each.Physical modality parameters are chosen based upon the acuity of the injury. They are used to prepare the tissues for additional therapy and can generally be applied by trained veterinary nurses. The most commonly used physical modalities include neuromuscular electrical stimulation, laser, therapeutic ultrasound, extracorporeal shock wave therapy, and ice/compression units. Precautions for using modalities with pediatric patients involve issues with open physes. Laser and therapeutic ultrasound are both contraindicated in the physeal areas for this patient group.Puppies who have hip dysplasia, elbow dysplasia or angular limb deformities will present with soft tissue impairments that can be addressed through rehabilitation techniques. Puppies who present with canine hip dysplasia will have joint capsule pain, lack of gluteal muscle development, and pectineus muscle pain. Elbow dysplasia patients present with joint capsule pain and shortening, muscle atrophy, and adaptive shortening of the muscles around the joint. Puppies who have angular limb deformities involving the radius and ulna will present with of carpal range of motion, elbow incongruity with pain, and adaptive shortening of the muscles around the carpus and elbow.Puppies with hip dysplasia pain related to joint capsule and periosteal pain can be treated via manual therapies including Grade 1-2 joint mobilizations to alter nociceptor response. If the physes are closed, laser can be applied to treat pain. Lack of gluteal development can be addressed through manual therapies, stretching the hip flexors that have become shortened. Therapeutic exercises that encourage hip extension and abduction include applying peanut butter to the groin of the puppy who will then actively abduct and extend the hip to lick the peanut butter. Cavaletti poles as well as backward and sideways walking will assist with muscle strengthening. Tummy rubs can be used to encourage active hip abduction. Hip stabilizer muscles can be strengthened through use of wobble boards and blocks. Pectineus pain can be treated via manual therapies to stretch the muscle after therapeutic ultrasound to heat and soften the tissues and laser to address pain.Puppies with elbow incongruity due to elbow dysplasias may

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present after surgical interventions. Joint capsule pain can be addressed through Grade 1-2 joint mobilization. Laser can be used to address pain and cryotherapy can be applied after activity. Muscle atrophy is addressed through therapeutic exercises including cavaletti poles, physioball work, and training to complete ''High 5's". Elbow stabilizers can be strengthened via block work and proprioception exercises. Adaptive shortening around the elbow is treated via Grade 2-3 joint mobilizations and passive as well as active stretches focusing upon the biceps brachii and brachialis muscles. The most common cause of angular deformity in the radius and ulna is premature closure of the distal ulnar physis. This results in the distal radius having cranial-medial convexity and secondary elbow subluxation. The age at the time of the injury and the time from injury to therapy determine the degree of deformity. Angular deformity generally occurs before 4 months of age, with premature closure occurring 3-4 weeks post injury. The gross deformity happens 2-3 weeks later. Surgical options include CORA based procedures and proximal ulnar osteotomy. When these puppies present to rehabilitation immediately post operatively, treatment includes cryotherapy to decreases swelling, Grade 1 joint mobilizations of the carpus and elbow as well as laser to treat pain. The soft tissue impairments associated with angular deformities include loss of carpal ROM, elbow incongruity with pain, and adaptive shortening of the muscles associated with elbow and carpus movement. Loss of carpal ROM is addressed through manual therapies: Grade 1-2 joint mobilizations and stretches, especially of the flexor carpi ulnaris and digital flexors. Therapeutic exercises are aimed at early weight-bearing work, later moving to strength exercises. Ultrasound is used to warm muscles prior to stretching, and laser is applied to speed wound healing. Elbow incongruity with pain and adaptive shortening are treated via joint mobilizations, triceps stretches, therapeutic exercise moving from early weight-bearing to later elbow flexion work, and ultrasound and laser as above.In conclusion, the proper approach to rehabilitation therapy involves an emphasis on proper, thorough soft tissue diagnosis, an emphasis on problem solving and creating and meeting goals that are functional for our patients, and an emphasis on safety for our immature patients.

WSV18-0189

HEMATOLOGY AND ENDOCRINOLOGY (SIMULTANEOUS TRANSLATION INTO MANDARIN CHINESE)

ALTERNATIVES TO ACTH STIMULATION FOR MONITORING TRILOSTANE THERAPY

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ALTERNATIVES TO ACTH STIMULATION FOR MONITORING TRILOSTANE THERAPY

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Since trilostane was first used to manage hyperadrenocorticism in dogs, the ACTH stimulation test has been recommended to monitor treatment efficacy. Although therapeutic cut-offs have changed over time, most endocrinologists are comfortable with interpretation of results and advising on any necessary dose changes. Its limitations are recognised. Timing of the ACTH stimulation test is crucial and it should be carried out 2-4 hours after treatment[1]. It is known that results of stimulation tests started at 2 or 4 hours after trilostane can yield significantly different results[2] and thus should be standardised for each individual dog. Additionally although considered excellent at identifying excessively controlled dogs, escape from inhibition can occur some hours later and therefore altering drug dosage may not be necessary[3].

Several studies have investigated alternative means of monitoring trilostane therapy using the ACTH response test for comparison. Varying but usually limited success has been reported for baseline cortisol, endogenous ACTH, cortisol/ACTH ratio, acute phase proteins and the urine corticoid:creatinine ratio[4-7]. None have gained widespread acceptance. However, these reports have been criticised for using the ACTH stimulation test for comparison as it has been suggested that it was never appropriately validated for the purpose of monitoring trilostane efficacy. Additionally, it has also been suggested that the results of the ACTH stimulation test correlate poorly with clinical control as assessed by owner questionnaire[8]. The recent problems with regard to availability of ACTH and its expense have highlighted the need for alternative monitoring strategies.One recent study evaluated the results of the ACTH stimulation test, baseline cortisol (3 hours post pill) and pre-trilostane cortisol (24 hours after trilostane administration for once daily dosing or 12 hours for twice daily dosing)

concentration compared with clinical control as assessed by owner perception[9]. Results indicated that baseline cortisol and pre-trilostane cortisol were better than the post-ACTH stimulation cortisol at differentiating between dogs with excellent clinical control and those that were inadequately controlled. The pre-trilostane cortisol was slightly better that the baseline cortisol and had the added benefit of being more useful at signalling potential oversuppression. It is this measurement that is now recommended for monitoring trilostane therapy. Broadly speaking a pre-trilostane cortisol > 40 and < 138 nmol/L, > 138 nmol/l or < 40 nmol/L indicates adequate, inadequate or excessive control, respectively. However, interpretation should only be carried out in light of clinical findings. Dogs with persistent clinical signs despite adequate cortisol concentration may still require an increased dose or frequency of administration. Dogs with good clinical control and cortisol concentrations suggestive of inadequate control may benefit from split dosing and dogs with excessive cortisol control may benefit from a lower dose. Dogs presenting ill when on trilostane therapy warrant an ACTH stimulation test to fully evaluate adrenal reserve.**References**

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UROLOGY

URINARY INCONTINENCE IN MALE DOGS

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Urinary incontinence in male dogs

Urinary incontinence is involuntary loss of urine during the filling phase of the bladder. Male dogs are less often affected by this problem than female dogs. Causes of urinary incontinence in dogs can be divided in congenital and acquired causes. Congenital causes can be ectopic ureters, congenital urethral sphincter mechanism incompetence, persistent urachus, bladder diverticula, hypoplasia of the bladder, prostate, or urethra, and hypospadia. Acquired causes can be hyperreflexia of the detrusor muscle, acquired urethral sphincter mechanism incompetence, detrusor atony because of bladder over distention, prostatic disease, neoplastic disease of the bladder, prostate or urethra, or neurologic disease (lower motor neuron disease).

While congenital urinary incontinence often starts in early life, acquired urinary incontinence can occur at any time. However, congenital diseases can become clinical later in life. For instance ectopic ureters can appear later in life because of the urethral closure pressure decreasing with age. Also neutering can decrease the urethral sphincter pressure and uncover an ectopic ureter.

Incontinence can be continuous and the dog loses urine all the time. In this case, the urethral closure is constantly overwhelmed. This might occur with ectopic ureters. Oftentimes the dogs only lose urine while sleeping. In this case the urethral pressure is overcome by the bladder pressure only after a certain filling volume in the bladder. This might be the case in urethral sphincter mechanism incompetence. Incontinence only when the dog is excited might indicate detrusor hyperreflexia. Urinary tract infection can occur together with other causes of urinary incontinence or can be the cause of the incontinence. With diseases like ectopic ureters or urethral sphincter mechanism incompetence the dogs urinate normally, while with diseases like urinary tract infection or neoplasia pollakiuria or stranguria can be seen.

The size of the bladder before and after urination can help to differentiate causes of urinary incontinence. A bladder that is always small indicates continuous loss of urine, a bladder that is not empty after urination indicates an obstructive lesion or detrusor atony. A bladder that is atonic and easy to express indicates lower motor neuron disease or overflow incontinence.

Urinalysis is indicated to identify urinary tract infections and to determine urine concentration ability. Bloodwork might help to identify underlying diseases.

Radiography, ultrasound, computer tomography or cystoscopy might help to identify the cause of the incontinence.

Congenital urethral sphincter mechanism incompetence can occur in combination with other malformations or alone.

Urethral sphincter mechanism incompetence in adult dogs can be associated with neutering and is more common in large breed dogs than small breed dogs. In one study Boxer, dogs were the most common breed with adult onset of incontinence

Therapy depends on the underlying disease. For urethral sphincter mechanism incompetence the alpha-adrenergic agonist phenylpropanolamine has been proposed. In 44% of the dogs response was considered good to excellent (Aaron et al 1996). Testosterone cypionate was used a recent study for urethral sphincter mechanism incompetence in 11 male dogs. Based on owners' assessments, a good to excellent response was reported in three of eight dogs (38%) (Palme et al 2017). Adjustable urethral hydraulic occluders have been successfully used in male dogs.

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WSAVA ONE HEALTH DISASTER MANAGEMENT

RADIATION-ASSOCIATED DISASTER MANAGEMENT - THE JAPAN EXPERIENCE

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Abstract

Following the great east Japan earthquake, Fukushima Daiichi Nuclear Power Plant (FDNPP) accident that happened 1-4 days later. In this proceeding the author will introduce our experience what we observed and worked for recovering from disaster. Apart from the initial radioactive iodine, the major source of radiation was radio-Cesium, namely ^{134}Cs and ^{137}Cs. Hopefully Strontium-90 was less than 1/1000 of radio-cesium and was negligible. To present knowledge, apart from observation of abnormalities found in batterfly, fish and birds, there is no report or observation on mammalian species those were suffered from acute or chronic radiation effects those include chromosomal or DNA damage, anomaly, and/or radiation induced tumor or cancer in Fukushima. However, accidents will happen. But forewarned is forearmed. Prevention is better than cure. VMAT (Veterinary Medical Assistant Team on disaster) was created and its guideline was proposed in Fukuoka (2012) and in Gunma (2016). They also started training for veterinary experts. Therefore, you can never be too prepared before it starts.

Introduction

On 11th March 2011, the great east Japan earthquake (magnitude 9.0) happened followed by gigantic Tsunami (wave height was over 10m and the maximum run-up height was over 40m) and the disaster caused by Fukushima Daiichi Nuclear Power Plant (FDNPP) accident that happened 1-4 days later. In severe damaged area, there was no electricity, no transport system and/or even no cars available simply because the roads were covered with mud with sea water and rubble, cars with no driver, and occasionally ships on the road. Moreover, many roads were cracking everywhere near FDNPP. In this series of the events over 15 thousands people were dead and about 91% of the cause was drowned by tsunami.

In this lecture the author would like to talk about the impact of nuclear accidents on animals and what we have seen and learned about Fukushima, especially focused on the eye from a veterinary radiologist working in the referral hospital and university. And our experience and suggestive instruction from animal victims in the ex-evacuation zone since the disaster caused by FNPPF accident according to the following regime.

The Purpose of this study is to explain the following successive issues

- What happened soon after the FDNPP accident...escape from evacuation zone and refugee
- How to rescue animals; A proposal to the government from animal/radiology experts
- Problems and efforts for rescuing companion animals and creating animal shelters and its management
- Investigations of companion animal radiation exposure and cumulative radiobiological effects
- Investigations of farm animal radiation exposure and cumulative radiobiological effects
- Investigations of wild lives' radiation exposure and cumulative radiobiological effects
- What we have learned from the nuclear accident and what should be done for the future prevention?

Results and discussion

Soon after the FDNPP accident, Fukushima local government declared to evacuate people within 2km from FDNPP, followed by Japanese government (JG)'s order to evacuate within 3km but was changed to 10km on that night. On the morning of next day JG declared state of emergency... and finally, JG decided to create 20km radius evacuation zone from FDNPP and people within 20-30km radius should have to keep sheltering in each house instead of evacuation. But along to the see coast, roads and electricity were no longer useful, therefore even such many people who succeeded in evacuation could not move to anywhere. Later of the FDNPP accident, the Ground radioactivity was monitored from air flight and radioactivity monitoring cars, which were opened the public for radioactivity-related information for initial evacuation.

IFAW: international fund for animal welfare immediately respond to this big event, calling Japanese and American veterinary, radiology and evacuation/management experts to discuss and give tutorial information to support JG's decision making for animal support. In the beginning of May 2011. They have got together in Tokyo with observers from MAFF and MoE, with attendance of a politician. This committee member successfully did make a guidance supportive to the JG (Nuclear Accidents and the Impact on Animals COMMITTEE RECOMMENDATIONS) in two days. This was also sent to JG, media including SNS and politicians who may relate for this action. Hopefully this could partially moved JG to make it possible to start rescuing animals. However in reality, only companion animal rescue was planned but farm animals were decided to euthanize by only with the agreement of owned farmers. Many farmers disagreed to kill them, since there is no clear reason.

Initial problems for animal rescue was there was no place or shelter, or prepared facility to accept animal refugee. This was one of the reason why the local government did not agree to start rescuing companion animals. But during the meantime of this no action by local government, valuable animals (retrievers and/or qualified breeds, and pappies in breeders) were stolen and sold by someone. Or someone in fanatic or aggressive animal welfare organizations took away to their own shelters and charged relatively expensive money for giving (back) to the (genuine or) second owners. Such unwanted information also spread out through SNS. And some (personal) organization of animal welfare asked fund raising to rescue animals through SNS, which created scam or fake organization only to imitate animal welfare activities. Some aggressive animal welfare organization tried almost to thread local government by long time phone attacks.

However an animal shelter in temporal use was created in June and voluntary staffs to manage or support were asked to join. A voluntary staff especially a famous actress" temporal help worked effectively since it soon spread through SNS. Companion animal rescue has just started on the opening day of the temporal shelter. However, the rescuing had to stop by the first day, which was simply due to the very limited capacity of the shelter to accept them. Alternatively, this was because owners of companion animal could not take with them for evacuation

This very severe experience moved the ministry to alter the law. The refugee principally should evacuate with their pets together and each local government should prepare for that.

Wada et al. (2017) has monitored radioactivity of animal refugee in a shelter in Fukushima frequently by animal whole-body counter. Concentration of ^{137}Cs at the time they caught was in the range of 10 to 10^7Bq/kg in dogs and 10^3 to 10^6Bq/kg in cats. And they concluded the biological average half-life of radiocesium was 54 days in dogs and 30 days in cats, and they discovered their biological half-lives prolongs from 20 to 70 days as the grew up. This whole-body counting system for small animal sounds valuable for long term monitoring of radioactivity in the body and most likely applicable to another small animals including sheep, goat and other wild life.

Team of the veterinary universities including Iwate, Kitasato, Tokyo and Miyazaki joined to collaborate for investigate if there is/are any biological effects of the low-dose radiation. This team tried to evaluate radiation exposure from environment and internal exposure via ingestion of grass. Since hundreds of cattle within 20km from FDNPP are kept alive because these farmers disagreed to the JG's decision to euthanize without any reasonable explanation. Farmers needed to know if there is/are any unwanted biological effects of the low dose radiation on cattle. Some cattle showed some white spot. The team also evaluated routine blood test,

ambient radiation dose rate, radioisotope analyses from biological samples and soils in the farm where cattle are kept.

The cumulative radiation dose in the farm was estimated at most 1.2 to 1.5 Gy (or Sv for human effective dose) for 7 years since the FDNPP accident happened. However blood test and biochemistry from cattle was normal range and showed no abnormality. Comet assay to detect some degree of DNA degradation

Sasaki et al reported that the major sickness or abnormality was sporadically observed. The major cattle diseases or abnormalities found in the evacuation zone was nine cases (3.7%) of enzootic bovine leukosis (EBL) and three cases (1.2%) of goiter were diagnosed. Estimated integrating dose of external exposure in EBL cases ranged from a maximum of 1200 mSv to a minimum of 72 mSv. There was no evidence of a radiation effect on pathological findings in any of these autopsy cases. This is ironical, since they were kept outside so long, most of them are now positive for bovine leukemia virus infection and found sick or dead due to the onset of leukemia. Geographic distribution of the soil contamination, ambient radiation dose rate and its time course will be also discussed.

Some investigations on wild lives were performed in non-mammalian species including monkeys, wild boars, fish insects and plants. In the initial observation demonstrated that there was decrease of white and red blood cell in blood of monkeys some extent, but so far extended decrease of the blood cell is not reported.

- What happened soon after the FDNPP accident...escape from evacuation zone and refugee
- How to rescue animals; A proposal to the government from animal/radiology experts
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- Investigations of wild lives' radiation exposure and cumulative radiobiological effects
- What we have learned from the nuclear accident and what should be done for the future prevention?

Apart from the initial radioactive iodine, the major source of radiation was radio-Cesium, namely ^{134}Cs and ^{137}Cs. Hopefully Strontium-90 was less than 1/1000 of radio-cesium and was negligible. To present knowledge, apart from observation of abnormalities found in pale glass blue butterfly, fish and birds, there is no report or observation on mammalian species those were suffered from acute or chronic radiation effects those include chromosomal or DNA damage, anomaly, and/or radiation induced tumor or cancer in Fukushima. However, accidents will happen. But forewarned is forearmed.

Prevention is better than cure. VMAT (Veterinary Medical Assistant Team on disaster) was created and its guideline was proposed in Fukuoka (2012) and in Gunma (2016). They also started training for veterinary experts. Therefore, you can never be too prepared before it starts. Concerning preparation of shelters and or VMAT, periodic intimate communications (and simulative trainings, if possible) are inevitable to keep good contact among veterinarians, assistants, supporting staffs local government and volunteers.

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IMMUNOTHERAPY: SUBCUTANEOUS VS. SUBLINGUAL

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Introduction

Alopecia is the loss or absence of hair on the body where hair is normally present. Hair growth in the dog follows the hair cycle, namely anagen (growing phase), catagen (intermediate phase) and telogen (resting phase). Hair is shed in the telogen phase as a new hair begins at the anagen phase.

Alopecia

The first step in determining the underlying cause(s) of alopecia is to make sure that the alopecia is not congenital i.e. from birth. This is characteristic for certain breeds such as Mexican Hairless Dog, Chinese Crested Dog and Peruvian Hairless Dog. It can also be abnormal in dogs affected by ectodermal dysplasia (has concurrent teeth abnormalities).

The next step is to differentiate if alopecia is due to self-trauma or spontaneous. Self-traumatic alopecia is usually due to pruritic causes e.g. allergies and will not be covered in this presentation. If you are unsure, a trichogram can assist in this differentiation i.e. broken hair tips suggestive of self-traumatic alopecia vs smooth tapered hair tips for non-pruritic alopecia.

Non-pruritic alopecia can be broadly divided into inflammatory vs non-inflammatory and symmetric vs non-symmetric alopecia. Inflammatory causes usually result in non-symmetric (focal to multifocal) alopecia. Conversely, non-inflammatory causes usually result in symmetric or diffuse alopecia.

Non-pruritic non-symmetrical inflammatory alopecia

Differential diagnoses	
Common causes	• Demodicosis • Dermatophytosis • Bacterial folliculitis (usually pruritic but can be subtle)
Less common causes	• Sebaceous adenitis • Ischemic dermatopathies e.g. dermatomyositis • Alopecia areata • Epitheliotropic lymphoma

Diagnostic work up

Signalment and history

Age
- Demodicosis and dermatophytosis typically affect young dogs
- Epitheliotrophic lymphoma most commonly in old dogs

Breed
- Sebaceous adenitis more common in Standard Poodles, Akita Inus, Vizslas and Hovawarts
- Dermatomyositis usually affects collies, Shetland Sheepdogs and crosses

History
- Concurrent muscle atrophy/weakness suggestive for dermatomyositis

Physical and dermatological examinations
- Comedones due to follicular plugging by demodex mites or sebaceous adenitis
- Follicular casts for sebaceous adenitis and demodex mites
- Excessive scaling suggestive for dermatophytes or sebaceous adenitis
- Papules, pustules and crusting for bacterial folliculitis

Diagnostic tests
- Wood's lamp to check for dermatophytes i.e. apple-green fluorescence, and to select hair for KOH and fungal culture
- Skin scrapes: both deep and superficial for demodex mites
- Cytology for bacterial pyoderma and demodex mites
- Trichogram: Follicular casts for sebaceous adenitis, demodex mites around hair shafts, fungal hyphae/arthrospores along and within hair shafts
- Tissue biopsies for histopathology is required diagnose sebaceous adenitis, ischemic dermatopathy, alopecia areata and epitheliotropic lymphoma

Non-pruritic symmetrical non-inflammatory alopecia

Differential diagnoses
Hormonal disorders
Hypothyroidism
Hyperadrenocorticism (spontaneous vs iatrogenic)
Sex hormone imbalances: (endogenous and exogenous)
Hair cycle disorders
Hair cycle arrest aka "Alopecia X"
Recurrent cyclic flank alopecia
Pattern baldness
Anagen/Telogen effluvium
Follicular dysplasia
Colour dilution alopecia
Black hair follicular dysplasia

Various hormones affect the hair cycle by stimulating

anagen or prolonging telogen. While hormonal disorders can result in non-pruritic symmetrical non-inflammatory alopecia, there are also many other causes.

Diagnostic work up

Signalment and history

Age
- Middle aged to older dogs for hormonal disorders
- Young dogs for follicular dysplasia and pattern baldness

Breed
- Hyperadrenocorticism: predisposed breeds include Miniature Poodles, Dachshunds and Boston Terriers
- Hypothyroidism: predisposed breeds include Boxer, Old English Sheepdogs and Golden Retrievers
- Plush coated breeds such as Pomeranian, Alaskan Malamute, Samoyed and Siberian Husky suggestive of hair cycle arrest
- Recurrent cyclic flank alopecia: predisposed breeds include Airedale, Boxers and Schnauzers.
- Colour dilution alopecia affect dilute (blue or fawn) coated breeds such as Staffordshire Bull Terriers and Dobermans
- Pattern baldness: predisposed breeds include Dachshunds and whippet

History
- Polyuria, polydipsia and polyphagia and history of exogenous corticosteroids for e.g. allergies suggestive of hyperadrenocorticism
- Weight gain and lethargy suggestive for hypothyroidism
- History of owner using topical estrogen hormonal replacement therapy OR use of estrogen for urinary incontinence, could indicate estradiol induced alopecia
- Feminisation behaviour in intact dogs e.g. nursing or attraction of male dogs to other males could indicate sex hormone dermatosis
- History of seasonal alopecia and regrowth could indicate recurrent cyclic flank alopecia
- Prior stressful events e.g. disease, surgery or pregnancy could suggest anagen (alopecia within days) or telogen (usually 1-3mths prior to alopecia) effluvium

Physical and dermatological examinations
- Thinning of skin, comedones, hepatomegaly, pendulous abdomen and calcinosis cutis suggestive of hyperadrenocorticism
- Overweight, bradycardia and facial myxedema suggestive of hypothyroidism
- Gynecomastia, pendulous prepuce, vulvar enlargement and/or preputial dermatosis suggestive of hyperestrogenism
- Well demarcated alopecia and hyperpigmentation affecting lateral flanks/thorax suggestive of recurrent cyclic flank alopecia
- Alopecia affecting only black coloured hair is suggestive for black hair follicular dysplasia

- Palpate testes for asymmetry suggestive of testicular tumour

Diagnostic tests
- Based on clinical suspicion to allow ranking of ddx and diagnostic steps
- Trichogram
- o Irregular pigment granules along hair shafts suggestive of colour dilution alopecia
- o All hair bulbs in telogen phase for endocrine alopecia
- Complete blood count, biochemistry, urinalysis and culture may provide clues but are not specific e.g.
- o Stress leukogram with elevated liver enzymes (especially ALP), hyposthenuria or isosthenuria for hyperadrenocorticism
- o Non regenerative anemia and elevated fasting cholesterol for hypothyroidism
- o Anemia, thrombocytopenia, leucocytosis or leucopenia due to myelosuppression by estrogen

Endocrine testing
- o Hypothyroidism e.g. fT4 (ED), TT4, TSH
- o Hyperadrenocorticism: Screening (e.g. LDDST, ACTH simulation test) and differentiating (e.g. LDDST, eACTH, abdominal ultrasound evaluating adrenal glands) tests
- o Baseline and post ACTH stimulation tests for sex hormones (estrogen levels may be normal due to overlap between healthy vs affected dogs)
- Typically if endocrine tests are negative/normal, the next step is to perform skin biopsies for histopathology and/or castration or ovariohysterectomy with histopathology of testes/ovaries.

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WSV18-0010

WHEN DO CATS WITH CHRONIC KIDNEY DISEASE NED PROTEIN RESTRICTION?

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INTRODUCTION

Cats with chronic kidney disease (CKD) frequently lose weight or have low body condition score (BCS). Although not yet shown in prospective studies, preservation of body weight and lean body mass (LBM) may enhance survival and quality of life in aging cats and those with CKD. Indeed, some loss of weight is part of normal aging, and is not caused by apparent illness. Nutrition offers the possibility to improve longevity as well as quality of life (QoL).

Sarcopenia, the age-related loss of lean body mass (LBM), is a gradual process: initially it is unapparent because increases in body fat persist, and may even cause increases in body weight. However, the loss of LBM has profound effects on survival. Studies have identified decreased survival associated with thin body condition.[1-3]

In one study of cats with large cell lymphoma, those that lost > 5% body weight during treatment had a significantly shorter survival time than those who were able to maintain their weight. [4] Patients with other cancers that had a slightly increased or ideal body condition score (BCS) survived up to six times longer than underweight cats.[5] In chronic kidney disease (CKD), cats with higher BCS lived longer than those with lower BCS.[6] Similarly, cats with heart failure in ideal or slightly greater BCS fared better compared to cats that were underweight or obese.[7]]

Is it possible to prevent cats with CKD from losing muscle and body condition? Is it possible to prevent cats with CKD from losing muscle and body condition? Several studies have shown a decreased ability to digest fat[8,9], protein[8,10], and micronutrients[11] in otherwise healthy aging cats. Because of this, a high energy, highly digestible diet with an increased protein content may be appropriate. Studies in healthy cats suggest that 5 – 6g protein/kg body weight is needed to maintain or enhance LBM.[12-13] One small, unpublished study in middle-aged cats with mild CKD showed they could maintain LBM over 30 weeks when fed only 6.5g protein/100 kcal.[14] However, it is unclear what the actual protein intake was, as no data on actual food intake or body weight was included (i.e., protein calories ingested). If cats are eating only small amounts of food, a higher percentage of calories from protein may be needed to meet their needs.

Old cats need protein. The majority of cats with CKD live a long time. Given the importance of protein in cats, how do we optimize the benefits of a renal diet but correct or prevent further muscle wasting?

CLINICAL IMPLICATIONS

Step 1: Evaluate

Just as every cat is different, the needs of different cats with CKD may differ. It is important to start by finding out exactly what, (foods, treats, supplements), and how much, a patient is eating. A simple diet history form can be found at: www.wsava.org/nutrition-toolkit. Evaluating body condition entails not just assigning a body condition score (BCS, Figure 1), but also getting a weight at every visit. Both body weight and BCS are subjective ways to evaluate caloric adequacy: weight reflects the current state and BCS, gives a longer term perspective.

Figure 1. Body Condition Score (from www.wsava.org/nutrition-toolkit => Body Condition Score Cart for Cats .pdf)

Calculating the percent weight change is an easy way to follow trends and get a better idea of weight relative to size. This important measure helps alert both the practitioner and the client to insipient (or blatant) physiologic alterations.

Loss of muscle can occur without fat loss or a decrease in BCS and individuals can retain an obese or overweight BCS yet be under muscled. Muscle condition scoring (MCS, Figure 2, Table 1) is a subjective way to evaluate LBM. Therefore, both subjective BCS and MCS should be performed in all cats, to evaluate fat mass and lean mass independently of each other.[17] When clients participate in determining BCS, it is more meaningful to them. Similarly, they can be taught to assess muscle condition.

Figure 2. Muscle condition score (from www.wsava.org/ nutrition-toolkit => muscle condition score chart for cats pdf)
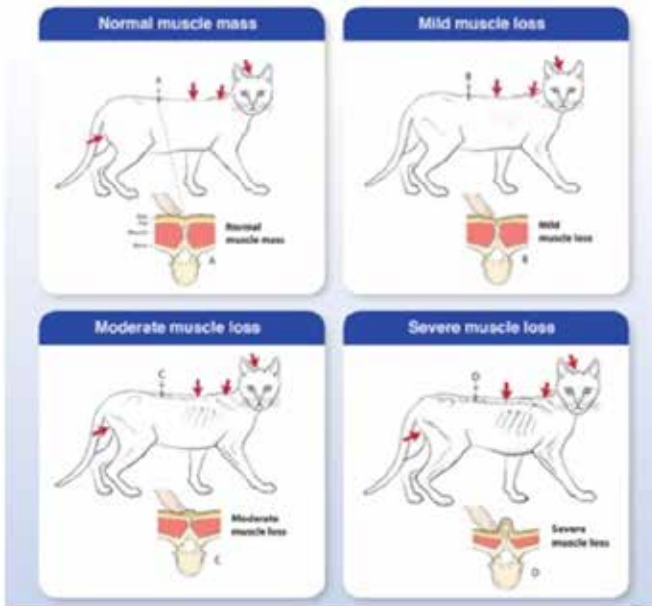
Table 1. Muscle condition scoring criteria [18]

Score	Assessment of Muscle Condition
0	On palpation over the spine, scapulae, skull, or wings of the ilia, muscle mass is severely wasted
1	On palpation over the spine, scapulae, skull, or wings of the ilia, muscle mass is moderately wasted
2	On palpation over the spine, scapulae, skull, or wings of the ilia, muscle mass is mildly wasted
3	On palpation over the spine, scapulae, skull, or wings of the ilia, muscle mass is normal

Older cats may have difficulty getting to food without discomfort; this is often due to arthritis, but loss of muscle mass and function can be a contributing factor. Weight loss can occur due to reduced access to food or reduced intake due to a decline in the ability to taste, smell or see food. Pain (commonly from oral disease) and nausea can interfere with eating. Nausea, associated with hypergastrinemia is believed to occur in some cats with CKD. Gastroprotectants or proton pump inhibitors may be beneficial depending on the cause of the gastric upset. Omeprazole has been shown to be superior to H2 antagonists (i.e., ranitidine) in reducing gastric acid production.19

If physical and laboratory evaluations fail to reveal a cause for the weight loss, before attributing this problem solely to old age or before considering medical intervention, the cat's environment should be considered. What may not be threatening to us, or even to the cat in the past, may be perceived as a source of stress in the older, less confident cat. A discussion about ease of access to all-important resources may find a crucial defect that may be readily corrected 20. A Household Resource Checklist may be downloaded from www.cliniciansbrief.com/sites/default/files/Applied%20 Behavior_Household%20Resource%20Checklist.pdf.

To encourage intake, multiple bowls of dry food should be located around the home in safe, quiet, private places. Moist food should be fed 2-4 times per day. Water should be fresh and, like food bowls, should also be in a variety of locations, and not adjacent to food bowls. Treats should be complete and balanced (e.g., SmartbitesTM). Bowls should be wide to allow whisker clearance (especially in a clinic setting). For some cats with cervical or coxofemoral degenerative joint disease or lumbosacral spondylosis, raising the bowl may be helpful. (Photo 1.)

Photo 1. A) Lumbosacral and coxofemoral changes make sitting down uncomfortable. B) Raising food and water bowls may help cats be more comfortable.

Step 2: Calculate

Previous weight, BCS and MCS may suggest the ideal body weight for the individual cat. There are several formulae available but a simpler "rule of thumb" is:

- Intact, active cats need 60-80 kcal/kg ideal weight/day

- Sexually altered, less active cats need 40-50 kcal/kg ideal weight/day

- Older cats (> 12 years) need 70 kcal/kg ideal weight/day.

Using the information regarding what the patient is currently eating, along with physical examination findings, (including hydration), provides a starting point for a nutritional plan. Both the weight loss and muscle wasting need to be addressed.

Caloric requirements should be calculated in order to determine the amount of food required). Ideally, this should be based on a good diet history with known intake as individual calorie requirements can differ greatly from calculated averages. If fed dry food, it should be available at all times, in quiet locations that are readily accessed by this individual. If a cat prefers to eat only a small quantity at a time, moist food may not be appropriate as it is less calorically dense than dry food. While moist food may be preferable in providing more moisture, some cats are reluctant to eat it.

When recommending a change in the diet, recommend that the client monitor closely the amount eaten and make the change gradually over 3 or more weeks. It is not imperative that the cat switches completely to the new food. Within reason, it is more important that they eat adequate amounts rather than what they eat.

EXAMPLE:

4.0 kg cat BCS 3/9, moderate muscle wasting who previously weighed 4.5 kg BCS 5/9 good muscle mass

At 70 kcal/kg/day, this cat needs 315 kcal/day for 4.5 kg ideal weight.

Protein requirement is at least 5 g/kg ideal weight/day: this cat needs > 23g protein/day

Example using a dry or canned renal diet

Let's say the dry formulation has 3936 kcal/kg (3.94 kcal/g) or 398kcal/8 oz cup can as fed.

It has 6.61 g protein/100 kcal

For a 4.5 kg cat who needs 315 kcal/day this equals 80g or 4/5th of a cup of this diet/day.

For 315 kcal @ 6.61g protein/100kcal there would be 21 g protein in the day's dry. This is less than the minimum of 23g/day needed to maintain muscle condition.

For the same diet, the canned has 193 kcal/5.5 oz can, therefore 315 kcal = 1.6 cans

It has 7.23g of protein/100kcal. For 315 kcal @ 7.23g protein/100kcal there would be 23 g protein in the day's canned food.

It is important that the cat has a good appetite and eats the entire quantity of dry or canned food. With lower calorie intake, protein intake will be deficient. A renal diet has benefits, (i.e. low phosphorus, buffering agents, +/- n-3 fatty acids, etc.), but should the response to the diet reveal inadequate muscle mass, then one needs to supplement the diet with protein or change the diet and use an intestinal phosphate binder, in order to both meet the cat's nutritional needs while still addressing the CKD.

Step 3: Communicate

Once the ideal body weight has been estimated, either based on historical information or on an educated guess, and the calories needed to maintain this ideal weight are calculated, it should be noted in the medical record. Regardless of diet chosen, the daily quantity of food (dry and or moist) should be calculated and recorded. The amount of food (grams, cups, cans) that the cat needs to eat must be conveyed to the client both verbally and in writing. Also, make sure that they understand that this is a starting point. For the sake of simplicity, and because the cat and the client may have preferences, the author likes to send home samples of each recommended diet for the cat to try. Once the cat has made their (initial) choice, the quantities can be calculated and given to the client. Do not try to introduce the new diet in the clinic: a vigilant cat is unlikely to try or like a new diet. Additionally, they may develop a diet aversion.

Step 4: Reassess

It is important to recheck patients 1-2 weeks after making nutritional recommendations to assess the effect of the food on the cat's condition. This is no different than reassessing the impact of other medical recommendations, (e.g., medications, fluids, etc.), or repeating laboratory tests. It is not uncommon that a patient's decline or lack of improvement is because of feeding factors rather than an inherent progression of their illness. Determine whether the cat is eating enough of the recommended food to result in an improvement in their weight, BCS and MCS. Find out how the client feels about the diet, feeding the diet, the cat's enjoyment and QoL on this diet. Providing nutrients may be the most challenging part of any therapeutic regimen, and recovery or attaining the best possible QoL in cats may depend on our ability to ensure optimal nutrition. Again, communication with, and motivation of, the client is crucial.

Step 5: Upping the ante

If body weight, condition and MCS are not improving, additional intervention is required. Verify that the diet appeals to the cat, taking into account its texture, aroma,

taste, and consistency. Confirm that the cat is able to hide when they feel the need and has a safe, private places to eat. Feline facial pheromone may be beneficial to reduce stress.20 Verify that inadequate food intake isn't due to other factors, such as dysphagia, pain (e.g., oral, dental, gastrointestinal, multisystemic, etc.), dislike of the diet (e.g., boredom, altered palatability, spoilage), aversion, fear (e.g., environmental changes including those in the social demographics). Palatability may be improved by bringing the temperature of moist food to body temperature and by changing the consistency of the diet.

Rehydration and correction of electrolyte imbalances is important but may have been overlooked or the need may have changed. Anti-emetics (e.g., maropitant, mirtazapine, dolasetron, ondansetron) have a place if the cat is vomiting.

Appetite stimulants including cyproheptadine (1 mg/cat PO BID), mirtazapine (1-2mg/cat PO q48h)21 may help to jump-start a cat's appetite, but it is important to keep track of total calories consumed. A cat eating small amounts of baby food will not meet their caloric needs: they need 2-3 jars/day. Meat baby food is not balanced, but is sufficient for several weeks. If a cat is eating, but not enough, supportive feeding (assisted syringe feeding or tube feeding) must be considered. There are several diets specifically designed for the assisted feeding of cats (e.g., Purina ® Pro Plan® Veterinary Diets CN Critical NutritionTM), liquid balanced enteral diets for cats. Additionally, we can make a slurry from any canned food; blend with a liquid feline diet rather than water to minimize loss of calories. If the weight is improving, but MCS is not, supplementing with 1oz (28g) of cooked chicken/day may be a low phosphorus protein option.

If the cat is reluctant to eat the renal diet or the MCS is not improving, consider feeding the diet that cat prefers and utilizing intestinal phosphate binders. Aluminum hydroxide dry gel powder (USP) is well accepted by most cats and can be mixed directly into moist food or added to dry. (Put the AlOH dry gel powder and dry food together in a plastic bag, shake.) (www.zzcat.com/CRF/supplies/binders.htm)

Step 6: Supportive feeding

There are several options for assisted feeding each with advantages and disadvantages. Syringe feeding, nasogastric (NG), esophagostomy or gastrotomy tubes are the most common choices. In general, the author starts with syringe assisted feeding until the cat is stable enough to allow the brief anaesthetic required for the placement of an esophageal tube. Syringe feeding can be very successful bearing a few things in mind. Because the oral capacity of a cat is only ½-1 ml, small volume syringes should be used being sure to provide the entire caloric dose. Like an NG tube, syringe feeding is suitable

only for the short term. A large bore feeding tube should be considered early and as a temporary measure to improve the nutritional plane (BCS, MCS) of the patient with kidney disease.

In cats with concurrent liver disease, three doses of Vitamin K1 (1.0 mg/kg q12h SC) should be given prior to tube placement, biopsies or any other procedure that might result in bleeding. Placement of esophageal tubes is not complicated and details are discussed elsewhere. (An example is listed in Resource below.) Instrumentation is very basic requiring only the following: 14-16 Fr red rubber feeding tube/urinary catheter, long curved forceps, a scalpel blade, suture and bandaging materials (or a KittyKollarTM) and a multiple use injection port ("prn adaptor").

Calculating how much to feed requires that you know the calories they need to maintain their ideal, healthy weight as well as the caloric densities (kcal/ml) of the diet you are intending to use (see **Table 2**). Start by feeding 1/3 of the calories needed, on day two, feed 2/3, and on day three, feed the full calories needed for the ideal weight.

Table 2: Caloric densities of convalescent diets, for calculating feeding volumes:

ClinicareTM: 1 kcal/ml

Royal Canin/MediCal RecoveryTM: 1.04 kcal/ml

Hill's a/dTM: 1.17 kcal/ml

Purina PPVD CNTM: 1.33 kcal/ml

Blending a renal (or any other) diet with a liquid diet will provide a higher caloric density than if water is used.

EXAMPLE:

4.0kg sick cat BCS 3/9, healthy weight 4.5kg BCS 5/9

Using the calculator, 216 kcal by day 3

315kcal = 302 ml of Royal Canin Recovery ™;

269 ml of Hill's a/d ™;

236 ml of Purina PPVD CN ™

Example, using PPVD CN, the most calorically dense:

Day 1 feed 80 ml

Day 2 feed 160 ml

Day 3 feed 236 ml

With surgically placed tubes there is a delay in how quickly one can start to use them; with an esophageal tube only a 2-3 hour delay is required to ensure full recovery from anaesthesia. Cats can eat with the tube in place although it is recommended to avoid offering food for the first week to reduce the likelihood of them developing an aversion to the food offered. Once a cat is eating well with tube in place the question becomes when one can remove the tube. Weigh the cat and,

as long as they are eating well, avoid using the tube (medications are okay) for a week then reweigh them. If the weight is stable (or increased), then it is safe to remove the tube. Because of stoma formation, removal does not require anaesthesia. Remove the suture (purse-string or stay sutures) and pull the tube out. Suturing is not required for the skin opening. Cleanse minimal serous discharge that may occur for 2-3 days.

Feeding frequency: the number of feedings per day, (and hence intervals), is determined based on the volume of food tolerated per feeding. Start with 6 ml and increase by 6 ml increments to about 36-48/feeding for most cats. In the uncommon case of the patient who cannot tolerate even 6 ml boluses despite antiemetic therapy, trickle feeding may be instituted. Trickle feeding is a technique in which liquefied food is syringed into an empty fluid bag and administered gravitationally or by pump assistance via an intravenous line attached to the large bore feeding tube or by use of a large syringe filled with food and syringe pump. Renew food and delivery tubing and syringe at 12-hour intervals to avoid bacterial contamination. A promotility agent may be warranted as well.

The success of assisted feeding is measured objectively by weight gain. Subjective measures will include improved coat quality, increased energy, muscle recovery and innumerable other effects that the client will appreciate. An improved QoL is the goal whether recovery from the underlying problem is possible or not.

SUMMARY

Renal diets provide benefit for cats with CKD. It is imperative that the cats eat enough calories and protein to optimize their body and muscle condition scores. Each patient must be assessed individually and monitored. This requires client communication and recheck visits. Cats with chronic kidney disease with higher BCS will live longer, and have an improved quality of life.

USEFUL RESOURCE

Hodshon B, Tobias K. Esophagostomy Feeding Tubes, Clinicians Brief February 2014 (www.cliniciansbrief.com => esophagostomy feeding tubes)

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WSV18-0275

OPHTHALMOLOGY

MY APPROACH TO NONHEALING CORNEAL ULCER

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INTRODUCTORY PHILOSOPHY

When an ulcer hasn't healed at the first recheck, there is a tendency to throw up our arms and become frustrated. However, ulcers within this group have actually helped us by identifying themselves as "complicated ulcers" with one of only 3 causes possible in dogs and one of only 2 causes possible in cats.

REASONS FELINE AND CANINE ULCERS DO NOT HEAL

Based upon their clinical appearance including their fluorescein staining pattern, nonhealing ulcers can be defined as likely due to one of three causes in dogs:

- The primary cause is still present
- It is an indolent ulcer (also known as Boxer ulcers or superficial chronic cornea epithelial defects – SC-CEDs)
- It has become bacterially infected

The thought process is even simpler for cats. Because cats do not get SCCEDs, there are only 2 reasons an ulcer has not healed in cats – the primary cause is still present (and feline corneal ulcers are considered to be due to feline herpesvirus (FHV-1) until proven otherwise), or the ulcer has become bacterially infected.

Fortunately, each of the ulcer complications has a characteristic appearance:

- Ulcers in which the primary cause is still present typically appear like simple ulcers but remain chronic. That is, they don't necessarily worsen; they just don't heal. This should stimulate a detailed search for all of the known causes of ulcers (**Figure 1**)
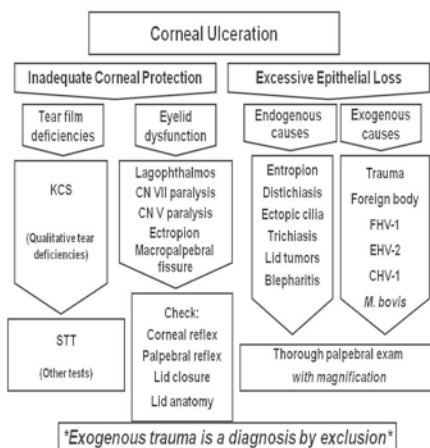
- Indolent ulcers as defined by the failure of epithelium to adhere to stroma due to a primary adhesion defect are seen in dogs only, typically boxer dogs or corgis of any age or older dogs of any breed. By definition, they are superficial, uninfected, chronic (or will become chronic), and have a lip of redundant non-healing corneal epithelium that is easily debrided with a cotton-tipped applicator (CTA). This lip often produces a characteristic "halo" fluorescein staining-pattern due to leakage of stain under the non-adherent lip. They arise from a failure of replicating and migrating epithelium to complete the final step in healing – to adhere to the underlying stroma via the epithelial basement membrane. Diagnosis is reliant on characteristic signalment, chronicity, appearance and staining-pattern of the ulcer, as well as the ease with which the epithelium is manually debrided with a CTA.

- Bacterially infected ulcers have one or more of 3 features in any combination - stromal loss (i.e., the ulcer is deep), corneal malacia (or "melting"), and/or infiltration of the stroma with white blood cells (which turns the stroma yellow-green).

Using these guidelines, **Figure 2** outlines an algorithmic approach to nonhealing ulcers in dogs and cats.

Figure 2.

Figure 1.
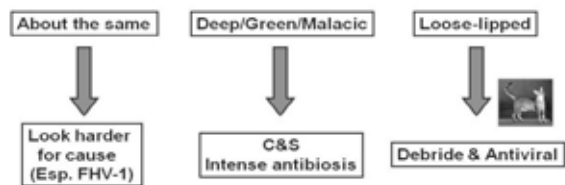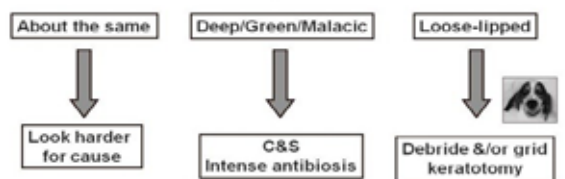
WSV18-0190

ENDOCRINE HYPERTENSION

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ENDOCRINE HYPERTENSION

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Systemic hypertension has been recognised with increasing frequency in small animal practice over the past 20 years. Although there is still some debate regarding optimal cut-off values, risk categories suggested within the ACVIM consensus statement have been widely adopted (Table 1)[1].

Risk category	SBP	DBP	Risk of end organ damage
I	<150	<95	Minimal
II	150-159	95-99	Mild
III	160-179	100-119	Moderate
IV	>180	>120	Severe

Table 1: Risk categories associated with systemic hypertension in dogs and cats (1).Consequences of uncontrolled hypertension can be severe. End-organ damage most commonly involves the eye, brain, kidney or heart and consequences include blindness, cerebrovascular events, hypertensive encephalopathy, myocardial hypertrophy and progressive kidney disease. Whilst hypertension can be controlled medically, assessment for the underlying cause is critical.There are numerous potential causes of hypertension including kidney disease, obesity, pain, stress and certain drugs. Essential hypertension is less clearly defined in dogs or cats. Other causes primarily relate to the endocrine system and include hypo and hyperthyroidism, hyperadrenocorticism, phaeochromocytoma, primary hyperaldosteronism and diabetes mellitus.Hypertension has been reported in from 5 to 22 % of hyperthyroid cats. In most cases the hypertension is mild and probably stress related. If severe and associated with clinical consequences, other causes of hypertension, such as renal disease, should be investigated. Although suggested to occur in hypothyroidism, evidence is limited.Hyperadrenocorticism is commonly associated with mild to severe hypertension in dogs. However,

despite this, clinical signs associated with hypertension are rarely reported[1]. As such dogs are not usually presented primarily with hypertension, but rather the clinical signs of hyperadrenocorticism predominate.

Severe hypertension is more often a feature of phaeochromocytoma in dogs. Given that this may often be the sole presenting complaint, other than vague and non-specific signs, investigation for phaeochromocytoma is indicated in all older animals presenting with hypertension. Demonstration of an adrenal mass together with increased urinary normetanephrine to creatinine ratio or plasma free metanephrine concentration appear the most promising for its diagnosis[2].

Primary hyperaldosteronism (Conn's syndrome) is caused by the excessive autonomous secretion of mineralocorticoids. It is rare in dogs and may be an underdiagnosed condition in cats. Most cases are due to the presence of an adrenocortical carcinoma or adenoma although adrenal hyperplasia has also been described. Clinical signs relate to systemic hypertension, hypokalaemia, or both. Muscle weakness, including cervical ventroflexion, and sudden onset blindness are most commonly reported. Adrenal ultrasonography will reveal an adrenal mass in most cases and circulating aldosterone concentrations, either alone or after ACTH stimulation, are high. Although demonstration of decreased renin activity is required for definitive diagnosis, this assay is not widely available. The identification of an adrenal mass and increased aldosterone concentration in an animal with consistent clinical signs is considered sufficient to confirm the diagnosis.

Hypertension is common in humans with diabetes mellitus and is considered one of the most important co-morbidities. There are variable reports regarding the prevalence of hypertension in dogs with diabetes mellitus. Up to 50 % have been shown to be hypertensive. However, associated clinical features are rarely reported. Hypertension in diabetic cats is uncommon.

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UROLOGY

LOWER URINARY TRACT DISORDERS IN BOXER DOGS

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Lower urinary tract disorders in Boxer dogs

Is there an association between urinary tract infection in young boxer dogs and early chronic renal failure?

Chronic kidney disease occurring in young Boxer dogs was first considered to be renal dysplasia (Lucke et al 1980). However later histologic examinations of kidneys of young dogs including one Boxer dog suffering from chronic kidney disease reviled lesions that did not have all the typical features of renal dysplasia (Peeters et al 2000). In a subsequent study of 37 Boxer dogs with juvenile nephropathy, the histologic picture was mostly not consistent with renal dysplasia (Chandler et al 2007). The nephropathy occurred at the age of 4 months to seven years. Twenty-nine of the dogs were female and eight were male. It was discussed if the lesions seen were caused by one disease or by different diseases. Interestingly urine culture was positive in 30% of the dogs. Even later seven Boxer dogs were described with renal lesions with a similar appearance as those of people suffering from vesico-ureteral reflux causing a so called reflux nephropathy (Kolbjornsen et al 2008). The kidney lesions were compatible with chronic pyelonephritis with severe cortical atrophy and fibrosis. Six of the seven dogs were related. If the dogs really suffered from vesico-ureteral reflux is not clear. Of young children with urinary tract infections about 30–45% suffer from vesico-ureteral reflux (Lellig et al 2017). If there is a relation between the early onset of kidney disease in boxer dogs and vesico-ureteral reflux or urinary tract infection is not known; however urinary tract infections in young female Boxer dogs seem to be common.

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WSV18-0117

ORTHOPEDIC SURGERY

LONG BONE FRACTURES: LEARNING SPLINTS & CASTS FROM PRACTICAL CASE EXAMPLES

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Casts and splints are available in most primary care veterinary practices and can be used successfully to treat selected fractures in dogs and cats. Unfortunately, when used improperly or in contraindicated scenarios, these fixation methods frequently cause severe complications including fracture disease, quadriceps contracture, mal-union, delayed or non-union, and/or infection; any of which can lead to limb amputation or euthanasia. Unexpected complications can negate their apparent affordability and, thus, lead to client dissatisfaction. Therefore, the key to successful treatment using these fixation modalities is a thorough knowledge of their indications, proper application and limitations.

Splints & Casts for Fracture Fixation

Coaptation includes both splinting and casting techniques. Neither splints nor casts provide rigid fixation, but cylindrical casts provide greater fracture zone immobilization than splints. Specifically, one must consider the ability of any fixation method to resist the disruptive forces acting upon the fracture to be treated. Disruptive forces to consider include bending, rotation, axial compression (axial collapse) and tension. We will consider each of these disruptive forces individually.

Control of Bending with Casts

All long bone fractures are subjected to bending due, in part, to the irregular shape of bones and the inherent eccentric loading at the joint surfaces and sites of muscular attachment. In order to control bending, casts must bridge a joint above and below the fracture. Since the patient's body wall prevents effective cast bridging of the hip and shoulder, casts cannot be used effectively for fractures above the knee or elbow. In theory, the most rigid cast stabilization would be imparted by direct application of the cast to the bone; this, of course, cannot be performed because of the surrounding soft tissues. Nonetheless, using this mechanical principle, one can readily appreciate that anything that increases the distance between the bone and the cast (muscle mass, soft tissue swelling, excessive cast padding, improper cast molding, etc) reduces its ability to control disruptive bending forces. Cast padding is necessary to protect from soft tissue injury over bony prominences, but it is important to use no more cast padding than is necessary for this purpose.

Control of Rotation with Casts

All long bone fractures are subjected to some rotational forces (properly referred to as "rotational moments"). These rotational moments are due, in part, to eccentric insertions of muscle-tendon units (MTU's). If you imagine application of a cast to a cylindrical pipe, you recognize that it would have no ability to resist rotation. Fortunately, the limbs of our patients have some bony prominences and joint angulation with which the cast can interact; these prominences and contours give casts their limited ability to resist rotational forces. The pelvic limb has more inherent prominences (tuber calcis) and angulation (tarsus) than the thoracic limb. It is, therefore, advisable to be certain to contour thoracic limb casts to the region of the olecranon as well as to place a small amount of flexion in the elbow and carpal joints.

Control of Axial Compression (Axial Collapse) with Casts

Gravitational force acting upon the body is countered by a ground reaction force. These opposed forces create axial collapse of long bones when there is complete fracture. Casts do not sufficiently interact with each bony segment to prevent axial collapse. Therefore, cast fixation is dependent upon bony architecture to resist axial collapse. The 3 main ways that bony structure can prevent axial collapse are: (1) incomplete fracture (also called a "greenstick" fracture), (2) fracture of only 1 bone in a 2-bone system (ex. an intact fibula adjacent to a tibia fracture, and (3) appropriate reduction of a transverse fracture. As a rule of thumb, 50% reduction of transverse fractures in the worst of two orthogonal radiographic views is acceptable in order to resist axial collapse.

Control of Tension with Casts

Pure tensile forces originate from the insertion of MTU's on traction apophyses. These traction apophyses ((humerus – greater tubercle; ulna – olecranon; femur – greater trochanter; tibia – tibial tubercle; tarsus – tuber calcis) are nothing more than levers and the muscles are motors that make the skeleton move through space. Casts have ZERO ability to resist these pure tensile forces. Thus, casts are unable to effectively treat fractures of traction apophyses. In most instances, either a figure of 8 tension band or a tension band plate will be required for adequate treatment of these fractures.

Other Relevant Factors for Splints & Casts

Articular fractures require rigid stabilization and perfect anatomic reduction; therefore, coaptation is not recommended for treatment of articular fractures (including most fractures of carpal and tarsal bones). Coaptation is prone to cause skin irritation and soft tissue wounds when used long term or in instances of poor patient / pet owner compliance. Similarly, external coaptation is prone to cause joint stiffness / immobility when used long term because it restricts normal limb use. I am cautious with the use of coaptation in puppies during early skeletal development (< 4-5 months of age) because abnormal limb use in the cast can contribute to skeletal developmental abnormalities such as patellar luxation, hip dysplasia, etc. When used in very young puppies, I strive to place the limb in a functional position (ie, pelvic limb has normal knee and tarsal angles rather than full extension) and I remove the cast at the earliest sign of functional radiographic healing (as early as 2-3 weeks in many instances). Alternatively, even though the fracture will heal with coaptation, I often opt for a surgical fixation that will permit immediate restoration of normal limb use (minimally invasive techniques) in these developing animals. Coaptation is best avoided in the treatment of distal fractures of the radius/ulna, especially in toy breed dogs, as it commonly results in nonunion; surgical fixation with a bone plate or ESF is favored for most of these fractures. Malunion in valgus and/or external rotation often occurs with coaptation because direction of the direction of the traction force applied upon the tape stirrups during cast application; if the team is aware of this tendency, these mal-alignments can usually be prevented. Though coaptation often seems to be the most economical means of fracture treatment, repeated recheck exams, cast changes with sedation, and complications often minimize this advantage except when applied to the most predictable of scenarios.

Splints & Casts Summary

Effective use of coaptation is restricted to a relatively narrow spectrum of long bone fractures encountered in veterinary small animal practice. Coaptation is best avoided in the following scenarios:

- Fractures above the elbow or knee
- Fractures of traction apophyses (such as tibial tuberosity, tuber calcis, olecranon, etc)
- Articular fractures including most carpal and tarsal bone fractures
- Fractures other than reducible/transverse fractures, greenstick fractures or fractures with an intact adjacent bone.
- Cases at risk of poor patient/pet owner compliance and/or slow healing.

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MANAGEMENT OF HEAT STROKE IN THE DOG

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Heatstroke is caused by the inability to dissipate accumulated heat. In dogs it is characterized by core temperatures above 105.8°F (41ºC) with CNS dysfunction. It results from exposure to a hot and humid environment or from strenuous physical exercise. Activation of inflammatory and haemostatic pathways initiates a systemic inflammatory response syndrome (SIRS) which often progresses to multi-organ dysfunction syndrome (MODS). Serious complications of heatstroke include rhabdomyolysis, acute kidney injury (AKI), acute respiratory distress syndrome (ARDS), and disseminated intravascular coagulation (DIC). Several factors are associated with the risk of developing heatstroke. These include prior occurrence of heatstroke or heat stress, obesity, breed (brachiocephalic, Golden and Labrador retrievers), body weight (>15kg), high environmental temperature and humidity, and lack of acclimation and fitness. Prior heatstroke may affect the thermoregulatory center in the preoptic zone which is responsible for heat sensation and dissipation. Excess body fat increases the body's natural thermal isolation and impairs normal heat dissipation mechanisms in obese people and animals. Large breed dogs are significantly more at risk of developing heatstroke, particularly exertional heatstroke, suggesting that the ratio between body size and surface area is an important factor of heat dissipation during heat stress. The median body weight of 54 dogs with naturally occurring heatstroke was 31 kg, also -supporting this theory. The most common clinical signs of canine heatstroke include collapse, shock, tachypnea, spontaneous bleeding (e.g. petechie, hematemesis, and hematochezia), disorientation/stupor, coma and seizures. Although the definition of heatstroke is based on hyperthermia causing shock and hypotension, it is important to remember that patients can be hyper-, normo- or hypothermic on presentation, particularly if cooling measures were initiated by the owners prior to presentation. Furthermore, in a retrospective study of canine heat related illness, hypothermia upon admission was a poor prognostic indicator. High body temperatures initiate a myriad of inflammatory, coagulation and tissue damage processes, varying in severity and progression between dogs. Thermal endothelial cell injury leads to diffuse vascular damage and initiation of coagulation and subsequent microvascular thrombosis. In addition, multi-organ cellular necrosis further stimulates the coagulation system and results in DIC, an important factor in the morbidity and mortality of heatstroke patients. The injured endothelium releases thromboplastin and factor XII, which activates coagulation and the complement cascade, inducing SIRS and widespread DIC. Hepatic injury and failure due to hypoperfusion, microembolism and direct hyperthermic damage may exacerbate the haemostatic disorders. In vitro studies have shown that high temperatures (>42°C) lead to platelet aggregation, activation of the coagulation cascade and enhanced fibrinolysis. Normalization of body temperatures inhibit fibrinolysis but not the coagulation cascade or platelet aggregation. In a retrospective study of 54 dogs with naturally-occurring heatstroke, 50% were diagnosed with DIC. In 11 such dogs, severe bleeding and widespread microthrombosis, characteristic of hemorrhagic diathesis were invariably noted at necropsy. As DIC may appear hours to days after the initial hyperthermic insult, dogs with heatstroke should be monitored closely for coagulation abnormalities and clinical signs of DIC for at least 24 hours after the insult. In a recent study in which serial monitoring of coagulation parameters were followed during the first 36 hours of hospitalization in 30 dogs with heatstroke, haemostatic analytes at presentation were not associated with mortality. However prolonged PT and aPTT at 12-24 hours post presentation, lower total protein C activity at 12 hrs and hyperfibrinogenemia at 24 hrs post presentation were significantly associated with mortality. Increased D-dimer concentration and low anti-thrombin activity were common at all time-points, but were not associated with mortality (Figure 1) (Bruchim and Kelmeret al, JVECCS 2016). Interestingly, in that study, which was performed 10 years following the first one at the same institution, DIC was not associated with mortality, the median number of fresh-frozen plasma units administered increased from 2 to 4 units per dog and mortality decreased from 50 to 40%.

Figure 1: Trends in haemostatic parameters throughout hospitalization in 30 dogs with naturally-occurring heatstroke with survivors (n=18) depicted in black and non-survivors depicted in gray. * depicts significant difference between survivors and non survivors. PT — prothrombin time, aPTT — activated thromboplastin time.
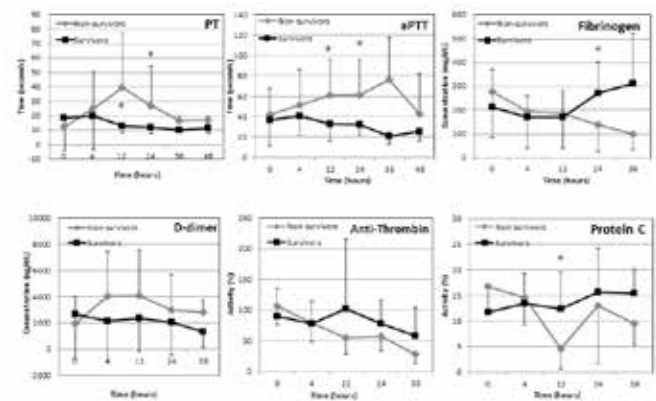
Heatstroke complications

Azotemia is a common finding in patients with heatstroke. It results from pre-renal and renal mechanisms, such as severe hypovolemia and direct renal tissue damage leading to tubular necrosis, a frequent finding at necropsy of dogs with heatstroke. Acute kidney injury represents a spectrum of conditions associated with sudden onset of renal parenchymal injury. Heatstroke-associated AKI is likely multifactorial. Such factors include decreased kidney perfusion due to dehydration and hypovolemia, direct thermal injury, myoglobulinemia due to rhadomyolysis, DIC, endotoxemia and the systemic inflammatory syndrome. Kidney injury may be mild and go unnoticed, but often, failure of the kidneys to meet the excretory, metabolic, and endocrine demands of the body ensues. Kidney function parameters can be classified to those measured routinely (e.g., serum creatinine), those not measured routinely, but are calculated using routine chemistry (e.g., glomerular filtration rate [GFR] using endogenous creatinine clearance and fractional electrolyte excretion) and unique urinary renal biomarkers, including neutrophil gelatinase-associated lipocalin (NGAL), C-reactive protein (CRP) and retinol-binding protein (RBP). NGAL is a 25 kD protein, covalently bound to neutrophil gelatinase. Normally, its expression and concentration are low; however, its expression is markedly induced when renal tubular epithelial injury occurs, and it is now identified as one of the earliest and most robustly induced proteins in both human and animal patients. RBP is a low molecular weight protein, freely filtered and is completely reabsorbed by renal epithelial tubules, rendering it a marker of renal tubular function. C-reactive protein (CRP), on the other hand, is a relatively high molecular weight protein, and therefore is normally not filtered through the glomerulus. Thus, its urine concentration reflects changes in glomerular capillary permselectivity characteristics. In this study we have found that renal biomarker analysis, GFR and sodium fractional excretion can identify kidney damage earlier, immediately at presentation and therefore aid the clinicians in the overall assessment of the animal. Nevertheless, the presence of AKI based on the traditional parameters during hospitalization of serum creatinine levels >1.5 mg/dL at 12 and 24 hours after presentation were found to be independent risk factors for death in dogs with heatstroke. Therefore, careful monitoring of renal function and early intervention are warranted.

Severe hyperthermia may lead to cerebral hypoperfusion, neuronal necrosis, direct vascular damage, cerebral edema, hemorrhage and multifocal vascular thrombosis with tissue infarction that may lead to CNS dysfunction and death. The canine brain is considered more resistant to thermal injury compared to the human brain and other physiological factors, such as respiratory alkalosis, shock and hypoglycemia, may play a more significant role in the observed CNS clinical signs in canine heatstroke. Thermal and biochemical injury to the pulmonary endothelium may lead to non-cardiogenic pulmonary edema, also known as ARDS. Histopathologic lung lesions in dogs suffering from heatstroke include pulmonary infarcts, marked alveolar hemorrhage or edema.

A few extra-cardiac mechanisms were proposed as contributing processes to the development of cardiac arrhythmias. These included myocardial hypoperfusion, lactic acidosis and electrolyte imbalance and possibly direct thermal injury. Post mortem findings in 11 dogs with heatstroke showed mild to severe subendocardial, myocardial and epicardial hemorrhages and hyperemia in all dogs.[4] These findings suggest that DIC has a pivotal role in the pathogenesis of the reported cardiac arrhythmias. Antiarrhythmic therapy should be considered, however, only if the patient has related clinical signs.

In humans and experimental studies, marked increases in core temperatures are associated with blood flow redistribution, which is characterized by coetaneous vasodilatation that occurs at the expense of decreased intestinal blood flow. This splanchnic vasoconstriction may cause ischemia and limit local vascular heat exchange, thereby promoting bowel tissue hyperthermia. Both intestinal ischemia and hyperthermia may promote oxidative stress that stimulates cytoskeleton relaxation, thus contributing to the opening of tight junctions and/or injuries to the epithelium. These morphological and functional changes enhance intestinal permeability, thus facilitating the translocation of bacteria and endotoxins that are normally contained within the intestinal lumen, and subsequently worsening a systemic inflammatory response syndrome that may culminate in multi-organ system failure and death. Gastrointestinal bacterial translocation has not been specifically documented in dogs with naturally-occurring heatstroke; however given the massive hemorrhagic diarrhea and hematemesis that rapidly ensues in dogs with severe heatstroke, it is reasonable to assume that it is a major contributing factor to SIRS, sepsis and MODS that may occur in severe cases.

In summary, clinicopathological findings in canine heatstroke are mainly related to the primary thermal insult; however, secondary deterioration occurs due to dehydration, shock and a poor perfusion to the tissues. Thus, early diagnosis and intervention are crucial to prevent further multi-organ dysfunction and exacerbation of coagulation abnormalities. Time lag from insult to admission (>1.5 hrs) was a crucial factor for survival in canines suffering from heatstroke.

Treatment options

Cooling - whole body cooling prior to admission is highly recommended. The literature provides different cooling methods (e.g, cold enema, gastric lavage and ice baths); however, other successful and perhaps more practical methods use evaporative cooling via whole body irrigation with tap water and placement of a fan facing the animal. Animals with thick undercoats may benefit from shaving prior to wetting. A cool environment with low humidity is also beneficial. Cooling with ice directly on body surfaces and/or peripheral blood vessels should be avoided as it may result in cutaneous vasoconstriction and decrease heat loss ability. During cooling, the patient's temperature should be monitored every 5-15 minutes to avoid hypothermia. Cooling should be terminated when body temperature has reached 39.5°C (103°F). Cooling does not result in suppression of the inflammatory response, but will prevent further cellular destruction. Most canine heatstroke victims suffer from distributive shock, as described above. Although the absolute intravascular volume has not changed significantly, vasodilatation and venous pooling of blood lead to a relative hypovolemia. As the animal is cooled, the vasomotor tone will return to normal. Therefore, judicious fluid therapy is warranted. An initial crystalloid dose of 10-20 ml/kg should be administered and perfusion parameters (HR, MM, CRT, pulse quality, blood pressure, mentation and urine output) continuously reassessed to help guide fluid additional fluid therapy. When perfusion cannot be restored with crystalloids alone, synthetic colloids (hydroxyethyl starch solutions), vasopressor agents (dopamine, vasopressin and norepinephrine) and positive inotropes (dobutamine) should be considered.

Dextrose should be administered to hypoglycemic dogs as a single bolus (1ml/kg of diluted 50% dextrose not to exceed a maximum of 10 ml) followed by a 2.5-5% dextrose CRI, with close monitoring of the glucose concentrations. All dogs with heatstroke should be given oxygen therapy during triage. Animals with severe dyspnea or laryngeal edema should be intubated, although this can decrease self-cooling mechanisms inherent with panting. In the most severe cases, general anesthesia with 100% oxygen or positive pressure ventilation may be required. Mannitol therapy may be beneficial in animals with cerebral edema causing intracranial hypertension, although it can also worsen cerebral hemorrhage, if present. Mannitol administration has beneficial effects on the kidney and will help restore urine output and flush tubular casts out in animals with AKI.A suggested treatment regime might include 0.5-1 gm/kg of mannitol over 10-20 minutes after the initial fluid resuscitation, followed by 1-2 additional boluses over the ensuing 12 hours. Benzodiazepines (diazepam, midazolam) are administered as a bolus followed by a CRIif the animal seizures (about 33% of the cases). Other causes for seizures such as hypoglycemia or metabolic and electrolyte imbalances should be ruled-out.

Antimicrobial treatment is not warranted in mild to moderate cases. In severe cases, broad spectrum antibiotics are indicated to treat sepsis due to presumed gastrointestinal bacterial translocation. A combination of antimicrobials effective against gram positive, gram negative and anaerobic bacteria is recommended in severe cases utilizing the "escalation-de-escalation" method. A combination of a potentiated penicillin and a fluoroquinolone or a third generation cephalosporin could be considered. Gastric protectants such as H_2 blockers (e.g famotidine) or proton pump inhibitors (pantoprazole) should be administered to prevent further gastric damage. Antiemetics and promotility agents are essential for prevention of vomiting and consequent aspiration pneumonia. If urine output remains insufficient despite adequate fluid replacement and means arterial blood pressure is > 60 mmHg, medical therapy with furosemide and/or mannitol should be considered. Overhydration must be avoided in anuric/oliguric patients and fluid therapy adjusted based on urine output and intravascular volume status of the patient. Hemodialysis may be indicated in dogs with oligoanuria despite medical therapy, as well as those patients with severe overhydration, uremia or electrolyte derangements. The treatment of the hemostatic abnormalities due to DIC is based on stabilization of the coagulation system with fresh frozen plasma and concurrent prevention of thrombosis with anticoagulants. Hemofiltration has been suggested as an effective treatment modality in an experimental model of severe canine heatstroke, causing early clearance of accumulated serum cytokines, creatinine and BUN. Clinical data is unavailable at this time.

Serial monitoring of the patient's clinical and clinicopathological parameters is essential for early identification of complications and appropriate intervention. Continuous monitoring of vital signs, including temperature, femoral pulse rate and quality and capillary refill time to assess perfusion, hydration and shock status, is warranted. In addition, PCV/TS, serum glucose, coagulation profile (including TEG or ROTEM when available), CBC, lactate, blood gas (arterial or venous), arterial blood pressure, and urine output should be monitored. The mental status of the patient should be evaluated frequently and continuous ECG monitoring is recommended as arrhythmias may develop during the first 24 hrs after the heatstroke occurs.

Mortality rates in dogs suffering from severe heatstroke are reportedly between 40-50%. Animals with heat-induced illness have a reportedly lower mortality rate (35%). At the author's institution, mortality rates decreased to 40 and 43% in 2 recent studies (Bruchim et al. 2016, Cell stress chaperon, Segev et al. Veterinary

J, 2015) as compared to 50% 10 years earlier (Bruchim et al. 2006); however larger scale studies are needed to determine if this trend is real.

In conclusion, heatstroke in dogs is a life-threatening condition, resulting in serious secondary complications such as DIC, AKI and ARDS, and a high mortality rate despite appropriate treatment. Early admission and treatment along with whole body cooling by the owners and caregivers are important for survival. The diagnosis of canine heatstroke should not rely exclusively on hyperthermia or the presence of neurological abnormalities upon admission, but should be based on the combination of the history, clinical signs and laboratory results. Treatment and monitoring should be intensive and prolonged since complications can have a delayed onset and present serious risk factors for mortality.

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Keyword: AKI, canine, CRP, DIC, NGAL.

WSV18-0291

HOW I TREAT WOUNDS TO THE HEAD

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Anatomy

The companion animal skin consists of two main layers. The general composition of the outer layer, the epidermis, is of avascular keratinised stratified squamous epithelium. The thicker vascular dermis lies underneath the epidermis and consists of tough fibroelastic tissue with a supportive and nourishing function. The dermis rests on a layer of loose connective tissue known as the subcutis or hypodermis, composed of adipose tissue, the cutaneous trunci muscle (where present) and direct cutaneous arteries and veins. This layer is particularly abundant in most dogs and cats, but the quantity and elasticity of skin differs from breed to breed and on the physical condition of the animal. Variations in structure are present at different sites of the body surface. For instance the nose have a thick protective keratin layer. In addition, in most skin areas specialized epithelial appendages exist such as hair follicles and sebaceous glands.

Blood supply

In dogs and cats, direct cutaneous arteries are responsible for supplying large areas of skin. They run parallel to the skin in the hypodermis and arise from perforator arteries. Musculocutaneous arteries branch off the perforator arteries and supply small portions of the skin and run perpendicular to the surface of the skin.

The subdermal plexus in dogs and cats is of major importance in companion animal reconstructive skin surgery and should always be preserved when undermining skin for local flaps, especially when no direct superficial arteries can be incorporated in the proposed flap. Axial pattern flaps are flaps based on such direct cutaneous arteries and veins that supply a specific region of dermal tissue. Since the terminal braches of these vessels supply the subdermal plexus, axial pattern flaps have better perfusion than local flaps and are widely used in veterinary reconstructive surgery.

Skin tension

Tension on the wound edges is the most common reason for skin reconstructions to fail and occurs when insufficient skin is available to close the initial defect. The elasticity of the canine and feline skin is primarily the result of the lack of firm attachment of the subcutis to the bone, muscle and fascia. The skin is loose and abundant on most parts of the body, particularly on the neck and trunk, but is less pliable on the limbs, tail and head, especially around the bridge of the nose, nasal planum

and medial canthi. This is the result of linear alignment of fibrous tissues within the skin in this area.

The tension lines of the head and neck region resemble the orientation of the underlying muscles. Generally speaking, incisions should always be made parallel to tension lines to minimize wound tension during closure. Incisions that are made in an angle or perpendicular to these lines may result in wound deformation, wound dehiscence and necrosis. If this is impossible, methods to reduce skin tension should be employed upon closure. These include, from simple to more advanced, undermining the wound edges, selecting tension-relieving suture patterns, using tension-releasing incisions or skin stretching and tissue expansion techniques. If these methods do not allow primary closure of the wound, secondary intention healing or reconstruction with skin flaps or grafts have to be considered.

Reconstruction of the face

Reconstruction of the facial area can be challenging. There are 3 major reasons for the challenge: 1. skin is less abundant than in other areas of the body, 2. There are major essential structures that make proper reconstruction more difficult and 3. It is an obvious area, i.e. people immediately see if something is not completely right. Specific facial reconstructive techniques include the caudal auricular axial pattern flap, the crescentic nasojugal flap, the facial artery axial pattern flap.doc and the superficial temporal artery axial pattern flap.

Wound closure techniques

Most plastic and reconstructive techniques used in companion animals involve the creation of new surgical wounds. The general surgical principles of using aseptical techniques, proper instruments, and delicate tissue handling while creating a surgical wound apply here as well. In addition, appropriate suture materials and suture techniques have to be used for any type of surgery, but for reconstructive surgery in particular.

Complications in performing plastic and reconstructive surgery

Complications of wound closure in plastic and reconstructive surgery are similar to those in general soft tissue surgeries and include wound dehiscence, infection, hematoma or seroma formation, and excessive scar formation. Most complications can be avoided by a proper pre-operative planning and skin mobility assessment, by using a meticulous surgical technique and achieving haemostasis. Chances of flap survival will increase if the size and localisation of the wound is suitable for receiving the flap, if the wound is neither contaminated nor infected and if the wound is not older than 4-6 hours. It is also important that the recipient bed for the flap is fully prepared. Another complication that should be avoided is the development of dead space, which can lead to the formation of abscesses, seromas or hematomas. Formation of dead space can be overcome by placing drains, subcutaneous and walking sutures and bandages. The authors recommend the use of either passive or active drains whenever possible while taking care not to damage the blood supply at the base of the flap by making exit ports.

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SVA FELINE

HYPERCALCEMIA IN CATS - UPDATE ON DIAGNOSIS AND TREATMENT

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Hypercalcaemia in cats- update on diagnosis and treatment

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Diagnostic investigation for ionised hypercalcaemia may be prompted by total hypercalcaemia on a biochemical profile, urolithiasis, neoplasia, constipation or undiagnosed non-specific signs, even when total serum calcium (tCa) is normal. Ionized hypercalcaemia has deleterious long-term consequences and should be managed even in asymptomatic patients.

Differential diagnoses for hypercalcaemia in the cat

Idiopathic hypercalcaemia (IHC) is the most common cause of ionized hypercalcaemia in cats. Cats of any age can be affected. Long-haired cats are over-represented. IHC is characterised by mild to moderate hypercalcaemia, normophosphataemia and an appropriate parathyroid response (i.e. ↓PTH). Clinical signs vary with the degree of hypercalcaemia but around 50% of cats are asymptomatic. The diagnosis is made by excluding other causes.

Chronic kidney disease (CKD) is a common cause of **total** hypercalcaemia. In renal azotaemia, the complexed calcium fraction is usually increased while the ionized fraction is normal. In the azotaemic patient with elevated tCa, measurement of iCa is indicated. If iCa is normal then no further investigation of the hypercalcaemia is required. However, if iCa is elevated in an azotaemic patient, ionized hypercalcaemia may be contributing to the renal dysfunction. Further investigation of hypercalcaemia is indicated. Phosphate-binder use should be reviewed as long-term use of aluminum-based phosphate-binders can cause toxicity which resembles idiopathic hypercalcaemia. Resolution may take months after medication withdrawal. Calcium, particularly carbonate- based binders, should be withdrawn as should calcitriol therapy. A rare differential in the setting of ionized hypercalcaemia and azotaemia is tertiary hyperparathyroidism, where the set-point of the parathyroid glands is increased in advanced CKD (↑ iCa ↑PTH).

Humoral hypercalcaemia of malignancy (HHM) results when tumour-derived parathyroid hormone-related peptide (PTHrP) or other mediators mimick the action of PTH. HHM is less common in cats than dogs. Feline HHM is reported in head, neck and bronchopulmonary carcinomas, lymphoma, osteosarcoma, fibrosarcoma and multiple myeloma.

Primary hyperparathyroidism is rare in cats. Most cases are over 8 years of age. Adenomas are most common, but hyperplasia and carcinoma are reported. Involvement of more than one gland is unusual. Cervical masses are palpable in 50% of cases. A high or normal PTH in the face of ionized hypercalcaemia is consistent with autonomous secretion from the parathyroid gland. Low or normal serum phosphate is expected in primary hyperparathyroidism and HHM. Ultrasound may assist in detecting parathyroid lesions. Surgical patients undergoing parathyroidectomy should be monitored closely for post-operative hypocalcaemia because of feedback atrophy of the remaining glands.

Hypervitaminosis D. Sources of vitamin D intoxication include dietary, rodenticide ingestion, certain houseplants (Cestrum diurnum), calcitriol supplementation and ingestion of analogues (e.g. calcipotriene). Commercial assays measure 25-OH cholecalciferol (calcidiol). Calcitirol assays are less widely available. Vitamin D analogues may not be detected by these assays. PTH and PTHrP are low or undetectable in hypervitaminosis D. If cholecalciferol or ergocalciferol have been ingested, calcidiol levels will be elevated for weeks because of lipid storage. Calitriol levels are normal or occasionally increased. In calcitriol toxicity, calcidiol levels will be normal. Calcitriol may be increased or, because it has a short half life in circulation, levels may be normal.

Granulomatous disease. Activated macrophages can convert calcidiol to calcitriol. Hypercalcaemia has been reported in occasional cases of nocardial, mycobacterial, cryptococcocal, blastomycosis, histoplasmosis and actinomycetal infection.

Hypoadrenocorticism is a rare endocrinopathy of cats and <10% of cases have hypercalcaemia.

Signs associated with hypercalcaemia

Clinical signs may relate to the hypercalcaemia itself and any underlying disease. Signs associated with ionized hypercalcaemia range from inapparent to severe depending on the rate and the magnitude of iCa elevation. Lethargy, anorexia and vomiting are most common followed by polyuria/polydipsia (PUPD), lower urinary tract signs (e.g. stranguria, haematuria, pollakiuria, periuria from urinary tract infection, inflammation or obstruction), weight loss, weakness and tremors. Constipation has been associated with hypercalcaemia in cats. Severe ionized hypercalcaemia can cause obtundation, arrhythmias, seizures and death.

Tips for diagnostic investigation of the hypercalcaemic cat

⬆tCa is confirmed by repeat testing on a fasted, non-haemolysed sample in a well-hydrated patient. Information from the history and physical examination helps to rank the differentials. CKD and neoplasia should be ruled out early on in the investigation.

Where ⬆tCa is persistent then iCa should be measured concurrently. Where tCa is elevated and iCa is normal, no further investigation is necessary. If iCa is elevated consider the following;

- **the age and signalment of the cat** primary hyperparathyroidism is a rare disease of older cats. Long-haired cats are overrepresented for idiopathic hypercalcaemia

-**potential for vitamin D toxicity** review diet history, access to rodenticides, medications, houseplants

-review **physical examination** findings for evidence of neoplasia, cervical lesions, bone pain.

- is there physical or laboratory evidence of **renal disease**? This is the most common cause of ⬆tCa

- what is the phosphate level? phosphate is typically low in PTH and PTHrP-mediated hypercalcaemia. Elevated phosphate occurs with renal disease, vitamin D intoxication and osteolysis.

- imaging may show evidence of neoplasia, urolithiasis or bony lesions (multiple myeloma, other neoplasia, osteomyelitis) Cervical ultrasonography can help to rule in primary hyperparathyroidism.

- PTH, PTHrP and vitamin D testing. Assays have been validated for feline PTH (measured as the intact molecule, iPTH or PTH 1-84) and PTHrP. Vitamin D metabolites do not differ between species. An inappropriate PTH level (elevated or top half of reference range) in the face of concurrent ⬆iCa indicates a parathyroid-dependent process. Whether this results from primary or teritary hyperparathyroidism can be concluded from the rest of the clinical data. Where PTH is appropriately low (bottom half of reference range or undetectable) in the face of concurrent ⬆iCa, then all other diagnoses should be considered. Where PTHrP is elevated then malignancy becomes the major differential. Where PTHrP is normal or subnormal malignancy cannot be ruled out.

- idiopathic hypercalcaemia is diagnosed by ruling out all other causes.

Practicalities of laboratory investigation

- EDTA (ethylenediaminetetraacetic acid) chelates calcium so should not be used for samples for calcium measurement

- for tCa, serum or heparinised plasma collected after a 12 hour fast is recommended.

- adjustment of tCa to TP or albumin is not recommended in cats

- iCa can be measured in-house or submitted to a commercial laboratory.

- for iCa assay, anaerobic collection, the use of dry heparinised syringes and storage at 4ºC (if not processed immediately) reduce errors.

- the availability of PTH and PTHrP should be investigated locally. These tests are currently available at www.dcpah.msu.edu. Sample handling requirements as per website should be followed carefully.

Treatment for idiopathic hypercalcaemia

The long term consequences of hypercalcaemia include renal damage, urolithiasis and soft tissue calcification. Diet change may be helpful and should be individualized. If the iCa elevation is minimal, phosphate is not elevated and diet change has not been effective, then regular monitoring may be all that is required. Glucocorticoids reduce intestinal calcium absorption and bone resorption and may increase calciuresis. The use of frusemide or glucocorticoids long term is not recommended.

Bisphosphonates inhibit bone resorption by promoting osteoclast apoptosis at sites of active bone turnover. Oral alendronate is effective for treatment of idiopathic hypercalcaemia but dosing precautions are necessary and the risks associated with long-term alendronate therapy are not yet fully understood.(1, 2) Food substantially reduces the bioavailability of oral alendronate. Medication must be accompanied by a water swallow because alendronate has the potential to cause drug-induced oesophageal disease (resulting in oesophagitis that may progress to oesophageal stricture formation). We use a starting dose of 10 mg per cat once weekly PO given on an outpatient basis. Medication is administered following a 12 hour fast, with a 5 ml water swallow, and the cat is fed 2 hours later. Once iCa is controlled, the dosing interval is progressively increased to determine the minimum effective dose. Medication-free periods should be considered in the long term. In humans and dogs long term bisphosphonates can cause osteonecrosis of the jaw.(3) Patella fractures and cortical bone thickening in a cat on long term alendronate therapy are reported.(4)

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WSV18-0141

ANIMAL WELLNESS & WELFARE

EMOTIONAL PAIN. WHY IT MATTERS MORE TO ANIMALS THAN PHYSICAL PAIN AND WHAT TO DO ABOUT IT

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EMOTIONAL PAIN: WHY IT MATTERS MORE TO ANIMALS THAN PHYSICAL PAIN AND WHAT TO DO ABOUT IT

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WHAT IS PAIN, AND WHAT IS EMOTIONAL PAIN?

Pain has two meanings, both presented in standard and medical dictionaries. One meaning refers to standard nociceptive, or physical, pain. The other refers broadly to all types of unpleasant feeling states—emotional as well as physical. Unpleasant emotional states are associated with feelings that hurt—they cause suffering. This is emotional pain. Types of emotional pain for which substantial evidence exists in animals include fear (and phobias), anxiety, separation anxiety (or separation distress), isolation distress (loneliness), boredom, frustration, anger, helplessness, grief, and depression.

THE FUNCTIONAL VALUE OF UNPLEASANTNESS

The aversiveness of physical pain serves to command attention, interrupt ongoing behavior, and motivate actions aimed at mitigating the aversive experience. It has been theorized that the function of emotional pain is analogous to that of physical pain, focusing attention on threats and motivating behavior to minimize the threat. For example, it is widely accepted that physical pain promoted survival by protecting the individual from bodily harm – precisely the function of fear. Indeed, pain and fear frequently operate in unison, as in avoidance learning.

EVIDENCE THAT EMOTIONAL PAIN IS NOT JUST A METAPHOR

Much of our language refers to unpleasant emotional states as "painful" and using pain-related terms like broken heart, heartache, crushed, burned, and reopened old wounds. But is this just a metaphor? In recent years evidence has been mounting to indicate that it is not, and that the view of unpleasant emotional states as a form of pain appears to have a valid scientific rationale.

1. A common neuroanatomical basis

Research in humans and nonhuman animals has provided convincing evidence that social pain and physical pain rely on shared brain processes, both anatomically and physiologically.

2. Sensitivity to physical pain corresponds to an enhanced sensitivity to the emotional pain

Findings from several human studies provide evidence that when an individual shows an enhanced sensitivity to one type of pain they also show an enhanced sensitivity to the other.

3. Eliciting physical pain produces the experience of social pain

One study found that social and physical pain cause common psychological consequences. Both social and physical pain produce feelings of being ignored and excluded; previously, only social pain was found to lead to these effects.

4. Methods for alleviating one type of pain alleviate the other

In animals and humans, physical and socio-emotional pain are alleviated similarly by 2 different methods: social support and drugs. Regarding drug therapy, it was recently demonstrated in humans that acetaminophen – a drug well-known for its analgesic effects for physical pain – can alleviate some forms of emotional pain. (Note: Acetaminophen should not be used in animals in any capacity other than as specified in current veterinary drug manuals.)

EVIDENCE THAT SOCIAL PAIN CAN BE MORE DISTRESSING THAN PHYSICAL PAIN

Can emotional and physical pain be compared? Empirically, studies have historically argued that emotional factors weigh more strongly in animals 'behavioral choices than does physical pain. In one study, an electrified grid was placed between puppies and persons to whom they had formed a social attachment. The puppies crossed the grid, receiving shocks the entire way, to reestablish contact with the person. In another study, infant rats were removed from their mothers and placed on the opposite side of an electrified grid. The mother rats could hear their pups' distress vocalizations, but to reach them required walking across the active grid. The mother rats crossed the grid, picked up the pups, and carried them back across the grid to their nest, receiving constant electric shocks in both directions. Anecdotal stories provide further evidence for the greater distress potential for emotional pain. In a well-publicized news story out of Brooklyn, New York, a mother cat was nursing a litter of 4-week-old kittens in an abandoned building that caught fire. The mother cat re-entered the blazing building five times to retrieve each of her five kittens one at a time. In the process, the mother cat received severe burns to her face and head, so damaging that her eyes were swollen shut, her facial hair and ear tips were burned off, and her face was badly disfigured from the burned skin. In light of data from numerous experiments in mammals showing that the infant's call of distress is highly arousing and motivating for the mother, this incident appears to

be a dramatic example of an animal choosing to endure severe physical pain in order to relieve emotional pain. In an experiment pitting an emotional pain (social separation) against a physical discomfort (hunger) in pair-housed tufted capuchin monkeys (**Cebus apella**), researchers directly compared the commodity of social companionship to the commodity of food, a known physiological necessity, in a series of preference tests following commodity deprivations. The majority of subjects chose their social companion over food even after lengthy periods (22 hr) of food deprivation, suggesting that social deprivation was more aversive than food deprivation to most of the monkeys.

Additional insights into the comparison of emotional and physical pain may be found in studies of human torture survivors. Studies of such victims have found, first, that that experiences of psychological and physical torture both have the same detrimental effects on the survivor's mental health. Second, findings have made it clear that the main objective of torture is not to inflict physical wounds or injuries; on the contrary, the objective is to leave psychological wounds. Indeed, even the real purpose of physical torture, which does bear physical scars, is to have a major impact on the long-term psyche of an individual.

TREATMENT OF EMOTIONAL PAIN

Once emotional pain is experienced, treatment principles also parallel approaches to managing physical pain. The objective is to eliminate the unpleasant feeling. Like treatment of physical pain, the first step in management should be recognizing and removing the source. Removing an animal from the fearful environment, lessening disturbing noises and stimuli, and providing hiding places can lessen the intensity of fear and anxiety. Offering mental stimulation (**e.g.** walks, chase games, interactive toys, chew toys, food-packed toys, videos, interactive play, novel objects to explore) and social companionship (**e.g.** increased human attention, accompanying owner to work, dogwalkers, doggie day care) eliminate the causes for boredom and loneliness, respectively.

A pain management technique much more useful for alleviating emotional pain than physical pain is counterconditioning. A goal of counterconditioning—a type of classical conditioning—is to change the animal's emotional response to a particular stimulus. Counterconditioning attempts to replace negative or unpleasant emotional responses to a stimulus with more pleasant responses. Specific examples of situations which have been suggested to benefit from counterconditioning include helping an animal overcome its anxiety associated with a new baby or pet in a household by associating the baby or new animal with pleasant feelings, changing an animal's perception of a stimulus such as a cat carrier or children from that of

fear to that of desire; and eliminating an animal's fear aggression by getting it to associate the object of fear with pleasant rather than unpleasant feelings.

Desensitization – which involves exposing an animal to an emotion-evoking stimulus – is a prominent technique in the relief of the emotional pain of separation anxiety and fears and phobias and is often used in conjunction with counterconditioning. It is advisable to consult with a certified animal behaviorist for proper implementation of desensitization techniques.

Emotional pain, like physical pain, can be alleviated centrally by nonpharmacologic and pharmacologic techniques. Nonpharmacologic methods involve gentle and soothing human contact, such as stroking, petting, and talking to the animal, which can attenuate feelings of anxiety and fear, loneliness, separation anxiety, and boredom. The distress of social isolation and separation when animals are housed apart from familiar and bonded companions (such as dogs hospitalized or kenneled) can be ameliorated by keeping familiar objects (**e.g.** toys, blankets, owner's clothing) with the animal, having the owner visit, and housing housemate pets together. Some behaviorists have suggested that other measures may help lessen the feelings of separation anxiety at home, such as pet sitters, outdoor pens, radios, stimulating and distracting toys (**e.g.** rubber toys stuffed with foodstuffs such as peanut butter and cream cheese, and toys that dispense kibble-type food treats when played with), and, for some animals, the addition of another pet. Aromatherapy—lavender essence, chamomile, and the pheromones Adaptil® in dogs and Feliway® in cats— has also shown antianxiety effects. Other occasionally successful anti-anxiety methods in dogs include classical music and anxiety-wraps.Pharmacologic methods are frequently used to eliminate or lessen the intensity of unpleasant feelings of emotional pain. Anxiolytic and antidepressant medications are the mainstay of treatment for this purpose. Pharmacotherapy can be viewed as having two objectives: (1) relieve emotional discomfort (continuous long-term, or short-term to facilitate response to behavior therapy) and (2) change undesired behavior.References available from author on request

WSV18-0110

IS IT THE HEART OR THE LUNG? - THORACIC RADIOGRAPHY IN THE COUGHING DOG

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IS IT THE HEART OR THE LUNG? THORACIC RADIOGRAPHY IN THE COUGHING DOG

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Introduction

Thoracic radiographs are the imaging method of choice in the coughing or dyspneic dog or cat, but abnormalities of the pulmonary parenchyma are inherently difficult to interpret due to the overlap in radiographic appearance of different diseases. The question of heart vs. lung disease is a common one in patients with clinical signs of cough or dyspnea and in the following we will go over some interpretation criteria that help with this decision.

Importantly, even though we will mostly talk about thoracic radiographs in this presentation, radiographic findings have to be interpreted in light of clinical findings. Abnormalities in heart rate, heart rhythm and presence of a heart murmur are important information to take into account. It is equally important to assess clinical findings over the course of the disease and after treatment has been initiated based on the primary differential diagnosis to determine if adequate treatment response is present.

Radiographic findings of heart disease

Radiographic findings that point to heart disease as the primary cause of cough or dyspnea in a dog include changes in the cardiac silhouette, pulmonary vasculature and pulmonary parenchyma. Cough and dyspnea is caused by left sided heart disease or heart dysfunction. Increased size of the left heart results in a taller cardiac silhouette. In a lateral radiograph this usually results in dorsal displacement of the trachea. In a ventrodorsal (VD) or dorsoventral (DV) projection the cardiac silhouette may be elongated as well but could also have a rounded appearance only if the dog is deep chested and the heart is in a very upright position. Left atrial enlargement results in a convex bulge in the caudodorsal contour of the cardiac silhouette in the lateral views. The main stem bronchi are splayed in the VD or DV projections and there may be a bulge visible in the left lateral aspect of the contour of the cardiac silhouette approximately at 2-3 O'clock. Dorsal deviation of the main stem bronchi

by an enlarged left atrium may result in bronchial compression and cough, particularly if a component of bronchomalacia and bronchial collapse is present. Left atrial enlargement is typically pronounced in dogs in left sided congestive heart failure and a lack of left atrial enlargement should prompt the clinician interpreting the radiograph to consider other causes for the cough.

Evaluation of the pulmonary vasculature should always go hand in hand with evaluation of the cardiac silhouette. It is especially important in dogs with cough or dyspnea where left-sided congestive heart failure is considered. In presence of left heart failure there is volume overload of the left atrium and subsequent congestion of the pulmonary veins trying to return the blood volume from the pulmonary circulation into the heart. Enlargement of the pulmonary veins is therefore an excellent radiographic sign of left heart dysfunction. Pulmonary venous dilation, however, may not be present if a patient has been treated with diuretics prior to obtaining the radiographs. Additionally, very mild pulmonary venous dilation may be difficult to recognize radiographically so a lack of pulmonary venous dilation should not be used to completely rule out heart failure.

Lastly, there are pulmonary changes associated with left sided congestive heart failure. Cardiogenic edema transitions from the interstitial space to the alveolar space and therefore has the potential to create variations of pulmonary patterns. Once there is pulmonary venous congestion with associated pressure increase in the vessels, fluid may be leaking into the interstitial space around the vessels. The lymphatics initially compensate by increased drainage of the interstitial space but ultimately may become overwhelmed, resulting in fluid buildup in the interstitial space. The interstitial fluid will cause opening of the tight junctions between alveolar wall cells and fluid will flow into the alveolar space. It therefore makes sense that pulmonary patterns can vary from interstitial to alveolar, depending on the stage of the disease. To make things more complicated the cardiogenic interstitial edema is present around the bronchial walls as well which can give the appearance of a peri-bronchial infiltrate and a radiographic bronchial or bronchointerestitial pattern. This pattern mostly occurs in large breed dogs with cardiogenic pulmonary edema. The distribution of pulmonary changes is often more helpful than the pattern itself – the pulmonary changes associated with heart failure tend to be located in the perihilar area or in large breed dogs in the caudodorsal lungs.

Radiographic findings of pulmonary parenchymal disease

Pulmonary parenchymal or large airway disease is the main alternative differential diagnosis in the coughing dog. Increased pulmonary opacity in absence of cardiac and pulmonary vascular changes are the main

findings that lead to suspected lung disease. To further characterize the type of lung disease, pulmonary patterns are often the intuitive choice. The concept of pulmonary pattern recognition was developed to aid in generating the most likely differential diagnosis based on the assumption that different disease types affect different compartments within the lungs. There is, however, a large degree of overlap between the radiographic pulmonary patterns in many types of lung disease and focusing on pulmonary patterns only is not the most successful way of interpreting radiographs. The distribution of the abnormal lung patterns as well as other radiographic findings are often more helpful than the pulmonary patterns alone.

Alveolar pulmonary pattern is characterized by marked increased pulmonary opacity, loss of visibility of vascular structures in the affected segment, presence of air bronchograms and if only on lung lobe is affected, presence of a lobar sign. Pneumonia is one of the most common causes of alveolar pulmonary pattern besides heart failure. Contrary to heart failure, alveolar patterns with pneumonia are typically ventrally distributed with a preference to the cranial and right middle lung lobes. Ventral alveolar patterns can also be caused by atelectasis which is differentiated from acute pneumonia by evidence of volume loss of the affected lung lobe causing a midline shift to the same side. Pleural effusion is rarely seen with pneumonia on dogs and presence of effusion should point to a different disease process such as trauma, neoplasia or primary pleural space disease with secondary involvement of the lungs.

Bronchial pulmonary patterns can be difficult to recognize as they do not result in overall increased pulmonary opacity but there is also a tendency to over-interpret the normal structure of the lung parenchyma as bronchial pattern. Signs of advanced bronchial disease include bronchiectasis which is characterized by lack of normal tapering of the bronchial lumen towards the periphery of a bronchus. Presence of bronchiectasis is often a sign that there is chronicity to the bronchial disease. Chondromalacia leading to airway collapse can be recognized by paying close attention to airway diameter between radiographic projection particularly if they were obtained during slightly different phases of respiration. In a normal dog there should be minimal variance between airway diameter in in- vs. expiratory radiographs.

Interstitial lung disease is characterized by diffuse increase in pulmonary opacity without loss of visibility of vascular structures or bronchial walls. Interstitial pattern is the least useful of the pulmonary patterns. Most disease processes have an interstitial component, and as outlined above when describing the pathogenesis of cardiogenic edema, many disease processes start out as or resolve as interstitial pattern.

Overlapping disease patterns

There are cases where it is very difficult if not impossible to decide of the radiographic findings are most likely due to cardiac or pulmonary disease. One of the more difficult diseases to recognize radiographically are pulmonary infiltrates secondary to pulmonary hypertension. Dogs with moderate to severe pulmonary hypertension can present with diffuse patchy alveolar infiltrates consistent with non-cardiogenic edema. The clinical presentation may include acute dyspnea and syncope and often heart murmurs suggestive of valvular insufficiency are present. The clinical and radiographic findings may lead to an initial misdiagnosis of congestive heart failure or pneumonia whereas the dogs improve both clinically and radiographically once therapy with sildenafil is instituted.

Atypical appearance of heart disease can occur if there is acute chorda tendinea rupture, or in cases of severe cardiomegaly, left atrial rupture with subsequent acute pericardial effusion. Pulmonary thromboembolism is another disease with a large variation in radiographic appearance, ranging from normal appearing lungs to hyperlucency or patchy alveolar pattern. These dogs typically present with marked dyspnea.

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WSV18-0329

INFECTIOUS DISEASES AND GASTROENTEROLOGY (TRANSLATED INTO MANDARIN CHINESE)

MANAGEMENT OF THE CANINE INFECTIOUS RESPIRATORY DISEASE COMPLEX (KENNEL COUGH)

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Objectives. The primary objectives of this session are to review the known bacterial and viral causes of the canine infectious respiratory disease complex CIDC followed by a discussion of optimal diagnostic tests, treatments and preventions.

The most common agents associated with CIRDC include canine adenovirus 2, canine respiratory coronavirus, canine influenza viruses (H3N8 and H3N2), canine herpesvirus, canine pneumovirus, canine parainfluenza virus, **B. bronchiseptica, Streptococcus equi** subspecies **zooepidemicus**, and **Mycoplasma spp**.. Dogs with CIRDC generally present with an acute onset of cough with or without sneezing. Nasal and ocular discharges can also occur depending on the causative infectious agent. Some agents like the H3N2 influenza virus induce fever. A very acute course of disease resulting in hemorrhagic pneumonia and death seems to be most common with **S. equi** var. **zooepidemicus**. If the affected dog has not completed a routine distemper, parvovirus, adenovirus 2, and parainfluenza vaccine series, canine distemper virus infection (CDV) can also be a cause of CIRDC. This virus also induces diarrhea and can cause mucopurulent ocular and nasal discharge.

Each of the CIRDC agents can be harbored by normal dogs making it difficult to interpret molecular diagnostic assay results in some cases. Coinfections can occur in some dogs and may potentiate clinical signs of disease. While vaccines are available in some countries for many of the causes of CIRDC, with the exception of CDV, immunity is not permanent and booster vaccines are needed yearly (2011 AAHA Canine Vaccination Guidelines; www.aahanet.org). In addition, even when dogs are vaccinated against canine parainfluenza virus, canine adenovirus 2, H3N8 canine influenza virus, H3N2 influenza virus, or **B. bronchiseptica**, clinical signs of disease can still occur if the dog is exposed. However, the clinical disease should be less severe and of shorter duration than in dogs that have not been vaccinated.

All dogs with a history consistent with acute cough should have a thorough physical examination performed preferably in the parking lot of the hospital or an examination room that is easy to isolate and disinfect. If pulmonary crackles are auscultated, thoracic radiographs are indicated; for biosafety reasons, the dog should be transported through the hospital on a gurney that can

be disinfected and the radiographs made at the end of the day so that the radiology room can be disinfected. Respiratory treatment guidelines are available from ISCAID which also discuss the diagnostic issues with CIRDC (Lappin et al, 2017). Cytology of discharges to attempt to diagnosis bacterial CIRDC and pick antibiotics was not recommended. However, if fungal diseases are suspected, cytology of nasal discharges can still of diagnostic benefit. If the affected dog is not febrile, is eating, and has no evidence of pneumonia, no specific tests are needed and the dog can just be treated as indicated by the history and physical examination findings.

If the dog has significant clinical illness or an outbreak is suspected, aerobic bacterial culture and antimicrobial susceptibility testing, **Mycoplasma spp**. culture (or PCR assay), and molecular diagnostic procedures for canine parainfluenza virus, canine adenovirus 2, canine distemper virus, canine respiratory coronavirus, canine influenza viruses, canine herpesvirus, pneumovirus, **B. bronchiseptica**, and **Mycoplasma spp**. (or M. cynos alone) can be performed. However, as discussed, nucleic acids of all of the CIRDC agents can be amplified from both healthy and diseased dogs and modified live vaccine strains of the organisms can also be amplified. Thus, a positive molecular assay result may not prove causation. Molecular assays also can give negative results by the time dogs are presented since viral shedding rates tend to peak very early in disease. For the influenza viruses, if the dog has not been vaccinated, a rising titer can also be used to prove an acute exposure and so saving serum from the first presentation to compare to a second serological test results 2-3 later can be beneficial.

Most dogs with CIRDC have viral causes and so antibiotics are usually not indicated. Most dogs maintain normal appetite and attitude and clinical signs generally resolve spontaneously within 10 days without antimicrobial therapy. The ISCAID Working Group recommended that antimicrobial therapy only be considered within the 10-day observation period only if fever, lethargy, or inappetence is present together with mucopurulent discharges (Lappin et al, 2017).

When dogs with suspected bacterial CIRDC are identified based on mucopurulent nasal discharge, fever, lethargy, or inappetence but no clinical evidence of pneumonia, the ISCAID Working Group recommended the administration of doxycycline (5 mg/kg, PO, q12hr or 10 mg/kg, PO, q24hr) empirically for 7 – 10 days as the first line antimicrobial option. This antibiotic should be effective for most CIRDC cases caused by **B. bronchiseptica** or **Mycoplasma spp**.. The other first line drug options recommended by the ISCAID Working Group were amoxicillin or amoxicillin-clavulanate (Lappin et al, 2017). Amoxicillin should be effective for most

secondary bacterial infections (to primary viral infections) and **S. equi var**. **zooepidemicus**, but not **Mycoplasma spp**. which lack cell walls. Amoxicillin-clavulanate may be required for some **B. bronchiseptica** isolates. Inhalational aminoglycoside therapy has also been anecdotally mentioned as a treatment for **B. bronchiseptica**-associated CIRDC. If the first drug chosen is ineffective and bacterial disease is still suspected after the first 7 days, the ISCAID Working Group recommended that a more extensive diagnostic workup should be considered prior to considering use of other drug classes like fluoroquinolones or azithromycin. Repeated diagnostic tests are not needed in dogs with CIRDC that respond clinically.

Dogs with cough due to uncomplicated CIRDC should have rest enforced, be handed with a harness, not a collar, fed soft or canned food if showing signs of discomfort when swallowing, and have the cough controlled with anti-tussive agents.

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NURSES (HILLS)

ASSESSING CANINE AND FELINE BEHAVIOR

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Assessing Canine and Feline Behavior

In order to know what the dogs and cats is telling the owner and diagnose why the dogs or cats are misbehaving, it is important to know their body language and their society. This lecture will cover how dogs and cats communicate with each other and how shall we understand what they are telling us.

Dogs try to communicate with humans instead of trying to dominate us. The famous myth is that they are trying to dominate the family member and take over us so we need o "dominate" them. Is this really true? Looking at the canine languages, we must understand that they are not trying to dominate us but they are trying to tell us what they feel.

Cats are thought to be non-social animals and they like what they like to do and they do not care what humans does. Is this true? Do they life alone with our creating society? The truth is that they are social animals if they have enough resources. They communicate with each other and they even help each other. This lecture will cover how cats communicate to each other and what to look for when we communicate with them.

WSV18-0062

COMMON CANINE ORAL PATHOLOGY DIAGNOSIS AND TREATMENT

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Learning objective: Overview of dogs dental oral pathologies will be presented. Most oral problems cause pain and infection, therefore appropriate diagnosis should be followed by management of the diagnosed diseases.

Dental disease is the number one clinical problem in small animal practices.

And thus oral pathology is exceedingly common in canine patients. In addition, there is a very wide variety of pathologies that are encountered within the oral cavity of the dogs. These conditions often cause significant pain and/or localised, regional and systemic infection.

The presented oral problems are statistically quite common and it is important to diagnose these conditions at the primary health care or general practices. Despite the fact that they may lead to serious complications, the affected patients rarely display their discomfort, and behave almost normally.

Periodontal disease is by far number one clinical oral problem in dogs and it includes gingivitis and periodontitis. It has been shown that periodontal disease is more common in older animals. this malady has inflammatory character and is associated with presence of infection thus have numerous consequences for overall health of the patient.

Persistent deciduous teeth are exceedingly common, especially in small and toy breed dogs. There should never be two teeth of the same type in the same place at the same time and these teeth should be extracted as early as possible.

Intrinsically stained (discoloured) teeth can appear pink, purple, yellow, or grey. The most common intrinsic stain seen in dogs is caused by pulp haemorrhage due to trauma.

A study by Hale showed that only 40% of intrinsically stained teeth had radiographic signs of endodontic disease, however 92.7% are non-vital.

Fractured Teeth seen in veterinary medicine are complicated and uncomplicated. Complicated fractures occur when the endodontic system (pulp) is directly exposed. Uncomplicated fractures are when the damage is limited to the enamel +/- dentin. Both types require therapy, however treatment for each is often different.

There are several reasons that teeth may be missing. These reasons include: congenitally missing, previously extracted or exfoliated, fractured (or extracted) with retained roots, or impacted.

The oral cavity is the fourth most common place to encounter neoplastic growths.

In dogs a large proportion of proliferations are reactive or benign. It is crucial to perform full diagnostic procedure and start treatment as soon as the growth in oral cavity was detected.

WSV18-0322

SVA PRACTICE MANAGEMENT

WHAT ARE THEY THINKING? RESPONDING TO MODERN AND FUTURE PET OWNERS

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1. SS - What Are They Thinking? Responding to Modern & Future Pet Owners

Based on just-released research by VetHelpDirect and Merck Animal Health, this talk is a comprehensive overview of the differences in pet owner desires (broken up by dog and cat owners) among pet owners born before and after 1980. The talk contains action steps veterinarians should be considering if they want to fully address the changing demand of today's pet owners.

Pet owners' expectations of the vet's online provision have changed drastically in the space of two years, recent research from VetHelpDirect shows that clients want to be able to communicate with the vet via social media, email and even virtual chat. They expect a high level of responsiveness from their vet with results from our latest studies showing that over 35% of pet owners in the UK expecting a response to questions asked via social media within one hour, this contrasts with 15% when the same survey was run 2 years ago. It presents a huge challenge to the veterinary practice and strategies are required to deal with this issue.

The age of the pet owner has a profound effect on their approach to pet care. Recent research shows that these owners are more influenced by online reviews than their friends opinions when it comes to choosing a vet.

The Merck Pet Owner Pathways Research told us that Millennials are prepared to spend on their pet and they are more likely to include their veterinarian in their pet owning journey than older demographics. We know that all age groups will pay more for a brand known for its social value but this effect is particularly marked in the millennial demographic. Nielson tells us that 49% of people are prepared to pay more for a brand known for its social value, this rises to 60% for 18 – 24 year olds.

Online millennial pet owner behaviour is different too, Instagram use is more widespread; however, rates of using Facebook still exceed this threefold and are close to the average rates for all ages. The much hyped SnapChat actually shows very low levels of use even amongst the millennial demographic.This new demographic represents an opportunity for veterinary practices, in order to reach Millennials consider Instagram but don't forget Facebook which is likely to be much more important, even for this demographic.

Most vets would accept that their clients and potential new clients go online for information when they are making decisions about their pet, including which vet to choose. In 2010 Google coined the term 'Zero Moment of Truth' to define this stage of the purchasing journey when people are choosing a service or product; this includes reading online reviews, watching videos and reading content. There is strong evidence that this phenomenon exists amongst pet owners looking for a vet and implies that vets must consider their digital marketing across several platforms. 'Micro-moments' are Google's latest iteration of this concept and take in to account the fact that people are now using the mobile internet far more widely and that internet research is now happening in tiny spaces of time or 'Micro-moments'. Vet practices need to consider that anything published is more likely to be accessed in short snatches of time, more likely by mobile and create copy and images accordingly.

WSV18-0304

HOW I TREAT RECTAL PROLAPSE

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If you would like a copy of the surgical procedure on DVD contact videovet@mac.com.

RECTAL PROLAPSE

Rectal prolapse is a sign, not a disease. Some of the underlying etiologies include intestinal parasitism, chronic diarrhea, dystocia, or any disease causing chronic tenesmus, stranguria, or abdominal pressing. Diagnosis is made by visual observation of a red tube-like protrusion of rectal mucosa.

Rectal prolapse must be differentiated from a prolapsed intussusception. The diferential diagnosis of rectal prolapse and prolapsed intussusception can be done by placing a finger or blunt instrument such as a thermometer between the prolapsed mucosa and mucocutaneous junction. If resistance is met, the diagnosis is rectal prolapse. If the finger or instrument is easily passed, a prolapsed intussusception is diagnosed.

Rectal prolapse can be managed by several methods including reduction and purse-string suture, amputation, or colopexy. The technique selected depends upon viability of the prolapsed tissue, size and reducibility of the prolapse, and recurrence after a previous technique has failed. In small animal practice, patients with rectal prolapse are generally presented early; before significant mucosal necrosis occurs. Therefore, initial management generally involves reduction and placement of a purse-string suture. This is accomplished by general anesthesia, application of 50% dextrose to reduce mucosal edema, gentle reduction of the prolapsed tissue, and placement of a purse-string suture shows the typical appearance of a rectal prolapse. This suture is tied just snug enough to prevent rectal prolapse yet loose enough to permit defecation. Topical anesthetic ointment (e.g.,1% dibucaine) [Nupercainal ointment, Ciba Pharm,Ciba-Geigy, 556 Morris Avenue, Summit, NJ 67901] is instilled in the rectum postoperatively and continued for two to three days after purse-string removal. The purse-string suture remains for two to three days. Diagnosis and treatment of the underlying cause aids in ultimate success.

A nonreducibile viable prolapse or a recurrent rectal prolapse may be treated by celiotomy and colopexy. A ventral midline celiotomy is performed and the prolapse reduced by gentle traction on the colon and concurrent manipulation of the prolapsed rectum.

Colopexy

See the DVD for a detailed video description of the colopexy technique. Once the rectal prolapse is reduced the colon is gently retracted into the abdominal cavity and brought against the left sublumbar body wall. Care is taken to pexy the colon in its 'functional' position in the abdominal cavity. Do not place excessive tension on the descending colon during colopexy. The peritoneal surface of the left sublumbar body wall is scarified. A similar sized area of colonic serosa on the antimesenteric border is also scarified. Scarified surfaces of the colon and sublumbar body wall are sutured using a two layer closure. The dorsal margins of each scarified surface are sutured together first, using a simple interrupted or simple continuous suture pattern with 3-0 or 4-0 synthetic absorbable suture material. Next, the ventral margins of the scarified surfaces are sutured together in a similar fashion completing the colopexy. Care is taken to make certain sutures do not penetrate the lumen of the colon. Abdominal closure is routine. Topical anesthetic ointment is instilled rectally after surgical correction and continued for five to six days postoperatively.

PROGNOSIS

The prognosis for rectal prolapse is favorable if the underlying problem can be controlled.

WSV18-0223

APPROACH TO THE BLEEDING PATIENT IN THE EMERGENCY ROOM

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Pathophysiology

Coagulation disturbances are common in emergency and ICU patients. It may be a sequel of various etiologies including intoxications (rodenticide intoxication), snake bite, GDV, pancreatitis, severe trauma, sepsis (e.g septic peritonitis, pyometra, pyothorax) or any etiology associated solely with thrombocytopenia (IMT, Erlichiosis). Although all the aforementioned etiologies carry various coagulation disturbances severity, clinical presentation, treatment, prognosis and outcome, they all share in common inflammatory process so called systemic inflammatory response syndrome (SIRS). SIRS and sepsis are thought to be intimately associated with coagulation system (Fig 1). The mortality is due to organ failure and increases with the number of organ system involvement and failure. Much of the organ failure is due to the microvascular and coagulation disturbances leading to enhance thrombosis and disseminated intravascular coagulation (DIC). The coagulation disturbances are dynamic and variable, depending on the etiology and the severity of the primary disease. Cytokine production, vascular damage and the release of tissue factor all contribute to the stimulation of coagulation cascade, down regulation of the fibrinolytic system and consumption of the endogenous anticoagulant.

In healthy animals, the normal clot formation and fibrinolysis mechanisms are well balanced, so coagulation and formation of clots occur only on demand. However, during massive trauma, infective, inflammatory/septic state this balance may be disrupted, leading to concurrent excessive clot formation and bleeding (Fig. 1). Thus, DIC may be considered as an uncontainable burst of thrombin generation and activation, resulting in systemic fibrin formation, followed by plasmin and kinnin activation, with simultaneous suppression of the physiologic anticoagulation mechanisms and delayed fibrin removal as a consequence of impaired fibrinolysis (Fig. 2).[1-3] During this excessive intravascular coagulation phase, platelets and coagulation factors are consumed, resulting in thrombocytopenia, thrombocytopathy and depletion and inactivation of coagulation factors.

DIC is categorized into bleeding, organ failure and non-symptomatic types according to the sum of vectors for hyper coagulation and hyperfibrinolysis. There are 3 types of clinical presentation of DIC; bleeding, massive bleeding and the organ failure hypofibrynolitic type.[4]

The bleeding, massive bleeding and hypofybrinolytic types

When the dominant feature of haemostatic disorder is hyperfibrinolysis, bleeding is the primary syndrome, therefore is called the bleeding type. This form of DIC is often seen in patients with major trauma, neoplasia (e.g hemangiosarcome, leukemia, lymphoma) and obstetric disease in women. In the other hand when the hypercoagulation is the more remarkable process along with hypofironolysis, organ failure is the common symptom. This type of DIC is called the organ failure type due to intravascular clott formation and deposition, resulting in organ failure commonly liver, kidney and CNS. This type is common in infectious diseases, particularly sepsis.[4]

The third type is the massive bleeding type or so called consumptive type, when both coagulation and fibrinolysis are highly activated resulting in whispered clott formation and massive bleeding in the same time, which may result in acute death. This form of DIC is seen in patients with heatstroke, snake bite and severe acute necrotizing pancreatitis or any major trauma.

When both vectors of coagulation are weak (coagulation and fibrinolysis) there are mild to non clinical signs directly related to the coagulation disorders, commonly seen in different neoplastic diseases in which only laboratory coagulation parameters are mildly abnormal, and there is a new chronic coagulative balance, tha can deteriorated to one of the above described clinical state of DIC.[4]

Sepsis and coagulation

The most common etiology involved in coagulation abnormalities in both human and veterinary medicine ICU is sepsis. Sepsis is one of the oldest and most elusive syndromes in medicine. However, with the advent of modern antibiotics, germ theory did not fully explain the pathogenesis of sepsis: many patients with sepsis died despite successful eradication of the inciting pathogen. In addition it was noticed that other etiologies not infective resemble sepsis in the clinical signs; tachycardia, tachypnea, elevated/decreased white blood cells count, hypoglycemia, with no apparent infectious site (e.g heatstroke, trauma, cancer). Thus, researchers suggested that it was the host, not the germ that drove the pathogenesis of SIRS/sepsis. The incidence of sepsis increase with the use of immunosuppressive drugs, chemotherapy and invasive procedures. It is a leading cause of death in human and in the veterinary medicine with high mortality rate of 30-50%. Common diseases associated with sepsis in the veterinary medicine are pneumonia, pyothorax, peritonitis, pancreatitis, prostatitis and wound infection.

During inflammation there is increase in plasminogen activator factor inhibitor I (PAI-I) induced markedly increase levels cytokines and liposaccharide, in the blood as a cause of hypofibrinolysis, with consequence of thrombus formation and deposition. Moreover, histones are highly conserved, positively-charged nuclear proteins, serving as the basic structure block unit of the chromatin, leak from damaged and activated immune system cells (e.g., neutrophils and mast cells) and by neutrophil extracellular traps (NETs) into the extracellular space, exhibiting toxic, pro-inflammatory and pro-thrombotic properties. Histones promote the apoptosis of vascular endothelial cells and platelet aggregation enhancing thrombus formation[5].

Fig. 1: The mechanisms leading to the development of disseminated intravascular coagulation
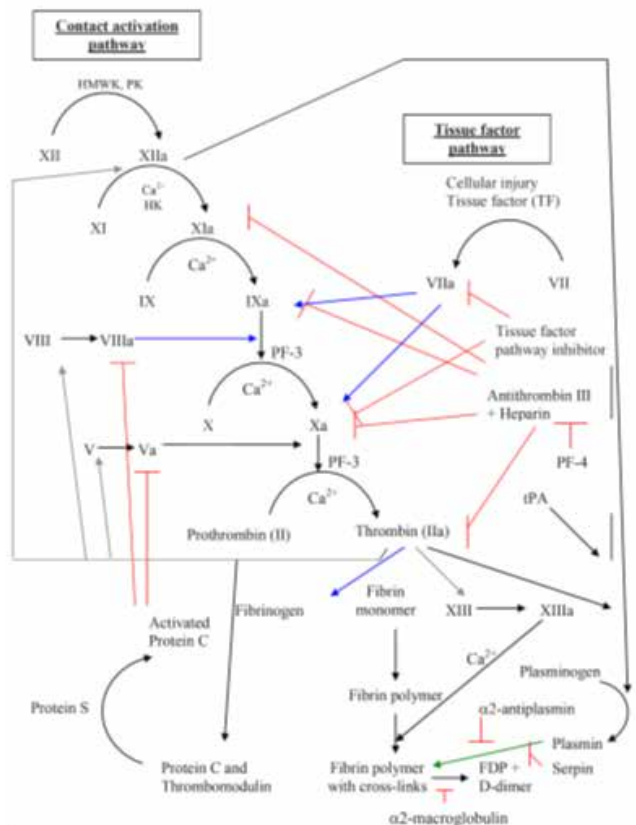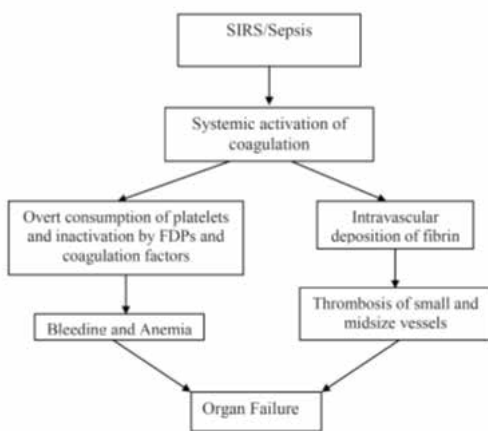
Fig. 2: Coagulation pathways: Contact Activation Pathway and Tissue Factor Pathway - formally known as the Intrinsic and Extrinsic Pathway respectively. PK - prekallikrein, HMWK - high molecular weight kininogen, tPA - tissue plasminogen activator. FDP - fibrin degradation products. Serpin - serine protease inhibitor. PF-3,4 - Platelet factor - 3, 4. The letter "a" following factor number (e.g. Xa) indicates activated factors. Arrows: Black - conversion/activation of factors, Red - inhibition action, Blue - reaction catalyzed by activated factor, Gray - actions of thrombin, Green - lyses.

Clinical signs

Dogs with DIC may present with several clinical presentations. Three phases of DIC are recognized: the peracute hypercoagulable phase, the acute consumptive phase and the chronic silent phase. Both the hypercoagulable and the chronic silent phases are non-overt. The latter phase appears to be common in dogs with malignancy and other chronic disorders. The peracute and acute phases may result from an acute phenomenon (e.g. sepsis, acute pancreatitis, heatstroke, electrocution), or it represents acute decompensation of the chronic silent process.[6] Acute DIC is extremely rare in cats. Regardless of the pathogenesis, dogs with acute DIC are often presented for treatment due to profuse spontaneous bleeding, either primary (petechiae, ecchymoses, hematochezia, melena, hematemesis and hematuria) or secondary (blood in body cavities) in concert with hemostatic disorders, and constitutional signs, secondary to anemia or to parenchymal organ thromboses leading to multiple organ dysfunction.[2,6-8] Clinical signs may be highly variable depending on the underlying primary disease and the phase of DIC (i.e. the

balance between thrombosis and hemorrhage). Most cats with DIC do not show evidence of spontaneous bleeding and the clinical signs are often those associated with the primary disease.[3]

Diagnosis

Diagnosis is complex and should be based on several and continuous blood coagulation parameter and clinical signs. The definition and diagnostic criteria of DIC in veterinary medicine are somewhat controversial; however it is generally agreed that DIC is suspected when an underlying clinical condition known to precipitate DIC occurs, clinical signs of bleedings tendencies (e.g hematochezia, melena, epistaxis ecc.) with at least one abnormality from the 4 following coagulation groups; 1. Thrombocytopenia < 150,000, 2. Coagulation parameters; prolonged prothrombin time (PT), activated partial thromboplastin times; aPTT and activated cloting time (ACT), 3. Inhibitor consumption; decreased protein C, protein S or antithrombin activities; PCA, PSA and ATA, respectively), and increased fibrinolysis; hypofibrinogenemia, increased D-dimer and fibrinogen degradation products concentrations (FDPs).[3](Table 2)

Table 2: *Laboratory screening tests for DIC in dogs and cats*

Parameter/test	Early Hypercoagulable phase	Clinical Manifestation phase
Platelet count	= □	□
Schistocytosis	None	□
Prothrombin time (PT)	= □	□
Activated partial thromboplastin time (aPTT)	= □	□
Activated clotting time (ACT)	= □	□
Anti-thrombin-III activity	= □	□
Fibrinogen	□=□*□	□
Fibrinogen degradation products (FDPs)	= □	□
D-dimer	= □	□
Total Protein C	=□	□
Thrombin-antithrombin complex (TAT)	□	□=□
Plasmin-antiplasmin complex (PAP)	□	=□

Legend Table 2: Laboratory results should always be interpreted with caution, in light of the history and clinical signs, since an abnormality in any single test is not specific for the diagnosis of DIC. These tests are not very sensitive markers of DIC, and may yield normal results in the early hypercoagulable stages of DIC. Serial monitoring of laboratory tests to assess the trends in individual DIC-suspected patients is useful.

* Depends on the underlying disease.

Thromboelastography (TEG)

This technique characterizes the coagulation function by recording a tracing that represents blood clot creation and breakdown. The tracing is a sum of the interactions between coagulation factors, platelets, fibrin, fibrinolysis and time. Different thromboelastographic patterns have been identified in a variety of haemostatic disorders, including coagulation factors deficiency, thrombocytopenia, increased fibrolysis and hypercoagulability.[9] In septic human patients TEG has been utilized to identify the hypercoagulable state that precedes the clinically recognizable phase of DIC.[10] In veterinary medicine, TEG has been evaluated in a number of studies in dogs and cats.[11-13]

Treatment

Treatment should be instituted immediately once a diagnosis of DIC has been established or when a high index of suspicion is present. Removing or eliminating the precipitating cause constitutes the cornerstone and the main therapeutic goal for patients with DIC. The wide variety of underlying disorders makes the therapeutic approach to DIC particularly difficult.[14]

The treatment aims for DIC in dogs and cats include the following:

1. Impeding, possibly stopping, intravascular coagulation and hemorrhage
2. Maintaining good parenchyma organ perfusion
3. Preventing secondary complications
4. Use of anticoagulant- heparin
5. Use of antifibrinolytic agents- Transhaxemic acid

Replacement therapy is the mainstay for the treatment of DIC [14]. A dual approach is used to halt intravascular coagulation:

Blood component therapy:

Administration of fresh or fresh-frozen plasma (FP or FFP, respectively) (at least 30-50 ml/kg /day given initially at 10 ml/kg/hr and then at 2ml/kg/hr). This is done to replace the consumed coagulation factors. Alternatively, fresh whole blood can be administered as a source for the coagulation factors, inhibitors as well as platelets. FP/FFP administration is aimed at halting the consumption of platelets, coagulation factors and inhibitors (e.g. AT, a2-macroglobulin) in order to arrest the ongoing hemorrhagic and coagulation processes.

Heparin - unfractionated and low molecular weight heparin:

Administration of heparin, only during the peracute hypercoagulable phase, when PT and aPTT are shortened and AT III activity is at least 80%. Heparin can also be administered following FP/FFP administration and only when the laboratory coagulation tests were normalized.

The literature lacks controlled studies on the use of heparin in DIC. Clinical reports and retrospective studies

do not provide a clear-cut indication whether heparin use is beneficial. Heparin effects may be variable, depending on the underlying cause and the stage of DIC.[8] Various studies have shown either positive effects, no effect or negative effects of heparin treatment and therefore its use in DIC is still under debate.

Heparin enhances thrombin and factor Xa inactivation through activation of AT III inhibitory actions and therefore is ineffective when AT III plasma activity is insufficient. Because AT III activity in DIC is usually low (as a result of consumption and possibly due to inactivation), it is advisable to provide the patient with sufficient quantities of this anticoagulant, most efficiently through blood component replacement. In a single study in dogs with different coagulopathies, FFP therapy did not result in increase plasma AT III activity.[15]

Heparin has another anti-clotting activity, through induction of the release of low affinity, microvasculature glycosaminoglycan-bound tissue factor pathway inhibitor (TFPI) pools into the circulation. Enhancement of TFPI activity represents an upstream, even more specific anticoagulatory action compared to AT III, in cases where coagulation is triggered by bacterial lipopolysacharides in sepsis.[16]

To the best of our knowledge, there are no controlled studies determining the appropriate heparin dose for DIC in veterinary patients, or even substantiating its use in this syndrome. Extrapolation from the human literature is difficult because human patients at risk for DIC are generally also at high risk for deep vein thrombosis (DVT). They are consequently usually treated with aggressive heparin for DVT risk. This is generally not an issue for our patients. Controlled studies are difficult to perform since DIC is not a primary disease, and populations with DIC vary widely in terms of manifestation and prognosis due to variability in the underlying disease. Several authors have, however, proposed that in DIC sodium heparin is given at dose of 50-100 IU/kg SQ q8h. This dose should be adjusted through monitoring of the aPTT and AT III activity, with the aim to prolong the aPTT by up to 30% above the upper reference interval in a hypercoagulable state or, when such value is achieved through replacement and supportive therapy.[2]

Low molecular weight heparin (LMWH) is composed of heparin fractions with molecular weights of 4000 to 8000 daltons. LMWH was found to be more advantageous than unfractionated heparin (UFH) in dampening the activated coagulation. In human patients with endotoxemia, it has been shown to significantly reduce mortality. In a double-blind, controlled study in human DIC Patients, LMWH was more beneficial compared to UFH in decreasing bleeding complications.[17]

UFH binds to AT III, resulting in a conformation change of AT III that leads to greatly enhanced inhibition of many coagulation factors, such as thrombin, Xa, XIa, XIIa and IXa. Unlike UFH, LMWH, due to its small molecular size, cannot simultaneously bind to both AT III and thrombin, and therefore inhibits thrombin to a lesser extent. However, when compared to UFH, LMWH has greater affinity to and enhanced inhibitory efficiency of factor Xa. LMWH also has a lesser tendency to bind to macrophages, plasma proteins and platelets, accounting for its limited hepatic clearance, prolonged half life and better bioavailability. In humans, LMWH has been found to have a 2-4-fold longer half-life than UFH, with greater bioavailability and more predictable anticoagulant effects.[18] In addition, with LMWH, the likelihood of developing heparin induced thrombocytopenia is reduced compared to UFH.[18,19]

Antifibrinolytic agents

A large body of evidence in human medicine supports the use of antifibrinolytic drugs for control of hemorrhage in a variety of clinical settings. Antifibrinolytic therapy is commonly used in human patients undergoing cardiovascular, pediatric and orthopedic surgery, dental procedures, or in cases of severe menstrual or postpartum bleeding, and mainly in major trauma. To date, antifibrinolytic drugs are clinically used in people for hemorrhage control, including ε-aminocaproic acid (EACA), tranaxemic acid (TxA), and aprotinin. The lysine analogues, EACA and TA, inhibit plasminogen, and to a lesser extent, increase antiplasmin activity, resulting in decreased fibrinolysis, with TxA having a 10-fold activity compared to EACA. The results of the large CRASH-2 trial showed that the administration of TxA within the

first three hours after hospital admission reduced mortality in trauma patients. Mortality rates were lowest among patients who received TxA within the first hour after hospital admission, and authors concluded that TxA should be given as early as possible to bleeding trauma patients.[20]

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WSV18-0052

NAVC SHORT TOPICS FROM EXPERTS

HOW I TREAT POST-OP TPLO WITH PHYSICAL REHABILITATION

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NAVC SHORT TOPICS FROM EXPERTS: HOW I TREAT POST-OP TPLO

WITH PHYSICAL REHABILITATION

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Canine rehabilitation is the application of physiotherapeutic techniques to evaluate and treat musculoskeletal impairments in our canine patients. It incorporates the use of objective outcome measures (goniometers, girthometers, etc.), manual assessments (including palpation, joint glides, and neurological assessment), gait analysis, and special tests brought from the field of human physiotherapy. This allows the therapist to tease out the specific structure and tissue type causing the impairments.The therapist evaluates the presenting complaint, subjective information from the owner, and objective assessment carried out during the examination to create a problem list. Each item on the problem list is addressed in the plan of care. Therapeutic plans generally involve a combination of manual therapies (joint mobilizations and soft tissue mobilizations), physical modalities (laser, therapeutic ultrasound, e-stim, shockwave), and therapeutic exercises. The modalities are generally used to prepare the tissues for the manual therapies and therapeutic exercises. Physical modalities should never be the sole therapeutic method applied to any patient.Therapeutic exercise plans are based upon the weight bearing status of the patient, with early interventions focusing upon functional weight bearing exercises, later progressing to functional strengthening exercises. All exercise plans incorporate proprioception, balance, strength, flexibility, and endurance. Exercise equipment includes physioballs (shaped as rolls, peanuts, eggs, donuts and balls), cavaletti poles, therapy band, rocker/wobble boards, and treadmills.Physical modality parameters are chosen based upon the acuity of the injury. They are used to prepare the tissues for additional therapy and can generally be applied by trained veterinary nurses. The most commonly used physical modalities include neuromuscular electrical stimulation, laser, therapeutic ultrasound, extracorporeal shock wave therapy, and

ice/compression units.When the post-operative TPLO patient presents to rehabilitation in our practice, the patient is still recovering from anesthesia. We assess the patient thoroughly, and using the objective data, create a problem list. For the typical TPLO patient this list will appear as follows:

Pain, Muscle Atrophy, Skin Incision, Joint Swelling, Osteotomy, Implant, Decreased ROM: stifle and tarsus, Hip Flexor Shortening. From this problem list, we create a narrative or assessment: 9 year old M/I Golden Retriever with 4 week history of LPL lameness presents immediately post-op with pain, atrophy of the muscles of the thigh and crus (mild), skin incision (staples), joint swelling with heat, radiographic evidence of osteotomy repaired with plate and screws, decreased ROM stifle (loss of approximately 25 degrees of flexion compared to R side) and tarsus (loss of 25-30 degrees of flexion compared to R side), loss of flexibility in hip flexors (moderate compared to R side).

This assessment provides the framework from which the therapist creates the treatment plan, based upon the **functional** goals for this patient. We may wish for 'normal' ROM for all joints in our patients, but for many, this is not a functional or achievable goal. For the 9-year old Golden Retriever in this example, moderate OA in hips, stifles and tarsi might preclude his obtaining 'normal' ROM. The therapist's goal for this patient is to reach ROM's that are functional and realistic. In our example here, the functional goals would be:

Pain control, symmetrical muscling, healed skin incision, elimination of joint swelling, healed osteotomy, functional ROM in stifle and tarsus, and functional flexibility at the coxofemoral joint.The treatment plan to achieve these goals will address each goal separately. Pain control will be addressed through use of TENS and cold compression. These will be applied immediately post operatively. Once pain is controlled, muscle atrophy can be addressed using NMES to create co-contractions of the quadriceps group and the hamstrings simultaneously, so no joint motion occurs during this acute phase. The skin incision is treated with laser daily to speed healing, and cold compression is applied to prevent swelling and pain. Joint swelling is addressed via manual therapies, specifically Grade 1-2 joint mobilizations. NMES and laser are used to decrease swelling as well. The osteotomy is treated via extracorporeal shock wave therapy prior to extubation, repeated at the time of suture removal, and again at the 4-week post op visit when initial radiographs are obtained. Weight bearing across the osteotomy is encouraged via early weight shifting exercises.Range of motion issues are treated using manual therapies. The stifle is treated with Grade 1-2 joint mobilizations until the swelling and discomfort are resolved. Grade 2-3 mobilizations are then applied as needed. Therapeutic exercises to increase ROM include work over cavaletti poles. Tarsal ROM issues in TPLO dogs can be more challenging to resolve due to their often long-standing nature. Here, Grade 3-4 joint mobilizations are employed to gain joint capsule lengthening. Hip flexor shortening is treated via stretches and soft tissue mobilization techniques to the iliopsoas, tensor fascia latae, and rectus femoris.In conclusion, treating post-operative TPLO patients requires a thorough evaluation of their orthopedic as well as soft tissue impairments, creating a problem list, generating a list of functional goals for each of the impairments, and carrying out a treatment plan that addresses each of the goals. The temptation is to look for 'protocols' to treat these commonly-seen patients, however, each patient recovers at their own rate, and they do not 'read the book' on how fast they are 'supposed' to reach each level of recovery. Creative problem solving, attention to detail, and focusing upon creating and meeting goals that are functional for each patient will result in superior results.

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PSYCHOLOGICAL TRAUMA IN ANIMALS

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PSYCHOLOGICAL TRAUMA IN ANIMALS

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POSTTRAUMATIC STRESS AND PTSD

Psychological trauma in humans has been well-described for several decades. More recently, a rapidly growing body of research provides extensive evidence supporting the existence of psychological trauma and its characterization in nonhuman animals. Although posttraumatic stress disorder (PTSD) has received the most attention from both the public and researchers, a key aspect of psychological trauma is that posttraumatic psychopathology includes far more than PTSD, in 2 senses. In the first sense, PTSD constitutes the most severe outcome on a continuum of fear-based responses to a potentially traumatic experience (PTE). At the other end of this continuum mild posttraumatic fears can be found, and in between are all levels of severity of fears as well as additional important psychological changes along with the fears (such as intrusive memories, flashbacks, nightmares, and more). The diagnosis of PTSD is made only if an individual exhibits a certain number of symptoms from each of 4 quite well-defined symptom clusters over a certain period of time. As specified in the **Diagnostic and Statistical Manual of Mental Disorders** (DSM 5), in addition to the history of exposure to a traumatic event, the 4 symptom clusters that distinguish PTSD from other posttraumatic psychological disturbances are: (1) **reexperiencing** the trauma through intrusive memories, dissociation, flashbacks, and nightmares; (2) **avoidance** of anything reminiscent of the traumatic event; (3) **negative alterations in cognition and mood**; and (4) **hyperarousal** symptoms such as hypervigilance and increased startle response (not present before the trauma). The criteria for children 6 years and younger – which likely have greater applicability to animals – require fewer symptoms in each of the diagnostic clusters.One of the most important lessons obtained from research is that most survivors of single, discrete traumatic events do not develop **any** form of psychopathology. People who experience even the most harrowing of traumatic experiences are likely to experience at least short-term distress but do not develop PTSD or any other posttraumatic psychiatric disorder. The most common trajectory is for recovery over time. Only a minority of individuals develop distress and functional impairment that rise to meet the criteria for one or more of the psychiatric disorders. Another important development in recent years is the recognition that many people exposed to a PTE show enough of the symptoms of PTSD to cause distress and functional impairment but too few symptoms to meet the diagnostic criteria for PTSD. In fact, research shows that the number of people in this category after trauma outnumber the people who receive the diagnosis of PTSD. Accordingly, numerous researchers feel that the more appropriate term for this continuum of posttraumatic responses is "posttraumatic stress", or PTS (see Figure 2). Here, PTSD would be the most severe form of PTS. Forms of PTS that fail to reach the threshold for a diagnosis of PTSD are referred to as "subthreshold PTSD" (also, "partial PTSD"). This conceptualization of PTS takes on even greater importance as we discuss posttraumatic conditions in animals, where meeting certain PTSD diagnostic criteria is often difficult to confirm (e.g., flashbacks and nightmares). In this way, we can refer to animals experiencing PTS without the concern of whether they strictly cross the diagnostic threshold for PTSD.

The second sense of the assertion that posttraumatic psychopathology extends well beyond PTSD is that PTSD does not capture the full spectrum of posttraumatic disorders. Posttraumatic disorders other than (or coexist with) PTSD include specific phobia, generalized anxiety disorder, and depression, among others.

Current research has identified the primary physiological system involved in PTS to be the hypothalamic-pituitary-adrenal (HPA) axis, which appears to undergo pronounced and persistent dysregulation. Studies of PTSD show that fear is the key emotion involved in the disorder and the adaptive functioning of fear conditioning, with the capability to distinguish between safe and unsafe stimuli and facilitate identification of danger, fails this disorder. Rather than a normal level of alertness with relaxed attention, individuals with PTSD have an elevated baseline of arousal: the individual suffering from PTSD continues to function in a "red alert" status of readiness, behaviorally primed for another stressful event. To severely affected individuals, almost every place becomes perceived as unsafe, resulting in a loss of one's sense of security.

As mentioned above, a large body of research provides compelling evidence in support of the notion that animals experience psychologica trauma. But do they experience PTSD as it occurs in humans? In the laboratory, a large number of animal models of PTSD have demonstrated phenomenology that closely resembles that of PTSD in humans. However, the subjective experiences of some symptoms such as flashbacks, nightmares, and intrusive recollections cannot currently be ascertained. Most animal models of PTSD are based on exposure

to stressors that the victim cannot control, that are unpredictable, or are both.In addition to the purported experimental models of PTSD, a few reports of proposed naturally occurring cases of PTSD in animals have been published.

l Traumatic stress disorder was reported in an adult female wolf (**Canis lupus**) born in the wild and then placed into captivity. The wolf showed symptoms similar to those of humans with PTSD, and included generalized fear, avoidance, hypervigilance, arousal, and exaggerated startle reactions.

l PTSD was reported in 2 sanctuary-housed female chimpanzees who had previously sustained prolonged captivity and biomedical experimentation. The animals exhibited a wide array of signs, including intense screaming, self-injurious behaviors, stereotypic rocking, trance-like states, ritualistically arranging each piece of food in a circle around oneself, sudden and unpredictable aggression, emotional instability, hypervigilance, attacking one's own hand or foot as though it did not belong to him/her, self-isolation, and hitting oneself continually in the head.

l Wild elephants showed signs that were interpreted as resembling PTSD symptoms and meeting the diagnostic criteria for PTSD, such as abnormal startle response, depression, unpredictable asocial behavior, and hyperaggression.

l Several anecdotal reports describe signs of posttraumatic stress in canine and feline survivors of Hurricane Katrina. Signs reported in the animals were severe personality or temperament changes, new phobias, chronic chewing or paw licking, and depression; trembling, excess salivation, pacing, aggressive behavior, loose stools, vomiting, lack of appetite, elimination in the house, avoidance of people, and twitching during sleep; and indelible fear of storms as well as nervousness, fear, or aggressive behavior in response to events reminiscent of the trauma, such as heavy winds, rain, or rushing water.

l In a study of previously traumatized chimpanzees (traumatic events included maternal separation, social isolation, biomedical experimentation, or similar experiences), researchers used PTSD diagnostic criteria adapted for children and determined that 44% of chimpanzees in sanctuaries met the set of alternative criteria for PTSD, compared with 0.5% of chimpanzees in the wild.

l Most recently, popular media accounts and a few scientific reports have described clinical signs in military working dogs (MWDs) which closely resemble signs seen in human PTSD. Extreme behavioral changes have been observed in an estimated 5 percent of MWDs after exposure to combat and violent events in Iraq and Afghanistan. The key to the diagnoses was that the dogs had not displayed symptoms prior to, or earlier in, deployment to war zones.

POTENTIAL CAUSES OF PSYCHOLOGICAL TRAUMA IN ANIMALS (CAUSES OF SEVERE STRESS)

Many sources of severe stress – which have the potential to cause psychological trauma – are relatively common in animals. These include:

1. Abuse – physical or emotional in nature

2. Aversive confinement – such as in prolonged shelter confinement and in CBEs

3. Multiple re-homing – involves repeated disruption of life events and social relationships, preventing the establishment of a secure base and sense of stability

4. Hoarding – extreme stress due to competition for scarce resources

5. Natural disasters – loss of home environment and social bonds, often including physical trauma

6. Fighting – organized dogfighting involving abusive treatment, training stress, severe physical injury that is commonly treated by the dogs' owners without veterinary services

7. Racing – Greyhound dogs and racehorses, often severely stressed

8. Forced work – for example, sled dogs, animals in entertainment (circus acts, movies and television, marine animal parks) – may be pushed beyond their limits

9. Service and Military duty – exposure to combat and explosions, search and rescue work, police work

10. Laboratory research and testing – stress in experiments designed to cause distress as well as "routine" fears associated with laboratory confinement and manipulations

11. Physical trauma and injury – wide variety of adverse physical conditions

TREATMENT OF PSYCHOLOGICAL TRAUMA

Recommendations for treating psychological trauma in animals have not been adequately developed. Research is lacking, and the variety of traumas suggest individualized treatment programs are likely to be more effective than a single approach to trauma in general. Currently we are relegated to simply treat the signs, such as fears and phobias, with standard behavioral therapeutic approaches. Future research will determine the best methods for the individual types of traumas.

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DIAGNOSTIC IMAGING

IMPROVE YOUR INTERPRETATION OF RADIOGRAPHIC PULMONARY PATTERNS IN THE CAT

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IMPROVE YOUR INTERPRETATION OF RADIOGRAPHIC PULMONARY PATTERNS IN THE CAT Lorrie Gaschen, PhD, DVM, Dr.med.vet,, DECVDI

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Introduction

After performing a systematic review of the entire thorax, careful scrutiny of the lung can be one of the most challenging parts of analyzing a thoracic radiograph. Using a practical approach to determine if the lung is abnormal or not then making a decision on whether the air space, the airway is important. Also important to realize is that most all diseases cross the borders of all parts of the lung.

Are patterns important?

The principles of the pulmonary pattern are important, but a lot of stress can be associated with trying to determine the correct pattern so that one can make the correct diagnosis. A better approach is to determine if the main opacity of the lung is in the air space or the airway. Most diseases are in one or the other. In between is the interstitium and the interstitium is often involved anyway. Pure interstitial disease is really only sure when structured nodules are present. Unstructured interstitial disease is often very difficult to diagnose as a sole abnormality. Determining if disease is in the air space or airway is the best way to set a course of action. By creating a best course of action based on which compartment is most affected and then re-assessing the radiograph at an appropriate time point following treatment is good medical practice.

The normal lung

The normal feline lung is rather lucent with few linear markings, mostly visible centrally. The periphery of the lung is almost devoid of vascular markings. Thinner body condition makes the lung look hyperlucent and bronchial and vascular markings may be more visible in the periphery. The opposite is true for obesity where the lung has a hazy interstitial appearance due to the opacity of superimposed fat. Both situations make the lung challenging to assess and emphasis on the clinical signs is important for interpretation.

Air space disease

The alveoli are filled with air and this allows the vessels and bronchial structures to be sharply marginated and therefore well visible. These structures are extremely small in the periphery of the feline lung and this is the reason the periphery is so lucent. When an increased opacity is identified, the first duty is to determine if it is effacing the border of any of soft tissue structure. The margins of the heart, pulmonary vessels, caudal vena cava and diaphragm should be scrutinized first. If any one or more of those margins are blurred, the pulmonary opacity is likely in the air space: it replaces air with soft tissue and the air no longer outlines soft tissue structures, making them less visible.

Air space disease may be lobar. If the right middle lobe is homogeneously soft tissue opaque and is the only lobe affected, then atelectasis due to lower airway disease is the likely cause. Aspiration pneumonia is rare in cats and would only be diagnosed if there is a clinical history of vomiting or regurgitation. If the clinical history fits with best with increased respiratory rate and wheezing, then atelectasis is the diagnosis.

Other radiographic features of air space disease are air bronchograms, consolidated lobes with lobar sign, and patchy opacities that silhouette borders of vessels, heart and diaphragmatic contours. Lobar consolidation is when the entire lobe is homogenously soft tissue opaque and not reduced in volume, with our without air bronchograms. It is usually due to pneumonia, neoplasia or contusion. Atelectasis is collapse of the lobe due to pleural space disease or bronchial obstruction. The lobe is opaque and there is a decreased volume, and mediastinal shift of the heart to the affected lobe

Common disease categories causing air space disease are pneumonia, edema, hemorrhage, atelectasis, infection, allergic inflammatory disease, and some neoplasia.

Multifocal, ill defined, patchy soft tissue opacities that obscure the airspace and vessels in their surrounding are often due to infection or edema. Fungal pneumonia and cardiogenic edema are the most common of these, but neoplasia and contusions also have this appearance. Histoplasmosis can also have a patchy ill-defined mixed or airspace pattern as can cardiogenic edema. Other causes of infectious pneumonia are mycobacterial, cryptococcal, blastomycosis, aspirgillosis, toxoplasmosis, paragonimus and aleurostrongylus.[1] Lipid pneumonia is less common but consistently seen in cats. Radiographic abnormalities in aleurostrongylus infection are dependent on severity and duration of infection. Early changes of bronchial thickening and small, poorly defined nodules progress to a generalized alveolar pattern in severe cases. After partial resolution of the alveolar pattern, an unstructured, patchy interstitial

pattern develops.**Airway disease**

Lower airway disease can appear radiographically normal or have varying severities of airway pattern. Tracing the trachea to the carina and then tracing each main bronchus of each lobe should be performed. Two-thirds of the way out from the carina, the visualization of the bronchial walls and vessels should slowly disappear. If larger numbers of branching structures are visible, then an airway pattern is present. However, clinical signs are not always present. The clinical signs of airway disease may wax and wane, but chronic airway patterns are persistent on thoracic radiographs, regardless of clinical activity. This is where reader bias can sway the importance placed upon the presence of an airway pattern, or even lead the reader away from other abnormalities due to tunnel vision. [2] Airway disease is usually due to allergic airway disease, asthma and heartworm infection. A recent study confirmed that the most common radiographic abnormality is a bronchial pattern, but an unstructured interstitial pattern can be present in many cats. More than half of the cats in that study had lung hyperinflation also. [2] Bronchiectasis can be identified in a smaller number of cats. Right middle lobar atelectasis can be seen, as can small nodules throughout the lung and represent mucous plugs with granuloma formation.

Severe inflammatory lower airway disease can lead to hyperinflation with a flattened diaphragm. The bronchial pattern can be mixed with small nodules due to mucous plugging and exudates.

Interstitial lung disease

Primary Neoplasia

Primary pulmonary neoplasia is relatively uncommon in cats and generally has a poor prognosis. Radiographically it is typically a solitary or multiple masses, or, a disseminated lung pattern or lobar consolidation that looks like pneumonia. Adenocarcinoma may be come cavitated. Bronchoalveolar cell carcinomas and squamous cell carcinoma are usually diffuse in the lung. Most pulmonary tumors are in the caudal lobes. Adenocarcinoma is reported as the predominant tumor type, but shares many features with less common tumor types. Prevalence of suspected intrapulmonary metastasis was higher than in previous radiographic studies of cats with lung tumors. [3]

Metastatic neoplasia

Metastatic neoplasia generally present as multifocal small round soft tissue pulmonary nodules. In cats, lung-digit syndrome is an unusual pattern of metastasis that is seen with various types of primary lung tumors, particularly bronchial and bronchioalveolar adenocarcinoma. Tumor metastases are found at atypical sites, notably the distal phalanges of the limbs; the weight bearing digits are most frequently affected, and multiple-digit and multiple-limb involvement is common.[4]

Pulmonary Fibrosis

Pulmonary fibrosis is a progressive fatal interstitial lung disease that is often idiopathic, occurs in multiple

species, and may be caused by a number of inciting factors. A recent study of nine cats showed that all patients had a broad range of radiographic characteristics that included broncho-interstitial pattern, alveolar pattern, pulmonary masses, pulmonary bullae, pleural effusion, and cardiomegaly. [5] Cats in that study with echocardiographic studies had characteristics that included right ventricular dilation and hypertrophy and pulmonary arterial hypertension interpreted to be secondary to primary lung disease. Cats with pulmonary fibrosis have highly variable radiographic characteristics and that these characteristics may mimic other diseases such as asthma, pneumonia, pulmonary edema, or neoplasia. [5]

Vascular Disease

Heartworm disease can cause enlarged pulmonary arteries. However, the pulmonary findings may also be rather unremarkable in infected cats. An airway pattern is often present in most cases as well. Cardiac abnormalities are not typical.

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43RD WORLD SMALL ANIMAL VETERINARY ASSOCIATION CONGRESS AND 9TH FASAVA CONGRESS

WSV18-0064

WSAVA DENTAL GUIDELINES

COMMON FELINE ORAL PATHOLOGY DIAGNOSIS AND TREATMENT

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Learning objective: Overview of cats dental oral pathologies will be presented. Most oral problems cause pain and infection, therefore appropriate diagnosis should be followed by management of the diagnosed diseases. For cats the focus will be on inflammatory diseases.

CAUDAL STOMATITIS is a severe inflammatory reaction of the oral tissues of cats. It is a **clinical diagnosis** of inflammation and proliferation of the gingiva and oral mucosa. Specifically, it is inflammation associated with the caudal mouth (mucositis), which is the delineating factor between caudal stomatitis and periodontal disease.

Multiple etiologies may exist that, either singularly or combined, create the inflammation. Possible causative agents include an inflammatory response to plaque bacteria, viruses (FCV), **Bartonella Henselae** infection, or altered immune status (FeLV or FIV).

Caudal Stomatitis is a clinical syndrome and does not indicate a specific etiology or diagnosis. Diagnosis is made by visual inspection of the oral cavity. Diagnostic tests to further define the disease should minimally include: dental radiographs, a minimum data base (CBC, chemistry panel, t4, U/A) to evaluate for underlying and/or concurrent systemic health problems, and evaluation of FeLV/FIV status. A biopsy should be taken and submitted for histopathology, especially if the inflammation is asymmetrical or otherwise atypical, or if radiographic findings are suspicious for neoplasia.

Surgical therapy

Controlling inflammation is the key to management of this disease process. Therefore, any tooth affected with inflammation from any cause should be extracted. All remaining teeth must receive strict homecare and routine professional cleanings to keep inflammation at bay. However, since the majority of patients have widespread inflammation, the most successful long-term treatment for cats with chronic gingivostomatitis is the COMPLETE extraction of all premolars and molars including the periodontal ligament as well as smoothing the alveolar bone. An additional step taken by many veterinary dentists is to perform careful aveloplasty to remove periodontal ligament remnants, which has anecdotally improved success rates.

Extraction of the canine and incisor teeth is indicated when the inflammation extends to include the gingiva surrounding them. Post-operative dental radiographs **must** be exposed to document complete extraction of all tooth roots.

Medical therapy

In cases where owners are reluctant to have multiple extractions performed early in the course of treatment, medical management may be attempted to reduce bacterial load and inflammation. The majority of the products utilized are oral medications, which require daily to twice daily administration. This is difficult to achieve in cats in general, and the oral pain and inflammation only serves to complicate matters. Finally, many of the products have significant side effects.

Systemic antibiotics may result in some improvement in the amount of oral inflammation. However, this is generally temporary at best, and most patients will relapse even during the course of antibiotic therapy. Rinsing with a. 0.12% Chlorhexidine gluconate solution may also be beneficial in some cases.

Corticosteroids are by far the most commonly used and effective drugs for immune modulation. However, long-term use of corticosteroids may have detrimental effects such as the induction of diabetes mellitus and opportunistic infections. Use the lowest effective dose and monitor biochemical values on a regular basis. Injectable treatment (methylprednisone 10-20 mg SC) is usually recommended initially, due to the degree of oral pain. This typically results in clinical improvement within 24-48 hours, and lasts for 3-6 weeks.

Cyclosporine A has been purported as an immunosuppressive drug for cats with chronic gingivostomatitis. Some have promoted as an alternative to extractions in order to avoid the use of glucocorticoids. However, this author prefers to withhold its use to those cases where additional medical management is necessary post-extractions.

There is scant information which supports its use other than one unpublished veterinary study, which showed efficacy in cases refractory to extractions. However, it may provide an alternative to long-term steroid therapy.

Feline interferon. There is currently significant interest in the use of this product for caudal stomatitis. It is reported to not only provide an antiviral effect, but to also provide an immunomodulatory effect and bring about a return to normal local immune response. The preferred method at this point is to inject 5 MU intralesional (often at the time of extractions) and then follow this up with the remainder of the vial (5 MU) diluted into 100 cc of sterile saline and administered Per Os by the owner at a dose of 1 ml once daily for 100 days.

TOOTH RESORPTION (TR)

Completely subgingival TRs (those that have not progressed to the crown of the tooth) likely cause no discomfort for the patient. This presumption is based on the fact that similar lesions in humans are non-painful. Once lesions progress to the crown of the tooth they

are typically very painful, however cats rarely show overt clinical signs. It is possible that the tissue filling the defect may provide some protection from sensitivity.

Most TRs are quite large before they become clinically evident. Therefore, it is very important to perform a thorough oral exam on all cats. Visualization of a resorptive defect near the gingival margin is almost diagnostic for a TR. The vast majority of feline patients afflicted with TRs will show no outward clinical signs. However, patients have been presented for oral pain, anorexia, ptyalism, lethargy, depression, dysphagia, and halitosis.

Type 1

The lesions are first clinically evident on the crown at the gingival margin when the internal resorption reaches the enamel. The gingiva surrounding the teeth with type 1 lesions is usually affected with a significant inflammatory problem such as L/P stomatitis or periodontal disease. In cases of periodontal disease, it is very common to have calculus covering the lesion. This calculus must be removed to properly diagnose the lesion. The clinically visible defect typically indicates a much larger subgingival defect (tip of the iceberg). Hyperplastic inflamed gingiva also often conceals the defect.

Type 2

Type 2 TRs are usually associated with only localized gingivitis on oral exam, in contrast to the more severe inflammation due to periodontal disease or gingivostomatitis seen with type 1 lesions. Type 2 TRs often begin just below the gingival surface near the cemento-enamel junction close to the gingival margin, or "neck" of the tooth. Visualization of a defect on the tooth surface or of gingival hyperplasia onto the crown surface is indicative of a TR. The lower third premolar is commonly the first tooth affected in these cases, however canines can also be affected without other teeth being involved. Cats with a type 2 TR will generally have more than one lesion and are at increased risk for developing additional lesions.

Management

Restoration of any TR carries a very poor prognosis because the odontoclasts remain present under the restoration, and therefore the resorptive process continues. In short order, usually around 6 months, the restoration will be lost and the pain and inflammation will recur. In addition, the visible lesion normally represents only a small part of the actual pathology (i.e. tip of the iceberg).

Treatment of choice for teeth with TRs is extraction. Recently, crown amputation has been suggested as an acceptable treatment option for advanced type2 lesions. Crown amputation can only be performed on teeth with radiographically confirmed type 2 TRs which show no peri-apical or periodontal bone loss, with roots which are being completely resorbed. Crown amputation should not be done for teeth with: type 1 TRs, radiographic or clinical evidence of endodontic or periodontal pathology, inflammation, or infection. It should also not be performed in patients that have any evidence of inflammation in the caudal tissues between the upper and lower molar teeth, or that are known to be positive for retrovirus. Those practitioners without dental radiology capability SHOULD NOT perform crown amputation. In these cases, the teeth should either be fully extracted or the patient referred to a facility with dental radiology.

EOSINOPHILIC GRANULOMA COMPLEX (EGC) is a group of conditions which share a common etiology, as well as some histopathological features. While these lesions have been reported in dogs (especially Siberian Huskies and Malamutes and Cavaliers), they are much more common in cats. The discussion in this section will relate to cats, although the disease process is similar in either species. The true etiology of these conditions is unknown. Local accumulation of eosinophils (and their release of inflammatory agents) is thought to initiate the inflammation and necrosis seen in most of these lesions. The presence of eosinophils suggests that these lesions are secondary to an immune-mediated or hypersensitivity reaction.

Management

The acute disease process is best treated with corticosteroids. However, corticosteroids should not be used for long-term disease control, due to the significant systemic side-effects. The typical initial protocol is prednisone 2 mg/kg q 12 hours for 3–4 weeks. Other corticosteroid options include intralesional triamcinalone (3 mg weekly) or methyl prednisone injections (20 mg q 2 weeks). Author prefers bethamethasone (diprophos) injections. Antibiotic therapy is required in some cases to induce remission or to treat secondary infection. In addition, there are cases that appear to respond to antibiotic therapy alone as has doxycycline at 10 mg/kg PO q24h.

Many cases remain idiopathic and require lifelong therapy. Options for long cyclosporine. Cyclosporine has recently been introduced as a veterinary labeled product for atopy and appears as effective as corticosteroids for atopic dermatitis in dogs and cats. Cats should be treated for 60 days with 25 mg cat (4.9 to 12.5 mg/kg), given 2 h before a meal. It has also been proven to be an effective medication for longterm therapy of oral eosinophilic diseases. In addition, a lower incidence of severe side effects may be expected in comparison to steroids. This is especially valuable in cases requiring long-term therapy.

Surgical removal of these lesions has been performed

with some success, including laser and cryosurgery. Finally, radiation treatment has been used effectively in some cases.

PERIODONTAL DISEASE

Periodontal disease is a very common problem in veterinary patients. It has numerous severe local and systemic ramifications, however outward clinical signs occur only **very** late in the disease course. This means that it is significantly underdiagnosed, and even when recognized and treatment recommended, clients are reluctant to comply as they do not perceive the problem. Regardless, proper and prompt therapy of periodontal disease is beneficial for the health of the patient as well as financially for the practice.

Juvenile gingivitis occurs in young cats around the time of permanent teeth eruption and is associated with marginal and free gingiva inflammation circumferentially. This is inflammation of the gingiva during and just after tooth eruption and may be accompanied by persistent deciduous dentition. FOT The gingival bleeding index is II or III so bleeding may occur on probing or spontaneously. Thick plaque deposition is present but gingival probing depth non necessarily exceeds 1mm. This is self limiting in most cases, however home care (brushing or chlorhexidine rinses are recommended to decrease the inflammation. If the condition does not resolve in a short period of time, additional diagnostics and therapy is recommended as this could tend to switch into juvenile periodontitis.

FELINE OROFACIAL PAIN SYNDROME (FOPS)

Feline orofacial pain syndrome (FOPS) is a pain disorder of cats with behavioural signs of oral discomfort and tongue mutilation. FOPS is suspected to be a neuropathic pain disorder and the predominance within the Burmese cat breed suggests an inherited disorder, possibly involving central and/or ganglion processing of sensory trigeminal information. The disease is characterised by an episodic, typically unilateral, discomfort with pain-free intervals. The discomfort is triggered, in many cases, by mouth movements. The disease is often recurrent and with time may become unremitting - 12% of cases in this series were euthanased as a consequence of the condition.

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NAVC SHORT TOPICS FROM EXPERTS

HOW I TREAT POST-OP FHO WITH PHYSICAL REHABILITATION

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NAVC SHORT TOPICS FROM EXPERTS: HOW I TREAT POST-OP FHO

WITH PHYSICAL REHABILITATION

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Canine rehabilitation is the application of physiotherapeutic techniques to evaluate and treat musculoskeletal impairments in our canine patients. It incorporates the use of objective outcome measures (goniometers, girthometers, etc.), manual assessments (including palpation, joint glides, and neurological assessment), gait analysis, and special tests brought from the field of human physiotherapy. This allows the therapist to tease out the specific structure and tissue type causing the impairments.The therapist evaluates the presenting complaint, subjective information from the owner, and objective assessment carried out during the examination to create a problem list. Each item on the problem list is addressed in the plan of care. Therapeutic plans generally involve a combination of manual therapies (joint mobilizations and soft tissue mobilizations), physical modalities (laser, therapeutic ultrasound, e-stim, shockwave), and therapeutic exercises. The modalities are generally used to prepare the tissues for the manual therapies and therapeutic exercises. Physical modalities should never be the sole therapeutic method applied to any patient.Therapeutic exercise plans are based upon the weight bearing status of the patient, with early interventions focusing upon functional weight bearing exercises, later progressing to functional strengthening exercises. All exercise plans incorporate proprioception, balance, strength, flexibility, and endurance. Exercise equipment includes physioballs (shaped as rolls, peanuts, eggs, donuts and balls), cavaletti poles, therapy band, rocker/wobble boards, and treadmills.Physical modality parameters are chosen based upon the acuity of the injury. They are used to prepare the tissues for additional therapy and can generally be applied by trained veterinary nurses. The most commonly used physical modalities include neuromuscular electrical stimulation, laser, therapeutic ultrasound, extracorporeal shock wave therapy, and

ice/compression units.When the post-operative FHO patient presents to rehabilitation in our practice, the patient is still recovering from anesthesia. We assess the patient thoroughly, and using the objective data, create a problem list. For the typical FHO patient this list will appear as follows:

Pain, Muscle Atrophy, Skin Incision, Ostectomy, Decreased hip ROM, Hip Flexor Shortening. From this problem list, we create a narrative or assessment: 9 month old Miniature Poodle with 2 week history of RPL lameness presents immediately post-op with pain, atrophy of the muscles of the thigh and crus (mild), skin incision (staples), radiographic evidence of ostectomy, decreased ROM hip (loss of approximately 30 degrees of flexion and 20 degrees of abduction compared to L side), loss of flexibility in hip flexors (moderate compared to L side).

This assessment provides the framework from which the therapist creates the treatment plan, based upon the **functional** goals for this patient. We may wish for 'normal' ROM for all joints in our patients, but for many, this is not a functional or achievable goal. For the 9-month old Miniature Poodle in this example, we know that the FHO will preclude his obtaining 'normal' hip ROM. The therapist's goal for this patient is to reach ROM's that are functional and realistic. In our example here, the functional goals would be:

Pain control, symmetrical muscling, healed skin incision, functional hip ROM, and functional flexibility at the coxofemoral joint.The treatment plan to achieve these goals will address each goal separately. Pain control will be addressed through use of TENS and cold compression. These will be applied immediately post operatively. Once pain is controlled, muscle atrophy can be addressed using NMES to create co-contractions of the quadriceps group and the hamstrings simultaneously, so no joint motion occurs during this acute phase. The skin incision is treated with laser daily to speed healing, and cold compression is applied to prevent swelling and pain. NMES and laser are used to decrease any swelling as well.Returning FHO patients to full weight-bearing can be quite challenging due to the ability of the miniature and toy breeds to ambulate on 3 legs. We address this issue by starting with the patient standing next to a stack of wooden blocks. The blocks are stacked to the approximate height at which the patient prefers to hold the paw and are then slid under the paw. Gentle perturbations are applied to encourage the patient to bear weight through the paw on the affected limb. Treats and encouragement are given whenever the paw is placed upon the blocks. As the patient becomes more comfortable with weight-bearing, the blocks are gradually lowered, encouraging the patient to slowly extend the limb toward a normal standing position. This process may be done over 7 to 10 days. TENS is

used to provide pain control during this exercise.The next exercise for these patients involves working on a rocker board or wobble board. There are many options available and each patient responds differently so the therapist must be ready to adapt to the situation. We generally start with the patient's front feet on a rocker board that is set up to rock side to side. If moving the board does not result in the affected limb being extended to the ground, the board can be switched to rocking forward and back. If this does not work, the patient can be moved to a wobble board. Finally, if all else has failed, the patient's rear feet are placed on the board and all positions are tried until success is found. Cavaletti poles are used to encourage longer weight bearing on the affected limb. This is accomplished by setting up the poles higher on the non-affected side. For this patient, with a right FHO, the poles would be set up with the left side of each pole slightly higher than the right side. As the patient steps over the pole, he must spend more time bearing weight on the right limb as he lifts the left limb over the higher end of the pole. Gradually, the height of the poles, number of poles, and complexity of the pattern are increased as the patient regains strength and range of motion.Range of motion issues are treated using manual therapies. The hip is treated with Grade 1-2 joint mobilizations until the swelling and discomfort are resolved. Grade 2-3 mobilizations are then applied as needed. Therapeutic exercises to increase ROM include work over cavaletti poles as above. Hip flexor shortening is treated via stretches and soft tissue mobilization techniques to the iliopsoas, tensor fascia latae, and rectus femoris.In conclusion, treating post-operative FHO patients requires a thorough evaluation of their orthopedic as well as soft tissue impairments, creating a problem list, generating a list of functional goals for each of the impairments, and carrying out a treatment plan that addresses each of the goals. The temptation is to look for 'protocols' to treat these commonly-seen patients, however, each patient recovers at their own rate, and they do not 'read the book' on how fast they are 'supposed' to reach each level of recovery. Creative problem solving, attention to detail, and focusing upon creating and meeting goals that are functional for each patient will result in superior results.

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ORTHOPEDIC SURGERY

LONG BONE FRACTURES: LEARNING INTRAMEDULLARY PIN & WIRE FIXATION FROM PRACTICAL CASE EXAMPLES

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Intramedullary pins and cerclage are available in most primary care veterinary practices and can be used successfully to treat selected fractures in dogs and cats. Unfortunately, when used improperly or in contraindicated scenarios, these fixation methods frequently cause severe complications including fracture disease, quadriceps contracture, mal-union, delayed or non-union, and/or infection; any of which can lead to limb amputation or euthanasia. Unexpected complications can negate their apparent affordability and, thus, lead to client dissatisfaction. Therefore, the key to successful treatment using these fixation modalities is a thorough knowledge of their indications, proper application and limitations.

Intramedullary (IM) Pin Fixation

As with external coaptation, one must consider the ability of any internal fixation method to resist the disruptive forces acting upon the fracture to be treated. We will consider each of these disruptive forces individually.

Control of Bending with an IM Pin

Intramedullary pins, because they are placed in the central axis of the bone, are very good at resisting bending forces in all planes. An IM pin's ability to resist disruptive bending forces is related to the pin's radius raised to the 4th power; thus, small changes in pin radius have a profound effect on its stiffness. In theory, a pin that completely fills the intramedullary canal would impart the greatest bending stiffness; in reality, it is not feasible or advisable to completely fill the IM canal because of the irregular shape of the bone (bones are not uniform cylinders) and the relatively thin cortical bone typically has many grossly invisible "micro-cracks" that can easily propagate into fissures or fractures if the pin is too large. As a rule of thumb, we typically select an IM pin that fills 60-75% of the IM canal at it's narrowest portion (even smaller pins, ~ 30-50% canal fill, are used when combined with external skeletal fixation or bone plating). When deciding between two sizes of IM pins, it is typically better to initially choose the smaller pin; one can always "step up" in pin size if desired, but it is not possible to "drop down" to a smaller diameter pin if the first pin was too large. As we look at other disruptive forces, you will quickly note that an IM pin is a "one trick pony" that is only capable of resisting bending forces.

Control of Rotation with an IM Pin

An IM Steinmann pin has no ability to control rotation whatsoever. Because most, if not all, long bone fractures are subjected to significant rotational forces, supplemental fixation (cerclage wire, ESF, and/or bone plate) is nearly always required with an IM pin.

Control of Axial Compression (Axial Collapse) with an IM Pin

An IM pin has little/no ability to resist axial collapse. Therefore, an IM pin either requires bony architecture (ie, a properly reduced transverse fracture configuration) or appropriate supplemental fixation to resist axial collapse.

Control of Tension with an IM Pin

IM pins have minimal ability to resist the pure tensile forces of muscle tendon unit insertions. Thus, whenever you see a patient with a fracture of a traction apophysis, either a figure of 8 tension band or a tension band plate will be required for adequate treatment of these fractures in most instances.

Other Relevant Factors for IM Pin Fixation

IM pin fixation is only beneficial when the pin can be inserted without disruption to articular surfaces and other important structures such as ligaments, nerves, etc. The radius is not amenable to IM pin fixationbecause there are no extra-articular prominences for safe pin entry/exit; in addition, the IM canal of the radius is so small that an appropriately sized IM pin imparts very little bending stability. The tibia must be pinned using a normograde technique from the proximal endwith pin entry in the extra-articular margin of bone between the medial collateral ligament and the tibial tuberosity (retrograde pinning disrupts the proximal articular surface and cruciate ligaments). The femur can be pinned using the normograde technique from the proximal end or retrograde pinning in the proximal direction though caution must be applied with the latter method. In normograde pinning, the pin is typically introduced into the lateral-most margin of the trochanteric fossa. In the retrograde technique, care must be used to direct the pin cranially and laterally as it is advanced proximally to try to achieve an exit point within the lateral-most margin of the trochanteric fossa; the ability to direct the retrograde pin's exit point is progressively less controllable by the surgeon in progressively more distal fracture locations. During retrograde pin insertion into the proximal femur, it is also important to position the hip in extension, neutral abduction/adduction and neutral rotation so that the proximal pin tip is directed away from the sciatic nerve as it exits the trochanteric fossa. The humerus can pinned normograde from the either the proximal or distal end of the bone or retrograde in either directiondepending upon the fracture location and the desired position for pin seating in the distal segment.

IM Pin Summary

Intramedullary pins are uniquely capable of resisting bending forces in all planes, but this is the extent of their abilities.Therefore, IM pinning typically requires some form of appropriate supplemental secondary or primary fixation.

Cerclage Wire Fixation

To understand the ability of cerclage wire to resist these disruptive forces, the principles of proper cerclage wire application must first be discussed.

Only for Perfectly Reduced Long Oblique or Spiral Fracture Configurations

Full-cerclage wire fixation is an encircling wire that is capable of generating significant interfragmentary compression between the 2 bone segments, but only when properly applied to LONG oblique or spiral fracture configurations. The length of the fracture line must be at leasttwice the bone diameter; fracture lines that are 3 times the bone diameter permit greater interfragmentary compression (ie, improved fracture stability). Sufficient interfragmentary compression is only achieved when the long oblique or spiral fracture configuration can be perfectlyreduced (in this instance, "close" doesn't count!). After closely studying your high quality, orthogonal view radiographs, one simple rule kept in mind can save you a lot of problems: **if you see multiple cortical fragments (especially small ones), "put down the wire and nobody gets hurt!"**.

One Cerclage Wire is Never Enough for Fracture Stabilization

A single cerclage wire is neversufficient to stabilize 2 main fracture segments because it concentrates all of the disruptive forces into this single site. When properly used to stabilize a long oblique or spiral fracture configuration, each cerclage wire adds to the interfragmentary compression that is achieved. When proper wire spacing is used (discussed below), insufficient room for a 2^{nd}cerclage wire indicates that the fracture line is not sufficiently oblique for effective use of cerclage wiring.

Proper Cerclage Wire Spacing is a Must!

Proper wire spacing is required to permit vascular inflow and outflow from the cortical bone while achieving the needed interfragmentary compression. Adjacent cerclage wires are to be spaced 1 bone diameter apart from one another. Cerclage wires placed adjacent to the tips of long oblique/spiral fracture lines should be at least ½ bone diameter from the tip of the segment. Note that if a fracture line is less than 2 bone diameters in length, it would only be possible to place a single cerclage wire when the rules of proper wire spacing are applied. A fracture line that is 2 bone diameters in length will permit proper application of 2 cerclage wires and a fracture line that is 3 bone diameters in length will permit application of 3 cerclage wires.

Use the Correct Size of Wire

In small animal orthopedic surgery, 18gauge wire is used for medium and large breed dogs, 20gauge wire is used for medium and small breeds of dogs, and 22gauge is used for most cats and toy breed dogs. Braided wire is not used because it is not possible to generate sufficient wire tension with conventional tensioning and knotting techniques.

Cerclage Wire Knots

The twist knot is the most frequently used method to tighten and secure cerclage wires. In this technique, it is important that tension be applied as the wire is twisted in order to twist the wire ends uniformly around one another like a barber pole. On occasion, one end of the wire while twist upon the other like a snake on stick; this twist knot is not secure and the wire should be replaced. When an adjacent cerclage wire is placed, the additional interfragmentary compression that is produced often causes subtle, but relevant, loosening of the first wire. For this reason, when using twist knots, do not cut the twisted wire until all cerclage wires have been applied because additional tightening cannot be achieved once the wire is cut. Twist knots are usually cut between the $3^{rd}$and 4^{th}twist. Bending the twist knot after the wire has been cut loosens the wire, so the twisted tip is usually left protruding into the soft tissues where a fibrous capsule will form around it. If there are critical neurovascular structures adjacent to the twist, the knot can be twisted as the wire is bent toward the cortical surface in attempt to minimize the loss of tension associated with bending. Single- and double-loop knots can also be used and have their own distinct advantages and disadvantages. While there are mechanical advantages (especially for double-loop knots), one of the key disadvantages is the requirement for purpose-specific instrumentation. Practically speaking, any these forms of cerclage wire fixation can be used effectively in most instances provided the principles of proper use are strictly adhered to.

Preventing Cerclage Wire Loosening

Loose cerclage wires do not impart interfragmentary compression and, worse yet, they interfere with bone healing because they disrupt blood flow in and out of the bone. In short, "loose wires kill". In order to minimize the risk of loosening, full cerclage wires should oriented perpendicular to the long axis of the bone. As previously mentioned, application of an adjacent cerclage wire can loosen a wire that was previously tight; therefore, it is vital that all wires be checked for tightness immediately prior to surgical closure. For my pre-closure "wire-looseness" check, I like to use a periosteal elevator to try to shift each wire up or down the bone. Any loose wires are re-tightened (if possible) or replaced; a little extra work here can save hours of work and agonizing in the weeks to come. In conical segments of bone, cerclage

wires are prone to migration toward the narrower region. A small diameter transfixing k-wire can be placed adjacent to the cerclage wire on the narrower side, but this k-wire may encroach upon the IM pin in doing so. Alternatively, a triangular file can be used to etch a very subtle notch in the bone into which the cerclage wire can be tightened; this notch does not need to pass around the entire circumference of the bone and should not be deep into the cortex as this could increase the risk of fracture at this site; instead, the subtle notch need only accept a small portion (~ 5-10%) of the cerclage wire's thickness at 1 or 2 sites around the bone's circumference.

Control of Bending with Cerclage Wire Fixation

Cerclage wire is able to contribute to the control of disruptive bending forces assuming perfect anatomic reconstruction of a long oblique or spiral fracture with properly placed wires. Cerclage wire should never be used without supplemental fixation because its application is, by definition, restricted to the region the of the fracture rather than distributed along the entire length of the bone.

Control of Rotation with Cerclage Wire Fixation

Properly placed cerclage wires provide good rotational stability, at least in the short term. When a long bone is subjected to repeated cycles of complex loading forces that include concurrent rotation, bending, and compression, the wires will tend to loosen especially when supplementing an IM pin (remember, IM pins really only contribute to bending stability so the cerclage wire is heavily challenged in IM pin + cerclage scenarios). Thus, IM pin + cerclage wire fixation is typically reserved for cases in which rapid bone healing is anticipated (young patients, good soft tissue health, good systemic patient health, etc), the number of cycles of load can be controlled (good patient / pet owner compliance) and the magnitude of the load cycles are not extreme (smaller patients, good compliance, 3 other healthy limbs, etc).

Control of Axial Compression (Axial Collapse) with Cerclage Wire Fixation

Properly placed cerclage wires provide good resistance to axial collapse, at least in the short term. Once again, cerclage wires tend to loosen somewhat rapidly if subjected to repeated cycles of complex loading, especially if they are of larger magnitude (ie, running, jumping, playing, stairs, falling, etc). This tendency to loosen is particularly profound when cerclage wires are used in combination with IM pin fixation because IM pins have no ability resist rotation and compression; thus, the cerclage wires are heavily relied upon with IM pin + wire fixations. Thus, it only makes sense to restrict IM pin + wire fixation to rapidly healing scenarios with a controlled number and magnitude of cyclical loads as described in the paragraph above.

SVA FELINE

STATE-OF-THE-ART LECTURE PECULIARITIES OF FELINE HYPERADRENOCORTICISM

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Introduction

Hyperadrenocorticism (HAC) is an important differential diagnosis for cats with diabetes mellitus. While there are similarities to canine HAC there are also key differences. In addition to pituitary dependent (PDH) and adrenal dependent hyperadrenorticism (ADH), sex-steroid producing adrenal tumours cause HAC and are more common in cats than dogs.

Most, but not all cats with HAC have concurrent diabetes mellitus. Up to a third of cats with HAC have extreme skin fragility. Infections of the skin and nail-beds, urinary, respiratory and gastrointestinal tract secondary to cortisol-induced immune-suppression are also common.

Cats also respond differently to dogs to adrenal function tests including ACTH-stimulation and dexamethasone suppression tests.

Approximately 80% of feline HAC cases are due to PDH. Adenomas cause 90% of PDH cases and 50-60% of ADH cases, the rest are carcinomas.

Clinical presentation

HAC occurs in older cats (mean age ~10 years.) There is no breed predisposition and males are slightly over-represented (54%). Diabetes mellitus occurs concurrently in 80% of cases.

Table 1. Clinical findings from reported cases of feline HAC

Clinical sign	% cats with this sign
Polydipsia and polyuria	81
Abdominal enlargement ("pot-belly")	61
Polyphagia	60
Skin atrophy	59
Muscle wasting	47
Weight loss	47
Lethargy	41
Alopecia	37
Skin fragility (skin tears)	32
Unkempt hair coat	30
Weakness/plantigrade stance	18
Hepatomegaly	13
Weight gain	12

About 50% of cats with diabetes mellitus and HAC have insulin resistance, but it is less severe than in cats with hypersomatotropism (acromegaly). Cats

with HAC more commonly present with weight loss, while weight gain is common in acromegaly. Alopecia is typically bilaterally symmetrical, may involve the thoracic, ventral abdominal, flank and limb regions and is patchy or generalized. Failure of hair regrowth after clipping is common. Secondary skin and nailbed infections are common as well as opportunistic bacterial and fungal) infections including urinary, respiratory tract and oral cavity infections.

Diagnosis

Haematology & Biochemistry

Changes on CBC are non-specific such as a stress leukogram and mild anaemia. On serum biochemistry hyperglycaemia (85%), hypercholesterolaemia (35%), mild to moderate increases in ALT (30%) and/or urea (30%) are the most frequent findings. Since cats have no glucocorticoid-induced isoenzyme of ALP, elevations of ALP (17%) only occur secondary to diabetes. Total thyroxine is normal or decreased (sick euthyroid syndrome). Cats with diabetes have glucosuria, and most maintain urine concentrating ability and have USG > 1.020.

Urine cortisol to creatinine ratio (UCCR)

Protocol: Two home-collected morning urine samples on consecutive days. The UCCR has high sensitivity but low specificity for diagnosis of HAC in cats. It is used as a rule-out screening test due to the high predictive value of a negative result. UCCR results do not vary with age, gender, neuter status or breed, but hyperthyroid cats have significantly higher values.

Low dose dexamethasone suppression test (LDDST)

Protocol: 0.1 mg/kg dexamethasone EV, serum cortisol measurements at 0, 4 and 8 h

In healthy animals, hypothalamic secretion of corticotropin-releasing hormone is suppressed for at least 24 h after dexamethasone administration, thereby suppressing pituitary ACTH secretion and adrenal cortisol secretion. Using this protocol, 4 and 8 h serum cortisol levels in healthy cats will be suppressed to <~40 nmol/L. Cats with HAC are resistant to pituitary suppression and have an 8 h cortisol above the reference range. Overall, the LDSST in cats has high sensitivity and moderate specificity for the diagnosis. Cats with well or poorly controlled diabetes mellitus, without HAC, have comparable LDDST results to healthy cats.

Adrenocorticotropic (ACTH) stimulation test

Protocol: 5μg/kg or 125μg/cat of co-syntropin (synthetic ACTH) IV, serum cortisol measurements at 0 and 1 h. Healthy cats do not show cortisol level increases above the reference range. However, as few as one third of cats with HAC show positive test results. Thus, with its poor sensitivity and only moderate specificity, the ACTH stimulation test should not be used as an initial diagnostic screening test.

Discrimination between PDH and ADH

High dose dexamethasone suppression test (HDDST)

Protocol: 1mg/kg dexamethasone IV and serum cortisol measurements at 0, 4 and 8 h. Suppression of >50% of baseline cortisol at 4 or 8 hours is suggestive of PDH but only occurs in approximately 40-50% of PDH cats.

UCCR with high dose oral dexamethasone suppression for diagnosis and discrimination

Protocol: Home collection of two morning urine samples on consecutive days followed on day two by administration of 0.1 mg/kg oral dexamethasone at 8 am, 4 pm an midnight then collection of a third urine sample the next morning (day 3). If the UCCR of the first two urine samples (averaged) is within reference range then HAC is excluded, while an elevated result is consistent with HAC. Suppression occurs if the UCCR of the third urine sample is <50% of baseline and this result is consistent with PDH. Less than 50% suppression means that differentiation between ADH and PDH cannot be determined[7].

Endogenous ACTH measurement

Plasma endogenous ACTH levels are expected to be increased in cats with PDH and decreased in cats with ADH. Because endogenous ACTH values in affected cats may fall within the reference range, this test cannot be used as a screening test for HAC.

Diagnostic imaging

Protocol: Ultrasonography and bilateral measurements of adrenal length and height of cranial and caudal poles. Most cats with PDH have bilaterally symmetrical adrenal gland enlargement. However, in some adrenal size falls within reference ranges or adrenomegaly is unilateral. Adrenal calcification can be an incidental finding on abdominal radiographs in healthy cats. Cats with ADH have a unilateral adrenal mass or adrenomegaly. The uninvolved adrenal is atrophied or may be of normal size. Bilateral adrenocortical adenomas and bilateral tumours of different functional type (e.g. phaeochromocytoma) occur occasionally. The adrenal glands of hyperthy-

roid cats are approximately 20% larger than healthy cats. Diabetes mellitus does not cause adrenal gland enlargement in cats, but acromegaly does.
Computed tomography or magnetic resonance imaging (brain)
Approximately 50% of PDH cases are pituitary macroadenomas that can be detected using CT or MRI.

Treatment

Trilostane
Usually causes a reduction, but not resolution, of clinical signs and insulin doses if diabetic, as well as improved ACTH stimulation test results. Doses used for treatment of feline HAC range from 10 to 30 mg per cat orally SID to BID, however lower starting doses of 1-2mg/kg/day have been suggested more recently. A 2-4 h post-trilostane ACTH stimulation cortisol measurement of 50-150 nmol/L is recommended. Adverse effects include anorexia, lethargy, weight loss, pancreatitis and hypoadrenocorticism. Trilostane is mostly well tolerated and is the most efficacious drug currently available for treatment of feline HAC.

Mitotane
Not recommended for therapy since it is far less effective than trilostane, clinical signs often progress and ACTH stimulation test results remain unchanged.

Adrenalectomy
Unilateral adrenalectomy is the treatment of choice for feline ADH. Bilateral adrenalectomy is curative for PDH but is associated with a relatively high complication rate due to poor wound healing, immunocompromise and skin fragility.

Hypophysectomy
Microsurgical transsphenoidal hypophysectomy for PDH is an increasingly reported curative treatment for PDH. Complications included oronasal fistula, soft palate dehiscence, transient reduction in tear production and recurrence of HAC due to pituitary remnants.

Radiation
Radiation therapy for PDH generally involves multiple fractionated treatments. Availability of a gamma knife or a stereotactic capable linear accelerator can reduce this to a single treatment.

Prognosis

Trilostane is the medical treatment option with the longest reported survival times. Cure can be achieved with adrenalectomy or transsphenoidal hypophysectomy if immediate surgical complications do not occur. For cats with pituitary macroadenomas, neurological signs usually improve after radiation, and some, but not all have improved signs of HAC after radiation therapy, and reduced insulin requirements or diabetic remission. Cats with diabetes mellitus may have reduced insulin requirements or diabetic remission if they respond to treatment for HAC.

Sex-steroid secreting adrenal tumours

Tumours may secrete one or more adrenal sex steroids, e.g. progesterone, oestradiol or testosterone, alone or in combination. Typical clinical presentations include clinical signs of HAC, diabetes mellitus and behaviour change (aggression). Some adrenal tumours in cats co-secrete aldosterone and adrenal sex steroids, e.g. progesterone and aldosterone. These cats can present with clinical signs of HAC as well as behaviour changes and signs of hyperaldosteronism such as weakness and hypertension. Alternatively, overproduction of sex steroids can result in presentation with signs of oestrus or virilisation of neutered males e.g. urine spraying, aggression, skin thickening and penile spines.

Diagnosis

Diagnosis is on the basis of high basal and post-ACTH stimulation sex-hormone levels, detected on adrenal sex-steroid panels and adrenal imaging. Serum cortisol levels are usually low due to lack of enzymes within the adrenal tumour to convert progesterone to cortisol, or hypothalamic–pituitary–adrenal axis suppression of cortisol by sex-steroids. Adrenalectomy of the neoplastic gland is curative with regression of clinical signs over weeks to months

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INFECTIOUS DISEASES AND GASTROENTEROLOGY (TRANSLATED INTO MANDARIN CHINESE)

MANAGEMENT OF BACTERIAL AND VIRAL UPPER RESPIRATORY INFECTIONS IN CATS

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The primary objectives of this session are to review the known bacterial and viral causes of the feline upper respiratory disease complex followed by a discussion of optimal diagnostic tests, treatments and preventions. Please see the ISCAID respiratory treatment guidelines for further information on this very important topic (Lappin et al, 2017).

Bacterial causes. Almost all cats with mucopurulent or purulent nasal discharge have a bacterial component to their disease. Primary bacterial disease is rare but may be associated with **Bordetella bronchiseptica**, **Mycoplasma** spp. and **Chlamydia felis**. **Streptococcus equi** var. **zooepidemicus** may also be associated with clinical illness in some cats. In one Morris Animal Foundation sponsored study, we showed Mycoplasmas to be more common that FHV-1 and were associated with illness. Recently it was shown that **Bartonella** spp. are not causes of rhinitis in cats. Both **B. bronchiseptica** and **Mycoplasma** spp. can be associated with bronchitis in cats. Chlamydiosis in general, is a mild infection resulting only in conjunctivitis.

If primary infections are suspected, doxycycline 10 mg/kg, PO, once daily (or divided BID) or topical administration of tetracyclines (conjunctivitis) are usually effective. Amoxicillin is the other empirical treatment recommended for management of bacterial rhinitis of < 10 days duration (Lappin et al, 2017). Cats with acute disease only need to be treated for 7 to 10 days. Most cases of bacterial rhinitis are secondary to other diseases including trauma, neoplasia, inflammation induced by viral infection, foreign bodies, inflammatory polyps, and tooth root abscessation. Thus, if routine antibiotic therapy fails, a diagnostic workup should be performed. After other primary causes are excluded, rescue drugs like quinolones, azithromycin, potentiated penicillins, or cephalosporins can be considered. Pradofloxacin has been shown to be a good choice as a rescue drug for treatment of chronic bacterial rhinitis as this quinolone is effective against anaerobes and aerobes and enhanced activity against Mycoplasma spp. (Spindel et al, 2008).

Since bacterial rhinitis leads to chondritis and osteomyelitis, antibiotic therapy may be required for days to wee should be continued for weeks in cats with chronic disease.

Viral diseases. Herpesvirus 1 (rhinotracheitis; FHV-1) and calicivirus (FCV) are the most common viral causes of sneezing and nasal discharge in the cat. If oral ulcers are present, calicivirus is most likely. If corneal ulcers are present, herpesvirus 1 is most likely. FHV-1 has now also been associated with chronic stomatitis, facial dermatitis, and endogenous uveitis. Viral rhinitis with or without secondary bacterial infection can be recurrent. FHV-1 can be documented by direct fluorescent staining of conjunctival scrapings, virus isolation, or polymerase chain reaction. Since FHV-1 DNA can be detected in conjunctival cells of approximately 25% of healthy cats, the positive predictive value of these tests in diseased cats is low. Quantitative PCR may ultimately prove to correlate to the presence or absence of disease. Currently used PCR assays also detect vaccine strains of FHV-1. RT-PCR assays can be used to amplify the RNA of FCV. However, these assays have the same problems with predictive value as those to detect DNA of FHV-1. In one of recent publications, we showed that PCR assay results for FHV-1 or **Mycoplasma** failed to correlate to treatment responses to either an anti-viral drug or an antibiotic (Zirofsky et al, 2018). Thus, performance of these PCR assays have low positive predictive value.

Feline viral rhinitis with or without secondary bacterial infection can be recurrent. There are no consistently effective primary therapies. For FHV-1, lysine at 250-500 mg, PO, once or twice daily may be helpful in some cats and has been shown to be safe but should be given as a dose, not fed with food. Lysine has been shown to be ineffective for prevention of upper respiratory tract infections in 2 separate shelter studies and so should probably not be used for this purpose.

Administration of human alpha 2b interferon at 50 U, PO, daily may help some cats with suspected chronic calicivirus or FHV-1 infection. This can now be formulated for practitioners by prescription at some pharmacies (www.roadrunnerpharmacy.com/) in the USA. Topical administration of alpha interferon in saline to the eyes of cats with conjunctivitis or the nose may aid in the management of some cats but has not been proven in a controlled study. Lysine and alpha interferon are unlikely to lead to a cure, but hopefully will lessen clinical signs of disease. Intranasal administration of modified live, intranasal FHV-1 and FCV vaccines may lessen disease in some chronically infected cats. If there is a positive response to intranasal vaccination in a cat with chronic disease, I will use this form of immunotherapy up to 3 times per year (Fenimore et al, 2015). The intranasal vaccine has been shown to potentiate cell-mediated immunity to FHV-1 better than parenteral vaccination.

Acyclovir is an anti-herpesvirus drug for use in people but can be toxic to cats and so should not be used.

Famciclovir is safer and more effective than acyclovir and is now being used for long-term therapy. One dose that has been used is 1/2 tablet of a generic 250 mg tablet (125 mg), PO, q8-12 hr. The drug is safe at up to 90 mg/kg, PO, q8hrs and so the dose should be increased if the initial response is suboptimal and FHV-1 is still suspected. However, it is now known that famciclovir is excreted in high levels in the tears for 4 hours after a dose and so topical treatment with anti-FHV-1 drugs may not be needed if famciclovir is prescribed at 90 mg/kg, PO 2-3 times daily. Administration of one dose of famciclovir (125 or 500 mg) on admission to an animal shelter was ineffective in lessening clinical signs of disease. Topical cidofovir (product for humans) can be used for the treatment of FHV-1 conjunctivitis twice daily and was effective in a controlled research project. The drug was easier to administer (twice daily) than idoxuridine or other anti-FHV-1 ocular therapies and does not cause as much irritation. This drug is available in some compounding pharmacies (www.rxfixer.com).

Many of the cats with chronic recurrent signs of disease are likely to be infected by FHV-1 or FCV. Stress reactivation of feline viral infections is thought to be common, in particular for FHV-1. All the principles of stress relief for management of feline interstitial cystitis also apply to cats with recurrent signs of URI. In a recent published study, we showed that use of a facial pheromone diffusor could lessen recurrent signs of FHV-1 in a mild stress model in experimentally inoculated cats (Contreras et al, 2017). The commercially available probiotic **Enterococcus faecium** SF-68 (FortiFlora, Purina), is a known immune stimulant in cats and feeding this probiotic to cats with FHV-1 infection lessened recurrent disease in a stress model (Lappin et al, 2009).

Feline leukemia virus and feline immunodeficiency virus can induce immunosuppression predisposing to bacterial rhinitis. However, there is no universally effective treatment. Interferon alpha as described can be tried. In addition, AZT at 5 mg/kg, PO, twice daily can be tolerated and improved clinical parameters in some cats with FIV. Both FIV and FeLV have been associated with nasal lymphoma and so if upper respiratory tract signs occur in retrovirus positive cats, this neoplasm should be excluded.

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NURSES (HILLS)

BEST PRACTICE FOR COLLECTION, TESTING AND UNDERSTANDING OF BLOOD RESULTS

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BEST PRACTICE FOR COLLECTION, TESTING AND UNDERSTANDING OF BLOOD RESULTS

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Blood Collection

Blood collection is an important part of everyday veterinary practice. Many factors are taken into consideration when deciding where to withdraw the sample. **This includes:**

- Type of animal
- Size of animal
- Medical condition / site of injury
- Amount of blood to be collected
- Tests to be performed
- Experience of person taking sample

When taking a blood sample, the procedure should be performed in an aseptic manner. This includes:

- Clipping of site
- Preparation of site with surgical scrub solutions
- Washing hands
- Sterile needle and syringe

The sample should always be a 'clean stick', otherwise haemolysis of the sample will occur which may affect test results.

The aim when collecting blood is:

- To use a vein close to the surface of the body
- To be able to gently withdraw a suitable volume of blood (to minimise damage to blood cells)
- To cause minimal disturbance and discomfort to the animal

Preferred sites for blood collection

In small animal practice the jugular vein is often the preferred site for collection. However, if performing tests requiring only a small amount of blood –e.g. glucose, PCV / TPP, peripheral veins may be used.

Canine	Vena jugulars, vena saphena and vena cephalica for central venous blood. Edge of ear (close to base for peripheral blood smears
Feline	Same as dog but avoid saphenous and cephalic veins if at all possible
Pocket Pets	Tail vein, e.g. rats, ferrets, guinea pigs etc
Avian	Jugular vein, brachial artery (for large parrots), pedal artery (large water fowl)
Equine	Jugular vein
Bovine	Jugular vein, tail vein

Collection site	Suggested needle size*
Cat and small dog cephalic or saphenous vein	23-25G
Cat and small dog jugular vein	21-23G
Medium to larger dog cephalic or saphenous vein	21-23G
Medium to larger dog jugular vein	20-21G
Horse jugular	18G

When transferring blood from the syringe to the tube steps are taken to minimise damage to the cells:

- Remove needle from syringe so that blood is not transferred through a small bore
- Remove top from tube. This applies equally to a vacuum collection tube as to a screw-top tube unless specifically stated by the manufacture. Plunging a blood filled syringe + needle into a vacuum tube and allowing it to transfer into the vacuum tube increases the possibility of haemolysis
- Gently expel the blood into the angled tube so it dribble down the side
- Replace lid securely and (in an anti-coagulant tube) gently agitate to evenly mix chemicals with blood. Do this by slowly inverting the tube every 2-3 seconds for 30 seconds

Factors during the collection that can influence sample quality

Factor	How it may affect the sample
Fear and stress of the animal Experience and capability of person collecting the sample	Adrenalin release affecting glucose and some white cell count results.
Preparation of the collection site	Contamination of sample will give false results where sample is to be cultured.
Time of day when sample is collected	Some hormone levels vary throughout the day.
Feeding before sample collection	Will give a transient hyperglycaemia and hyperlipidaemia.
Using the correct tube for the required test	Tubes are used for specific tests. E.g. blood in a Potassium EDTA tube will give a false potassium level.
Actual site of collection	Arterial gasses cannot be estimated from a venous sample.

Why it is important to conduct pre-anaesthetic blood tests?

A full patient examination performed by the Veterinarian may not be enough to detect an underlining issue and unfortunately illnesses or diseases can go unnoticed. Pre-anaesthetic blood tests can check the kidney and liver function, presence of low red blood cell (anaemia), infection or inflammatory disease (high white blood cell count), dehydration (high red blood cell count and high total protein levels).

It is important to know the names and what increased and decreased values could mean to a patient in your nursing care.

TEST	HIGH	LOW
ALKALINE PHOSPHATASE (ALPKP)	Liver disease such as Cushing's, active bone growth (seen in young pets)	
CREATININE (CREA)	Kidney disease	Overhydration
BLOOD GLUCOSE (GLU)	Diabetes, stress (seen in cats)	Liver disease, infection or certain tumours.
AMINOTRANSFERASE (ALT)	Liver Cell injury/damage	
TOTAL PROTIEN (TP)	Dehydration, inflammatory condition	Decreased liver function, blood loss, gastrointestinal loss and kidney loss
BLOOD UREA NITROGEN (BUN)	Kidney disease or dehydration	Liver disease
PACKED CELL VOLUME (PCV)	Dehydration, marrow cancer	Anaemia

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The Animal Industries Resource Centre Course materials – Certificate IV in Veterinary Nursing

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SVA PRACTICE MANAGEMENT

EVOLVING YOUR PRACTICE TO BE FIT FOR THE 21ST CENTURY

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EVOLVING YOUR PRACTICE TO BE FIT FOR THE 21ST CENTURY

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In this session we will look at how successful organizations, with strong reputations for the service they deliver, have achieved that success

What type of practice are you?

When we ask this question of a veterinary practice the most common answers describe the practice in "our" terms;

"A Veterinary Hospital" - "A well equipped General Practice" - "A Mixed Practice"

When we ask this question of our customers the most common answers describe the practice in "their" terms;

"They care about my pet" - "They are kind and considerate" - "They are convenient to get to"

"They offer appointments at times to suit me"

Our customers' answers describe the practice in terms of its values; not in terms of its facilities

Your Practice's values?

Your practice is unique

The services that you offer to your clients reflect the skills and expertise of **all** of the staff

The standards of service that you provide will reflect the values of **your** Partner(s)

So, we need to be clear about what the "core values" of your practice actually are. Take the time to try to verbalise those that are important to you, such that you can share them with your staff and your clients.

Many businesses, including vet practices will create a short phrase or sentence that sums up their values, as well as a more detailed description of just what is important to them.

What can we learn from the best?

When we think of consistently high quality customer experience, which organisations come to mind? We will all be able to quickly identify service providers we use or are aware of in our own lives that deliver high quality client care with excellent service. What exactly do they do that sets them apart from the rest?

Delivering high quality service requires three things;
- Effective leadership and management
- Effective business systems
- Effective individuals

The organisation has developed appropriate systems that support and enable its employees to deliver on it's values every time that they interact with a client. Protocols and procedures are at the heart of everything that they do.

How do you wish to be perceived?

Your clinic's Culture and Values should influence every part of your business, because otherwise we risk not delivering on our customer's expectations, because we will have set expectations that we cannot consistently deliver. A useful way to look at this is using Normann's model, which is a framework for all service business
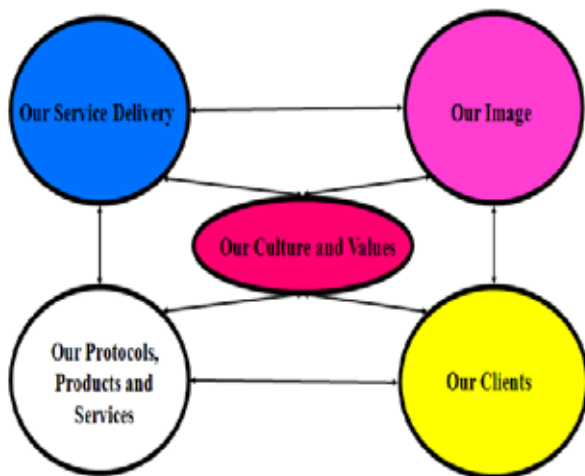
Culture and Values

The "core values" of the practice form your culture and values

Clients

Which customer groups should your values appeal to? How many of those do you have? Which customer groups would you like to appeal to? There is an old saying that you get the clients you deserve so you values must appeal to your core client base

Image

You image is the way that you look to your clients by what they say, and by what you communicate to them.

This includes the design and decoration of your clinic, your website, all forms of communication, how your staff looks, and the appearance of your clinical areas. Clients will judge you by what they see, and what you do, much more than by what you say. So, we need to ensure that every aspect of the way we look and feel to our clients reinforces our values to them

Protocols and Procedures

Your protocols and procedures document the way you plan to deliver key services. They should set out the standard of work that is acceptable to you, and guide staff in your "best practice". When developing them, you should be constantly checking that they will deliver on your values at all times.

In the veterinary profession we tend to focus on the needs of our patients, however we must ensure that we also meet the Physical, Psychological and Emotional needs of our clients too.

Service Delivery

This is the way in which we deliver our planned services day in, day out. It includes resource planning to meet our clients' needs, and ensuring we have sufficient trained staff to deliver the services we have told our clients to expect. This means that we need to recognise that as small business teams we will sometimes be restricted by the resources available and we need to have a "Plan B" so that all staff understand how we want them to cope in these times

It is also helpful to break our services down into sub-groups so that we don't forget the little but important details.
- a.   Core Services – which will include providing emergency care, providing wellness programmes, consulting services etc.
- b.   Supporting Services - such as Imaging, Ultrasound, Endoscopy, Nurse Clinics etc.
- c.   Facilitating Services – those things that provide convenience to clients such as Home visits, Home delivery, Pharmacy, Collection point, etc
- d.   People – the human resources we need such as Vets, Nurses, Receptionists, Bereavement counsellor, etc.
- e.   Entertainment – how we will help clients to enjoy their visit, such as Activities for children, magazines, waiting room TV, etc.

We can only improve our practice if we;
- ·   Make ourselves a commitment to improve our own performance
- ·   Share problems with each other
- ·   Are honest about our performance
- ·   Talk about the problems in an open manner
-

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ORTHOPEDIC SURGERY

LONG BONE FRACTURE: LEARNING EXTERNAL SKELETAL FIXATION FROM PRACTICAL CASE EXAMPLES

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ESF Nomenclature

The ESF System is comprised of many different types of devices including linear, ring and acrylic ESF devices. Among the most popular of the current generation of linear devices is the IMEX SKÔdevice and it will be the device used in our discussions and laboratories. Regardless of specific manufacturer, linear devices have 3 basic elements (Fig 1):

- 1. Fasteners (percutaneous fixation pins)
- 2. Connecting rods
- 3. Linkage devices (clamps linking the fixation pins to the connecting rods)

Fixation pins are termed either **half-**or **full-pins**(Fig 2) based upon how they are inserted.

- Half-pinspenetrate the near-skin surface and both the near- and far-cortex of bone.
- Full-pinspenetrate the near-skin surface, both cortical surfaces of the bone, and exit the far-skin surface of the limb.

The connecting rod(s), fixation pins and clamps define an ESF frame(Figs 1 & 2). Frame configuration is described by the number of distinct sides of the limb from which it protrudes ["unilateral" or "bilateral"] as well as the number of planes it occupies ["uniplanar" or "biplanar"].

- Unilateral-uniplanar (Type 1a) framesprotrude from just 1 side of the limb and are restricted to one plane. Type Ia frames (Fig 2 – left) are formed by connecting 1 or more half-pins of each main fracture segment.
- Bilateral-uniplanar (Type II) framesprotrude from 2 distinct sides of the limb, but are restricted to just one plane (typically the medio-lateral plane). Type II frames (Fig 2, right) are formed by connecting 1 or more full-pins of each main fracture segment.
- Bilateral-biplanar (Type III) framesprotrude from 2 distinct sides of the limb and occupy 2 planes. Type III frames are formed when both a Type 1a and Type II frame are applied to a bone. These are seldom used with modern ESF devices.
- Unilateral-biplanar (Type Ib) framesoccupy 2 planes, but because these frames do not protrude from 2 distinctsides of the limb (180°to each other) they are thought of as "unilateral". If the frame occupies more than 1 plane, but does not connect a full-pin in the proximal segment to a full-pin in the distal segment, it is a Type 1b. Standard type Ib frames (Fig 3) are formed when two Type Ia frames are applied to a bone.

The frame classification described above fosters accurate communication between colleagues and also provides a basic sense of frame stiffness under axial loading (Type III > Type II > Type I).

Type 1b frames are sometimes applied for the purpose of enhancing this multi-planar stiffness. Uniplanar frames are most able to resist bending forces that are applied in the plane of the frame (versus those in the plane perpendicular to the frame). As an example, a frame occupying the medio-lateral plane is less able to resist bending forces in the cranio-caudal plane. Application of multi-planar frames, therefore, imparts better multi-planar stiffness. In theory, this is best accomplished by placing the frames 90°to one another. Clinical practicality, however, dictates that frames be applied as regional soft tissue anatomy and bony cross-sectional structure warrant. As an example, Type Ib frames applied to the radius typically consist of a frame in the cranio-medial plane and a second frame in the cranio-lateral plane (Fig 3).

Type II frames are not as frequently employed as they once were. When the frames are comprised entirely of full-pins, they are called "maximal" Type II frames. A "minimal" Type II frame is comprised of one full-pin in the proximal main fracture segment and one full-pin in the distal segment, and the remaining positions are filled in with half-pins.

ExFix Pins

Smooth (non-threaded) Steinmann pins were originally used with ESF, however, premature pin loosening was a major problem and their use has been replaced by use of various threaded pin designs. Positive- versus negative-thread profile, cortical versus cancellous thread form, length of the threaded portion, and pin size must all be considered for optimal pin selection for a given bony insertion site.

Thread profile.**Negative profile threads**are cut into the pin at the expense of the core diameter. When threads are cut into the pin over its entire length, the pin loses its stiffness and is subject to bending or breakage. When threads are conventionally cut only into the end of the pin (end-threaded pin), the abrupt change in pin diameter is a **"stress-concentrator"**and these pins are predisposed to breakage at the junction of the threaded and non-threaded portions. Historically, SCATÔpins were designed such that threads engaged only the far-cortex of bone and the breakage-prone thread-shaft junction was located within the intramedullary canal, theoretically, protecting it from cyclic bending forces. In reality, these SCAT pins are seldom used with modern devices such as the IMEX SKÔdevice. The weaknesses of negative profile pins were initially overcome by introduction of fixation pins with a positive thread profile. **Positive profile threads** are raised above the

core diameter of the pin. This thread profile offers secure pin-to-bone fixation without having a breakage prone stress-concentrator. These pins were technically difficult to apply with older KE devices, but application is greatly simplified with the clamp design of the IMEX SKÔdevice. The disadvantage of these pins is their large "footprint" in available bone stock; this can be problematic in small bones or where ESF is used in combination with an intramedullary pin. Most recently, IMEX has introduced its DuraFaceÔpins that have a negative thread profile with a tapered thread run-out design at the thread-nonthreaded transition zone. This design feature eliminates the stress-concentrator issue of conventional negative-thread profile pins while adding pin stiffness when compared to a positive-profile pin of equivalent thread-diameter.

Cortical versus Cancellous thread form. A cortical thread form is used in most locations. The cortical thread form has a finer thread pattern and a greater number of threads per unit of pin length when compared to cancellous pins. Cancellous pins use their relatively coarse thread pattern and few threads per unit length to maximize purchase of very soft cancellous bone in locations where there is little cortical bone for purchase (proximal tibial metaphysis, proximal humeral metaphysis, and, in some instances, distal femoral condyle, pelvis and vertebral body). The notion that all metaphyseal bone is soft is not true; cancellous pins should notbe used in the humeral condyle, distal tibia or in the radius.

Regular versus Extended Length Pins.Extended length pins are available in end-threaded designs (for use as half-pins) and centrally-threaded designs (for use as full-pins). Extended length pins have both an increased overall pin length and increased span of threads. These pins are useful when standard thread length is insufficient to span the full diameter of bone or the soft tissue envelope is so extensive that standard length pins will not protrude sufficiently from the limb.

Linkage Devices (ExFix Pin Clamps)

The SKÔclamp offers a significant improvement over the KE clamp in terms of both mechanical performance and "user-friendliness". The mechanical performance is enhanced by its adaptation to use of relatively large diameter connecting rods as compared to the old KE clamp. The connecting rods are made of carbon fiber composite or titanium instead of stainless steel to reduce their weight. The SKÔclamp design also allows simplified introduction of a variety of pin sizes and designs including positive profile pins.

SKÔclamps have a two-piece aluminum body, a pin-gripping bolt (also known as the primary bolt) with a slotted washer and tightening nut, and a secondary bolt. The rod-gripping channel is formed by the hemi-circular cut-out shape of each half of the aluminum body. Tightening of the primary and secondary bolts allows

the SK clamp to tightly grip the connecting rod without deforming the shape of the clamp body. The clamps can either be pre-positioned on the connecting rod or can be assembled (or disassembled) on the rod at any desired location during surgical application. The gliding washer upon the primary bolt has a meniscus (slot) that enables the bolt to effectively grip a range of different pin diameters; the curvature of the meniscus corresponds to the smallest diameter pin shank that can be securely gripped by the clamp (Table 1). The back of the slotted washer has serrated teeth that engage the outer surface of the aluminum clamp body when the primary bolt is tightened. This provides a rigid lock between the fixation pin and the connecting rod. Finger tightening of the secondary bolt allows the clamp to be stabilized on the connecting rod during pre-drilling and pin insertion, but still permits the clamp to swivel slightly as the primary bolt is tightened so that the orientation of the clamp can "self-correct" to the fixation pin.

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APPROACH AND MANAGEMENT OF VIPER SNAKE BITES IN DOGS AND CATS

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Snakebites are a common problem in human and veterinary medicine. Vipers are member of the family **Viperidae**, a group of snakes found all over the world, except in Madagascar and Australia.

The viper family includes 223 species of venomous snakes that are divided into 2 main subfamilies: pit vipers (subfamily Crotalinae), true vipers (subfamily **Viperinae**). Vipers are characterized by a pair of long, hollow, venom-injecting fangs. They feed on small animals and hunt by striking and envenomating their prey. Pit vipers are the largest group of venomous snakes with 151 species and are responsible for ~150,000 envenomations of dogs and cats in the United States annually [1]. The other major subfamily, the true vipers, includes 66 species, and is also known as pitless vipers. They are distinguished by their lack of the heat-sensing pit organs that characterize the pit vipers. Among them, **Vipera berus** is found from Western Europe and Great Britain to the Far East [2]. **Vipera palaestinae** (**VP**) is the most common venomous snake in the Middle East. It is responsible for most envenomations in humans and domestic animals in Israel [3]. The snake is endemic to Israel, can be found in all country parts except the desert, and has adapted to life in agricultural and suburban areas. Envenomations were reported in people, dogs, cats, horses and a ram [3,4].

The **viper's** venom contains about 30 components, 16 of which were identified, with the most important ones being proteases, hemorrhagins (metaloproteinases), amino acid esterases, phospholipase-A2, phospholipase-B and neurotoxins [5]. The hemorrhagins activity leads to endothelial cell damage, causing high vascular permeability, bleeding and fluid extravasations into inflamed tissues. Phospholipase-A2 is considered the most important component in many snake venoms. It has both pro-coagulant and anti-coagulant activities as reflected by inhibition of the protrombinase, inhibition and activation of platelet aggregation, and activation of Factor V and plasminogen [5]. The amount of venom injected in a single bite increases with increased ambient temperatures, and may reach as much as one gram (dry weight); nevertheless, bites may contain no venom at all [6].

Most of canine Vp envenomations in Israel occur between May and October and between 14:00 – 22:00, and parallel the viper's seasonal and diurnal activity. This pattern, however, may also result from increased seasonal and diurnal dog activity [4].

The onset of clinical signs following envenomation may be delayed for several hours. This phenomenon is highlighted by the fact that 40% of all severe envenomations in humans are graded as mild to non clinical signs upon admission. In addition, 20% of the bites were dry envenomations (venom free) with an additional 25% classified as mild envenomations [1]. The severity of the envenomation is influenced by species and victim factors such as the bite location, victim body mass, post-bite excitability, the species and snake size, its age and motivation and the degree of venom regeneration since last bite [1].

Risk factors for mortality in dogs included envenomation during the first summer months and low body weight (<15kg), envenomations in the limbs and identification of the snake's bite as well as shock and bleeding tendencies [3,4]. In a recent study describing envenomation of 18 cats in Israel, variables associated with mortality included lower body weight, lower body temperature and haematocrit at arrival and 12-24 hours later, and lower total plasma proteins 12-24 hours post presentation.

Most canine **VP** envenomations are in the head and neck area (80%) and less frequently in the limbs (20%) [3,4]. In a study on dogs with Vp envenomation, skin marks consistent of snakebite were identified in only 51% of the dogs [4]. The most common local signs in canine snakebite include swelling, edema and hematoma which are attributed mostly to venom hemorrhaging activity. Acute lameness with pain may appear when limb envenomations occur [3]. Reported systemic signs include anaphylactic, hemorrhagic or neurogenic shock, tachypnea, tachycardia, local lymphadenomegaly and cardiac arrhythmias [3]. To date, no specific cardiotoxin has been identified in the viper's venom, although myocardial necrosis as a rare complication was reported in dogs and horses following **VP** envenomation [7]. Cardiac injury in **VP** envenomed dogs as was reflected by increased serum cardiac troponin T and I (biomarkers of myocardial damage) [8,9]. In the study, serum cardiac troponins concentration were increased in 65% of the dogs envenomed by Vp. Dogs with increased cardiac troponin were found to have a significantly higher occurrence of arrhythmias (58% vs. 19%)[8].

The most common hematological findings in **Viper** envenomation include hemoconcentration, leucocytosis and thrombocytopenia and nucleated red blood cells in the peripheral circulation. Drastic drop of total solids in the first 24 h after envenomation is very common, as well as mild elongation of prothrombin (PT) and activated partial thromboplastin (aPTT) times and decrease in anthitrombin activity. The most common biochemical abnormalities observed include increased activities of muscle enzymes: lactate dehydrogenase, creatine kinase and aspartate aminotransferase. Additional abnormalities

included hypertriglyceridaemia, mild hyperglycaemia, hyperbilirubinaemia, hyperglobulinaemia and hypocholesterolaemia [4].

Reported complications of **Viper** envenomation in dogs include bacterial infections (clostridial or other), local necrosis, upper respiratory airway obstruction due to laryngeal edema, acute renal failure, coagulation disorders namely venom-induced consumptive coagulopathy (VICC) and death [3]. For dogs with Vp envenomation, the reported mortality rate in the literature is relatively low (3.7-6%) [3,4].

Venom-induced hemostatic abnormalities, potentially culminating in venom-induced consumptive coagulopathy (VICC), include thrombin inhibition and activation, increased fibrinolysis, hypofibrinogenemia, release of kinins, endothelial damage, and platelet aggregation, destruction and dysfunction. [Viper]-envenomed dogs commonly develop VICC, its frequency increasing over time during hospitalization. Although VICC is similar to DIC in many ways, it differs from the former by a rapid onset and resolution, different mechanism of initiation and it is not usually resulting in end-organ effects. Venom-induced hemostatic abnormalities, potentially culminating in venom-induced consumptive coagulopathy (VICC), include thrombin inhibition, increased fibrinolysis, hypofibrinogenemia, release of kinins, endothelial damage, and platelet aggregation, destruction and dysfunction.

Cats are considered more resistant to snakebites than other animals[9]. However several studies published in the last decade have shown that mortality rate cats varied between 6-22%. Although bleedings are not so common in cats, haemostatic abnormalities were reported in all cats expressed by elongated aPTT in 100% and PT in 93% of the cats. Several RF were identified including; lower rectal temperature (**P**=0.02, 35.9^0 C vs. 38.0^0 C), lower haematocrit (29 vs. 44%, **P<0.001**) upon admission and lower haematocrit 24 hrs post admission (22 vs. 33%, **P=0.001**). In our emergency department we see 2-6 cases of cat's snakebites a year, as compared to 30-40 cases of dogs with snakebite. The discrepancy between dogs and cats for the limited cases might be the caution and the ability of cats to cope with snakes, cats are not referred to medical treatment since they do not show clinical signs nor are outdoor cats. Based on our limited experience, the diagnosis of cats with snake bites is not as obvious as in dogs. As the time lag from the snakebite to admission in cats is usually longer than in dogs, the most common clinical signs are local swelling with severe hematoma, which might develop over the 24-48 h post snakebite, depression and shock. Hematological findings are hemoconcentration, thrombocytopenia, hypoproteinemia, hemolysis and consequent anemia, and the appearance of peripheral nucleated red blood cells. Disseminated intravascular coagulation also is

a common complication in cats with snakebites with dramatic elongation of the PT and aPTT but relatively slight clinical signs that are characteristic to DIC in other species.

The clinical outcome following envenomation, however, is highly variable, partly depending on its anatomical location and the amount of venom delivered. Accordingly, clinical signs may remain local in up to 49% of human cases, limited to pain, hemorrhage, edema and regional lymphadenopathy, while in other cases in humans and dogs, systemic clinical signs may ensue, including shock, cardiac arrhythmias and bleeding. The reported mortality rate in humans is low, ranging from 0.5% to 2%, whereas in dogs it may reach 15%. Different **VP**-specific antivenin treatment protocols, the result of lower availability and financial constraints in veterinary medicine, may partly account for the discrepancy. Retrospective studies of **VP** envenomations in humans, unlike studies in dogs, espouse the use of specific antivenin to improve outcome, although robust evidence is lacking.

Different treatment regimes are used for **VP** envenomation in different (human or veterinary) institutions. These include antibiotics, antihistamines, steroids and specific antivenin [10]. Dosing and timing are controversial, and may vary in different medical institutions. The Hebrew University Veterinary Treating Hospital (HUVTH) treatment protocol includes:

1. Fluid therapy

    - Crystalloids- 10-20 mL/kg as a bolus.

    - Colloids / Fresh frozen plasma when DIC is suspected or when total solids (TS) are low (below 4 g/dL), as needed.

2. Antimicrobial therapy (ampiciline 25 mg/kg q8h for 5 days) to prevent clostridial or other infection that may have been transferred by the snake's teeth.

3. Antihistamine - Diphenhydramine (2 mg/kg q8h for 24 h), only after hypersensitivity skin test for the antivenom has been completed.

4. Steroids - The use of glucocorticosteroids in snakebites has always been controversial. They are used in cases of shock and severe edema, particularly when in the larynx area, as they may minimize and/or prevent further endothelial damage. However, steroids may slow and diminish antivenom activity and increase the risk for bacterial infection. Some clinicians believe that the use of steroids is contraindicated in snakebites [10]. In a retrospective study of 327 dogs with snakebite it has been shown that glucocorticoid

therapy was significantly associated with mortality (**P**=0.002) [4]. Therefore, the use of steroids in HUVTH is limited to cases in which laryngeal edema is severe and may prevent tracheostomy.

5. Antivenin- it has been shown that treatment with low specific antivenin dose (10 mL) did not decrease mortality rate in dogs [4]. However, in humans, the mortality is reported to decrease sharply from 6-10% to 0.5-2% since the introduction of the specific antivenin [12]. Our recommendations, based on the findings in human medicine and our clinical experience, are that antivenin is to be administered to effect in cases of:

- Clinical signs of shock.
- Severe swelling at the site of the bite.
- Abnormal coagulation parameters, including thrombocytopenia.
- Owner's financial abilities (10 mL cost in Israel approximately $250).

6. Monitoring for 24 h. This includes complete blood count (CBC), creatinine (q12h), PT and aPTT (q12h), PCV/TS (q12h), ECG (q2h), and blood pressure.

In conclusion, as was demonstrated by **VP**, a member of the **viper** family, **viper** snakebite has relatively low mortality rate both in human and in dogs. Further investigation of snakebites in cats is needed. Risk factors for mortality include the first months of the summer, low patient body weight, limb envenomation, systemic clinical signs and coagulation disorders. Treatment with steroid is controversial and should be further investigated, while antivenin should be considered in cases in which one of the risk factors to death is present and not as a fixed dose but rather to effect.

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NAVC SHORT TOPICS FROM EXPERTS

HOW I USE FECAL MICROBIOTA TRANSPLANTATION

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Introduction:

The intestinal microbiome is a complex system consisting of bacteria, fungi, protozoa and viruses. In mammalians, intestinal microbiota outnumber the host cells by a factor ten. In dogs, more than 10 different bacterial phyla have been described with Firmicutes, Bacteroidetes, Proteobacteria, Fusobacteria, and Actinobacteria being the most commonly represented. These microbiota provide essential benefits to the host health: they play a key role in the development and function of the gastrointestinal immune system, protect against enteropathogens, support the epithelial integrity and promote nutrient supply to the enterocytes. Recent studies have highlighted the importance of metabolites produced by intestinal microbiota, also known as the intestinal metabolome, and their contribution to host physiology. Therefore, the intestinal microbiome is considered to be an essential organ in mammalians. Consequently, changes in the intestinal microbiome are often paralleled by changes ranging from inability to process and absorb nutrients, to absence of important metabolites or appearance of undesirable metabolites in the intestinal mucosa with possible effects for instance on the enteric nervous system and higher centers. In human medicine, a large body of research has documented changes affecting intestinal microbiota and associated metabolites in diseases of the gastrointestinal tract and liver, but also in diseases affecting organs outside the digestive tract such as obesity, behavioral and psychiatric disorders.

Definition:

Fecal microbiota transplantation consists in transferring fecal microbiota from a healthy donor to a host with an abnormal intestinal microbiome. While the technique has been known and used for many centuries in various indications, it has regained popularity in recent years for the treatment of **Clostridium difficile** colitis in people, and its use has been approved by the Food and Drug Administration for that indication. The goal of FMT is to provide a healthy and viable sample of fecal microbiota to repopulate the recipient's large bowel. In addition, FMT also delivers essential metabolites such as secondary bile acids that may be lacking in the host.

Indications for FMT in small animals:

There is very little published data on use of FMT in dogs and cats. Apart from a few abstracts presented over the last 6 years, there is only one published study on the use of FMT as an adjunct to therapy in puppies with parvovirus infection. In that study, FMT was applied at patient admission and repeated every 48 h as needed. The results were encouraging since FMT lead to faster recovery from diarrhea and shorter hospitalization time. Investigations on the value of FMT in dogs and cats with chronic enteropathies (such as IBD) or in animals receiving antibiotics are underway at different centers.

Donor selection

It is a complex process in human medicine, where fears of transmission of known and yet unknown diseases are high. Overall, two options exist: (1) to obtain fresh feces from a family member or friend of the patient or (2) to order frozen feces from a fecal bank where fecal donors are subject to very detailed investigations of their medical history and lifestyle prior to being approved. Fresh feces are most commonly used in small animals, and donors should be clinically healthy with a normal body condition score, not prone to digestive diseases such as vomiting and diarrhea, and screened for intestinal parasites (nematodes, cestodes and protozoa), and ideally for enteropathogenic bacteria (PCR). The author also determines a dysbiosis index in order to check the health of donor's fecal microbiome. Moreover, fecal donors should not have received any antibiotics in the 3-6 months prior to collection. Additionally, in cats, FIV and FeLV testing is required.

Preparation and administration:

Fresh feces should be processed within 6-12 hours of collection. Numerous protocols are used for preparation of FMT solution. The author adapted current state-of-the-art processing from human medicine in his lab. The feces are weighed, and diluted with saline (volume = 4 x fecal weight), then thoroughly mixed prior to filtration through a layer of gauze. Immediately following preparation, the fecal solution is administered to the recipient in the proximal part of the descending colon via enema (or via colonoscopy) at a dose of 5 ml/kg. Although there is debate about the necessity to administer a sedative to recipients prior to the enema, it may be useful, particularly in difficult dogs and cats. In order to avoid rapid evacuation of the transplanted solution, recipients should be kept quiet for 30 min after administration. The need for repeat FMT depends on what disease is being treated. It is probable that dogs and cats with chronic enteropathies would benefit from repeated FMTs while a single or two FMTs might be sufficient to help with acute diarrhea. Oral administration of FMT through a nasoesophageal tube or inside gelatin capsules is another delivery method.

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Outlook:

Several studies are ongoing on the use of FMT in dogs and cats, and publication of results can be expected with 1 to 2 years. If their outcome is positive, it is likely that FMT will gain popularity in the treatment of acute and chronic GI disorders. In the future, it is possible that FMT will be recommended in small animals as part of the treatment of diseases primarily affecting organs outside the GI tract. Examples could include hepatic encephalopathy, obesity, behavioral disorders, etc.

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SVA FELINE

FELINE FEEDING TUBES: O/E-TUBE OR PEG-TUBE?

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Introduction:

The cardinal rule of feeding a malnourished or anorectic patient is 'if the gut works, use it'! This is a tricky proposition in cats who seem more prone to anorexia than dogs as a result of stress (at home or in hospital), finicky eating behaviours and dietary preferences. When these factors are added to an ill cat experiencing gastrointestinal signs, malaise, pain or lethargy, it becomes increasingly challenging to keep these patients at an appropriate plane of nutrition. Yet studies have shown that nutrition plays a key role in the full recovery of critically-ill patients[1]. Enteral feeding techniques preserve the mucosal barrier, prevent villus atrophy, help maintain the immunologic function of the GI tract, and can allow owners to treat their pets at home. While pharmacological appetite stimulants and naso-oesophageal feeding tubes are ideal for short term usage, neither generally allow adequate caloric feeding in severely sick patients and feeding may even be impossible in cats with anatomical or functional problems of the oral cavity or oesophagus.

This lecture will outline basic information for clinicians to consider using feeding methods intended for more long-term use (e.g oesophagostomy tube vs gastrostomy tube). It should be emphasised early on that with suitable training, these solutions **are achievable** for those in the general practice setting.

1) Oesophagostomy Tube

Oesophagostomy tubes are often favoured for intermediate to long-term use (>1-2 weeks). Examples of cases which could benefit from this method would include: nutritional support for severe orofacial injuries, cases of hepatic lipidosis/cholangitis/acute pancreatitis and for those non-compliant patients requiring long-term oral medications to manage their condition (e.g. mycobacteria infection).

It should be noted that if a patient is anaesthetised for an investigative procedure (e.g. exploratory laparotomy for full-thickness biopsies for suspected gastrointestinal disease), the placement of an oesophagostomy tube should be strongly considered as they can be easily removed if not required.

Oesophagostomy tubes are generally well tolerated by most patients. Because of its large bore (16-18F), it can often accommodate most blenderised diets (cf. naso-oesophageal feeding tubes).

Contraindications:

- Primary or secondary oesophageal disorders such as oesophageal stricture, oesophagitis and mega-oesophagus
- Persistent vomiting
- Absent gag reflex

Serious complications seem to be exceptionally rare.[2] The only minor complication occasionally seen with oesophagostomy tubes is stoma-site infection/granulation tissue[1,2] and (in the author's experience) continued anorexia due to oesophageal irritation. Prevention of stoma-site infection is usually achieved by daily cleaning of the tube site.

2) Gastrostomy Tubes: PEG Tubes (Percutaneous endoscopically placed gastrostomy):

Gastrostomy tubes are indicated in patients with chronic diseases requiring long-term/life-time nutritional support such as those suffering from chronic or irreversible diseases affecting the oropharynx or oesophagus. PEG tubes should not be placed when only short term usage is anticipated (<7 days), in cats considered poor anaesthetic candidates or when complications such as wound healing or severe coagulopathy are present.

Gastrostomy tubes can be placed 1) blind percutaneously (e.g. ELD applicator), 2) surgically during laparotomy or 3) endoscopically.

Percutaneous endoscopically placed gastrostomy (PEG) tubes are often the preferred method of gastrostomy placement because visibility is ideal and the procedure is fast, effective and safe in most circumstances.

The most common complications associated with PEG tubes include insertion site infection, peristomal pain, peristomal leakage, chewing/dislodgement of the tube by patient, and tube occlusion. Other less common complications include injury to adjacent organs (e.g. spleen, colon, small intestine), bleeding (intraperitoneal, abdominal wall, organs), 'buried bumper' syndrome, and gastric outflow obstruction.

Equipment:

A standard gastroscope is utilised to facilitate placement of the feeding tube. The commercial kits contain - besides the feeding tube with conical end - the following material, large over-the-needle catheter (16G), double looped strong suture material (long enough from mouth to side of abdomen, 70-100cm), scalpel, material to fix feeding tube on outside (clips or suture) and adaptor for feeding syringe.

There are two types of PEG tubes available, commercial kits (adapted from human medicine) which contain all material for quick placement, and "home-made" kits which are cheaper but more cumbersome to build/place and therefore more time consuming. With both 'kits', the placement takes place the same way – a "guide wire" of suture material is pushed via a needle through the abdominal and gastric wall into the gastric lumen, grabbed endoscopically and pulled via oesophagus through the mouth. The PEG is then attached and pulled via the "guide wire" into place. Commercial PEG tubes are either silicone or polyurethane and can withstand gastric acidity for the animal's lifetime. In cats 16 Fr to 20 Fr size is used.[3] The inner flange is different between different brands but all are suitable for cats.[3]

A step-by-step guide of PEG tube placement in cats can be accessed for more in-depth instruction.[3]

Once the PEG is in place, the site is lightly wrapped with occlusive dressing, a body or stretch netting is commonly sufficient in cats and no buster collar needed. Daily cleaning of the stoma with an antibiotic or iodine cream is important. Feeding can be begin 24hours post-placement after performing a contrast radiograph to confirm the absence of peritoneal leakage. Initially, the cat can be given 5-6 divided meals with an increase in volume, decrease in frequency, over the course of a week.. While problems with PEG feeding is rare[2-4], they can arise and should be dealt with according to guidelines.[2]

PEG tubes can stay in place for many months, even lifelong. A PEG tube must stay in place in place for at least 3-4 weeks to ensure adequate adhesion formation.[3] Removal should always occur endoscopically as the inside flange of commercial kits is too large to pass naturally when the tube is cut from the outside.

Low Profile Button Tubes:

These tubes lie flush against the body wall giving a more cosmetic appearance. It also seems to allow for the cat to resume normal activity without heavy bandages or risk of tube dislodgement via entrapment on objects. In the author's experience, they seem to have a low complication rate and offer high owner satisfaction compared to more traditional tubes.

They can be placed either as a 'two step approach' following the initial placement of a PEG tube, or a 'one step approach'. The latter approach eliminates the initial placement of a traditional PEG tube.

'Two-Step' Low Profile Placement:

These tubes can be placed percutaneously after a secure gastropexy and stoma have been achieved using a traditional PEG tube. Stoma tract maturation typically occurs in 4-6 weeks. Low profile buttons have two types of internal fixation flange: 1) balloon 2) non-balloon.

In the author's experience, balloon buttons seem to have a high failure rate due to spontaneous deflation and/or bursting resulting in dislodgement. For this reason, the author recommends the use of a non-balloon buttons due to its resilience to long-term exposure to gastric acid and their superior retention disc compared to balloon material. Due to availability (or lack thereof) of appropriately sized infant-sized low profile tubes in cats, the author will often chose to place a slightly larger traditional 20F PEG and replace with a smaller 18F x 1.5cm low profile non-balloon (AMT Mini-One Button Low Profile). This size seems to be suitable for most breeds/size and BCS. The tube uses a stiff stylet with string/toggle system to aid in flange deployment. Removal also relies on a stiff stylet to straighten the mushroom tip resulting in little resistance during traction.

'One-Step' Low Profile Placement Methods:

A One-Step method for placing low profile buttons can be used as an alternative to the above method. This can be achieved via 'pull technique' (see One-Step Low Profile, non-balloon button) which is placed in a similar fashion to a normal PEG tube (author's choice in cats 16G x 1.7cm with retention discs).

Summary

Enteral feeding devices should be employed early when it is recognised that the patient is not meeting its nutritional and energy needs or requires long-term pharmacological management in a non-compliant patient.. Proper tube placement, handling, and client communication are essential for tube longevity and patient safety. Enteral feeding is the preferred method of nutritional support in patients with functional gastrointestinal tracts. Remember, '**If the gut works, use it!**'

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ANIMAL WELLNESS & WELFARE

BEHAVIORAL ISSUES IN DOGS OBTAINED AS PUPPIES FROM PET STORES

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BEHAVIORAL ISSUES IN DOGS OBTAINED AS PUPPIES FROM PET STORES

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Large-scale, high-volume breeding of dogs for purposes of selling puppies occurs around the world. Because these commercial breeding establishments (CBEs) are seeking to maximize profit, the care of the dogs kept for breeding is often substandard, and in many cases inhumane. This creates the potential for innumerable problems, both physical and psychological-behavioral, to occur in the puppies.

In the United States most puppies sold by pet stores are purchased from intermediaries, who acquire their puppies from CBEs and then distribute them to the retail stores. In addition, many puppies produced in CBEs are sold directly to the consumer over the Internet.

Conditions in the CBEs are reported to vary widely, ranging from relatively clean to squalid, noxious, and gravely detrimental to animal health and welfare. CBEs are typically characterized by large numbers of dogs, maximal efficiency of space by housing dogs in or near the minimum space permitted by law, breeding dogs spending their entire reproductive lives in their cages or runs, group and solitary housing, dogs rarely if ever permitted out of their primary enclosures for exercise or play, no toys or enrichment, minimal-to-no positive interaction with humans, and substandard or no health care. Many but not all CBEs have cage flooring made of wire mesh, accumulation of feces, ammonia odor, no windows and poor ventilation, inadequate protection from inclement weather and temperature extremes, insufficient or contaminated water and spoiled food, serious untreated medical conditions (e.g., advanced dental disease), extensive matting of hair, odd or stereotypical behaviors by the dogs, evidence of starvation, and presence of deceased adult dogs and puppies.   **Results from studies**

Seven empirical studies have reported specific information about the behavior of dogs obtained as puppies from pet stores and/or were born in CBEs. In addition, one anecdotally reported study is included in this review of current knowledge. The studies come from 4 countries on 3 continents.

In a retrospective survey of 737 mature dogs, Jagoe (1994) investigated the relationship between early life experience and owner-reported behavior problems in adulthood. Twenty dogs were acquired from pet stores. Jagoe found that when compared with dogs from other sources, dogs obtained from pet shops showed higher levels of 'dominance-type' aggression (aggression directed toward people, especially the dog's owner and owner's family members). Pet store-acquired dogs also more often demonstrated social fears (fear of strangers, children, and unfamiliar dogs) compared with dogs from other sources.

Bennett and Rohlf (2007) studied the frequency of potential problem behaviors reported by owners in 413 companion dogs, 47 of which were obtained from pet stores. Mean scores on the unfriendly/aggressive subscale of behaviors were significantly higher for dogs obtained from pet stores and animal shelters compared with dogs obtained from breeders. Dogs obtained from pet stores also had significantly higher mean scores on the 'nervous' behavioral subscale than dogs who were home-bred.Pierantoni et al. (2011) compared owner-reported behaviors of 70 adult dogs separated from their mother and littermates at 30 to 40 days of age and the behaviors of 70 adult dogs separated at two months of age. 71 dogs came from pet stores. Results showed that he frequency of certain behaviors (fearfulness on walks, aversion to strangers, destructiveness, excessive barking, attention-seeking behaviors, toy possessiveness, and play biting) among dogs separated from their mother and littermates at the earlier age was higher if they came from pet shops rather than from other sources. For example, 80% of dogs separated early from litters and obtained from pet stores exhibited destructiveness more frequently compared to 20% of dogs not separated early.

McMillan et al. (2013) compared the owner-reported behavioral characteristics in 413 dogs obtained as puppies from pet stores and 5,657 dogs obtained as puppies from noncommercial breeders. They found that dogs acquired from pet stores were in general more excitable, energetic, and attached/attention seeking, and less trainable than dogs from breeders. Sexually intact pet store dogs were three times as likely to be reported showing owner-directed aggression as were sexually intact dogs acquired from breeders, and pet store dogs were nearly twice as likely to be reported to have shown aggression toward unfamiliar dogs (dog-directed aggression). Other behaviors reported more frequently in dogs from pet stores compared with breeders included stranger-directed aggression, dog-directed aggression, dog-directed fear, nonsocial fear, separation-related behaviors, escape behavior, sensitivity (disapproval), sexual mounting of people and objects, and house-soiling (urination and defecation).

Casey et al. (2014) examined the demographic variables and risk factors associated with owner reported aggressive behavior in dogs. Results showed a 1.8 times increased risk of aggression toward family members in dogs from 'other' sources (the category which contained pet shops) as compared to those obtained directly from breeders

Pirrone et al. (2016) conducted a study to compare owner-assessed potential problem behaviors in two groups of dogs: those obtained from pet shops and those obtained from official Italian breeders recognized by the Italian Kennel Club (E.N.C.I). 349 were acquired as puppies from breeders and 173 from pet shops. Compared with dogs acquired from breeders, dogs from pet stores were more likely than dogs from breeders to have an increased risk for owner-directed aggression, separation-related behaviors, and housesoiling. The authors also found a number of owner-related factors to be important, including no prior experience with dogs, nonattendance at training courses, and lack of awareness of the existence of veterinary behaviorists.

Gray et al. (2016) looked at differences in the behaviors of adult dogs based on the assumed quality of the breeding operation, using specific criteria to classify breeders into 2 groups: "responsible" or "less responsible." The study focused on three popular breeds – Chihuahua, pug and Jack Russell terrier.

Chihuahuas acquired from less responsible breeders were reported to show more aggression toward familiar dogs, unfamiliar dogs, unfamiliar humans, and the dogs' owners; they also showed more fear of unfamiliar humans, sensitivity to touch, separation-related behaviors, and chasing. Pugs from less responsible breeders were reported to show more fear of dogs, other fear, aggression toward familiar dogs, separation-related behaviors, and excitability. Jack Russell terriers from less responsible breeders were reported to show a decrease in trainability.Finally, an anecdotal report presented in a book chapter described a sample of 1,864 dogs exhibiting various behavioral problems found that 220 (approximately 12%) of the dogs displayed separation-related problems (Mugford, 1995). An analysis based on the source of the dog revealed that only 10% of purebred dogs obtained directly from breeders presented with separation-related problems, whereas "55% of purebred dogs originating from so-called 'puppy farms' or 'puppy mills'" presented with such problems. It was not reported how it was determined that the dogs came from puppy farms or puppy mills.

Summary

The data on dogs obtained as puppies from pet stores and/or born in CBEs shows that these dogs exhibit an increased incidence of behavioral and emotional problems that cause distress in adulthood compared with dogs from other sources, especially breeders. The most consistent finding among studies is an increase in aggression, which is most commonly directed toward the dog's owners and family members but also to unfamiliar people, and other dogs. Increased fear was also identified in response to unfamiliar people, children, other dogs, nonsocial stimuli, and when taken on walks. Undesirable behaviors related to separation and/or attention-seeking and a heightened sensitivity to touch have been reported.

Contributing factors for these reported outcomes are numerous. Some key factors include genetics, early life stimulus deprivation (inadequate stimulus exposure, inappropriate or lack of social exposure), stress (prenatal maternal stress and postnatal early life adversity), early weaning and maternal separation, transport and pet-store-related factors, and owner-related factors such as inadequate knowledge and experience with dogs as well as different levels of commitment to the pet dog.

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DIAGNOSTIC IMAGING

SPECIAL SONOGRAPHIC FEATURES OF THE FELINE ABDOMEN

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SPECIAL SONOGRAPHIC FEATURES OF THE FELINE ABDOMEN

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Louisiana State University School of Veterinary Medicine, Baton Rouge, LAHepatobiliary Organs

The feline liver can sometimes be tricky to examine in obese cats. A common error is to mistake the falciform fat for the liver, commonly seen in beginning sonographers. Careful attention to the hyperechoic liver capsule helps the sonographer to see the separation between the fat and the liver. Comparatively, the falciform fat should be hyperechoic compared to the liver.

The use of ultrasound and ultrasound-guided tissue sampling has now surpassed the use of survey radiography for the diagnosis of many liver diseases due to its broad availability and greater sensitivity. Diffuse parenchymal disease generally affects all lobes and may appear normal, iso- or hyperechoic. Examples include: cholangiohepatitis, diffuse prenodular (early) metastatic carcinoma or sarcoma, round cell neoplasia (lymphoma, mast cell disease and histiocytic sarcoma), patchy or diffuse fatty infiltration, vacuolar hepatopathy, storage diseases (amyloidosis, copper), toxic hepatopathy and early degenerative changes associated with micronodular hyperplasia and fibrosis. The overall accuracy of ultrasound as the sole criterion for discriminating among the categories of diffuse liver disease is <60% in cats. It is generally not possible to make a final diagnosis based on the combination of sonographic findings and biochemical and hematological data with diffuse liver disease. Tissue sampling, preferably for histological examination, is required for a definitive diagnosis in most instances, even if the liver appears sonographically normal.

Vacuolar changes in the liver associated with lipidosis usually cause hepatomegaly in conjunction with diffuse hyperechogenicity and rounded borders. Inflammatory disease can be associated with diffuse hypoechogenicity. If acute hepatitis or cholangiohepatitis is present, the liver may appear to have high contrast; a hypoechoic parenchyma with pronounced hyperechogenicity of the portal veins. Chronic inflammation of the liver will usually result in hyperechoic or mixed echogenicities. When fibrosis or cirrhosis is present, the liver may be smaller and hyperechoic. If nodular hyperplasia develops such as with vacuolar hepatopathy, the liver may appear more heterogeneous and nodular such as in neoplastic disease. Other differentials for this pattern include amyloidosis in cats.

Focal or multifocal changes in the liver parenchyma are easier to identify sonographically than diffuse changes. Hypo-, hyper- and anechoic lesions are easy to identify as they contrast better with the surrounding parenchyma. Therefore, cystic lesions are the easiest to detect, even when very small.

Anechoic cavitary structures in the liver can be due to necrosis, neoplasms or cysts. Cysts structures generally have sharply defined borders, can be round or irregular in shape and may even contain hyperechoic septa within them. Causes include congenital cysts, due to cavitations following trauma, biliary pseudocysts or parasitism. Unfortunately, biliary cystadenomas and cystadenocarcinomas may appear similarly.

Neoplastic disease of the liver may manifest as diffuse, multifocal or focal disease sonographically. Diffuse disease is usually due to round-cell neoplasia. Lymphoma, histiocytic sarcoma and mast cell tumor are the most common neoplasms that may lead to diffuse changes that remain sonographically undetectable. Carcinomas tend to be diffusely spread throughout the liver and often lead to a mixed pattern.

Malignant nodules have a highly varied appearance and size. They may appear as hypo- or hyperechoic nodules, target lesions or heterogenous ill-defined nodules. Hypoechoic nodules can be due to nodular hyperplasia, metastases, lymphoma, histiocytic sarcoma, primary neoplasia, necrosis, hematomas and abscesses. For this reason, tissue sampling is critical to a definitive diagnosis and the presence of hepatic nodules is not synonymous with malignancy. Hepatic target lesions have a positive predictive value for malignancy of 74% and emphasizes the fact that histological type cannot be predicted by the presence of target lesions.

Hepatic abcessation occurs rarely in small animals and may appear similar to a primary tumor, granuloma or hematoma due to their highly variable sonographic features. Sonographically, they may be round to irregular in shape with either a hypoechoic central region or of mixed echogenicity. Reverberation artifacts may be detected due to gas accumulations within the necrotic tissue. Focal peritonitis may be seen with abcessation and include free peritoneal fluid and focal hyperechoic mesentery.

The feline gall bladder is typically ovoid but can be bi-lobed. The cystic duct is highly tortuous in the cat. The gallbladder wall is approximately 1mm thick in the cat and the bile is anechoic. Gallbladder wall thickening and sludge are indicative of gallbladder disease.

Cholecystocentesis is helpful in these instances for performing bacteriology and cytology. Extrahepatic bile duct obstruction often results in dilation of the common bile duct, between the porta hepatis and the major duodenal papilla. The papilla is fairly easy to identify in cats as it is round and echogenic, located at the wall of the cranial duodenum, a short distance from the pylorus. The common bile duct is easily visible in most cats as a thin anechoic tube ventral to the portal vein, another large and easy to identify structure in the cat. Only about half of cats with obstructive biliary disease have a dilated gallbladder. The bile duct should be traced and observed for wall thickening, intraluminal echogenic or hyperechoic shadowing material and for papillary masses. Inflammatory disease of the papilla can be as obstructive as a cholelith. **Spleen**

The spleen is small in healthy cats and is very laterally located in the left cranial abdomen. Sometimes it is necessary to scan intercostally to find the spleen. Usually if one cannot find the spleen, it is small and dorsal and the sonographer needs to strive to find it intercostally.

Splenomegaly in the cat can be caused by extramedullary hematopoiesis, hyperplasia, chronic inflammation, immune mediated disease, neoplasia, and infectious organisms. Lymphoma is by far the most common neoplasm affecting the spleen. Mast cell tumor and histiocytic sarcoma are also possible and often cause splenomegaly. Histoplasmosis is a systemic fungal infection that affects many organs in the cat, including the spleen. Sonographically it appears enlarged and diffusely hypoechoic and in some instances can be mottled.**Gastrointestinal Tract**

The stomach

Gastritis is very difficult to diagnose sonographically and there are few specific signs. Cats rarely get ulcers unless induced by an overdose or chronic use of anti-inflammatory agents. Ulcers can lead to a focal wall thickening that, if not masked by gas and ingesta, can be identified sonographically. Linear foreign bodies anchored at the tongue may also cause bunching up of the stomach in addition to the jejunum. Hair balls are identified as a heterogenous structure with gas reverberations and shadowing.

The gastric wall in the cat is usually about 3mm thick. Thickening with loss of layering is mainly due to neoplasia. Gastric neoplasia can only be identified with sonography approximately 50% of the time compared with endoscopy. Gastric lymphoma often leads to a transmural, hypoechoic wall thickening, often diffuse or within a large section of the stomach.

The duodenum

The duodenum is very midline and to find it sonographically, one must focus on the porta hepatis, identify the stomach and trace it rightward and the pylorus and duodenum will be easily visible. Continuing, trace the duodenum along its short length and notice the small papilla at its cranial end for entrance of the common bile duct and pancreatic duct.

The jejunum

Randomly distribute, 2-3mm in thickness with distinct five wall layers. The most common abnormality of the small intestine is muscularis thickening seen in both inflammatory bowel disease and lymphoma. Linear foreign bodies have a specific appearance where the bowel is plicated together and often a thin hyperechoic band is evident pulling them together.

The ICCJ (Ileocecocolic junction)

The feline cecum is a small bulbous organ having a subtle curvature (concave side toward the ileum), located in the right abdomen ventral to the descending duodenum. A small constriction demarcates the transition with the colon and the ileum enters the ascending colon obliquely from the left, just distal to the cecocolic transition. The position of the ileum relative to the cecum is fixed by the presence of the ligamentum ileocecalis, in which the ileocecal lymph nodes are located. These lymph nodes are usually paired and can be found along the concavity of the cecum. The ascending mesocolon contains one to five colonic lymph nodes. **Urinary Tract**

Normal kidneys are 30-45mm in length. Fat deposited in the renal tubules in cats leads to a more hyperechoic cortex in some animals. Bilateral pelvic dilation is often due to obstruction in male cats. Uni- or bilateral pelvic or ureteral dilation in cats often occurs due to inflammation secondary to ureterolithiasis. If ureterolithiasis is suspected and ureteral dilation is noted in ultrasound, abdominal radiographs following an enema should be performed to screen for uroliths which may be challenging to find sonographically but easy to see radiographically. Any toxin that cat ingests can also affect the kidneys. Ethylene glycol and Easter Lily are two and the retroperitoneal space may develop fluid secondary to acute renal injury. Ethylene glycol affected kidneys may become so hyperechoic that shadowing will result. Polycystic kidney disease in cats and results in several cysts in both kidneys to complete absence of recognizable renal tissue. The cysts are thin walled, with a near and far wall hyperechoic border and anechoic content.

Congenital defects of the kidneys include hypo- and dysplasia as well as aplasia. When young cats have small and irregularly shaped kidneys, dysplasia is more likely than chronic renal disease. Chronic renal injury in cats is due to chronic nephritis, glomerulonephritis, amyloidosis and nephrocalcinosis. In all cases the kidneys are smaller in length, hyperechoic and often irregularly shaped. Loss of corticomedullary distinction is also apparent. Occasional small round anechoic cortical cysts are

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identified as well. Chronic renal injury can also lead to uremic gastropathy which sonographically appears as hyperechoic mucosal borders. Cryptococcus and feline infectious peritonitis can both lead to chronic renal injury and the sonographic findings are variable.

Renal neoplasia in the cat is often due to lymphoma which can appear a nodules or diffuse cortical echogenicity and enlargement. Specific to cats is a hypoechoic halo around the kidney that can be present with lymphoma, but also with renal infection such as with FIP.

Bladder: Suspended echoes that do not cause acoustic shadowing, reverberation, or twinkle artifact distal to the echo may be due to urine lipid. Clumping of these echoes may be present. Hyperbilirubinemic animals can have similar findings as can those with hematuria. Cystic calculi are hyperechoic and dependent and shadowing, not suspended. Cats may have accumulations of fine crystals that collect and shadow but when agitated break apart and look like a snow globe. Chronic cystitis can affect the cranioventral wall which becomes focally thickened with an irregular mucosa. Cats will develop a hyperechoic and sometimes shadowing border facing the lumen due to mucosal necrosis gas bubble entrapment, much like an ulcer. Pedunculated and broad-based masses can develop and can be benign polyps as well as malignant neoplasms. **Pancreas**

Chronic pancreatitis is poorly described in cats. The pancreas may be of normal size or enlarged with a heterogenous appearance. Hyperechoic foci with acoustic shadowing may represent mineralizations. Multiple hypoechoic round foci of a few millimeters in diameter may also be recognized. These may represent nodular hyperplasia or dilated pancreatic ducts. Cavities of the pancreas cats are typically either due to abscesses or pseudocysts and appear as anechoic or hypoechoic cavities, possibly with a thickened wall. A number of investigators have attempted to assess the sensitivity and specificity of ultrasound compared to other imaging modalities for diagnosing pancreatitis in cats, however, with greatly varying results. Ultrasound will most likely remain one of the most important diagnostic tools in both dogs and cats as it allows not only assessment of the pancreas, but also that of other organs that may be involved in the inflammatory process.

Identifying important landmarks is critical to localizing the pancreatic limbs as the pancreatic parenchyma can be difficult to differentiate initially from the surrounding mesentery. It may have indistinct margins and be isoechoic with the mesentery in the normal situation. The left lobe of the pancreas and body are easier to see than the right. Pancreatic ducts are easy to see in most cats. Also, the major duodenal papilla is the common entrance of the pancreatic and common bile duct in cats. The main landmark for identifying the left lobe and body in the cat is the portal vein. The pancreatic body lies directly ventral to the portal vein caudal to the stomach. The left lobe is caudal to the stomach and cranial to the transverse colon on the left side of the portal vein. It may continue caudally for a small distance to the level of the splenic hilus. The pancreatic duct is more commonly identified in cats and is seen as a small anechoic tubular structure in the body and left pancreatic lobe. The right lobe of the pancreas is small in the cat and is more difficult to identify. It is adjacent to the duodenum and follows it caudally. A small pancreatic duct can also be identified in it. The major duodenal papilla appears as a small nodule attached to the duodenal wall close to the cranial flexure.

The following are parameters that should be assessed when examining the feline pancreas:

The left and right limbs as well as the body should be examined and measured for thickness in the sagittal plane. Normal: Body: 0.5-0.9cm thick, Left lobe: 0.4=10cm thick, right lobe: 0.3-0.6cm. In acute disease, the pancreas may become enlarged as in dogs. However, this finding is much more inconsistent in cats. In chronic disease the pancreas may be of normal size or smaller. Unremarkable changes do not rule out pancreatitis in cats. Pancreatic size does not increase with increasing age.

Echogenicity: Normal: iso- to hypoechoic with the mesentery. Abnormal: The pancreas usually becomes hypoechoic in acute disease. In chronic disease the pancreas may have a normal, hypo or hyperechoic appearance. Pancreatic echogenicity does not change with increasing age.

Abnormal Echotexture: Often the pancreas appears heterogeneous. It can become nodular with irregular borders. Nodular hyperplasia has the appearance of small hypoechoic distinct nodules throughout the parenchyma. It is commonly seen in older cats. Nodules may be up to 1cm in diameter and the pancreas may be enlarged.

Pancreatic duct size: Normal:0.5-2.5mm diameter. Too little is known about the size of the duct in disease. However, there is a slight increase in size of the duct in older cats.**Adrenal glands**

Normal feline adrenal glands are 1cm in length, 3.7-4.9mm pole height, have an ovoid shape and are hypoechoic and can have mineralizations in older animals. Cats with hyperthyroidism may have 20% larger glands, which can be explained by the stimulation of the hypothalamic-pituitary-adrenocortical axis by hyperthyroidism. Treated hyperthyroid cats had a length of 1.1cm and up to a 4.9mm pole height. Untreated hyperthyroid cats can have a gland length of 1.5 cm and pole height of up to 4.9mm. Adrenal tumors most always lead to much greater size changes, rather in the centimeter rather than mm range. Adrenal tumors in cats with hyperaldosteronism are typically 2-4cm in size. Cats with acromegaly can have bilateral enlargement also.

WSV18-0328

INFECTIOUS DISEASES AND GASTROENTEROLOGY
(TRANSLATED INTO MANDARIN CHINESE)

MANAGEMENT OF PARASITIC AND BACTERIAL CAUSES OF INFECTIOUS DIARRHEA IN DOGS AND CATS

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Objectives. The primary objectives of this session are to review the known parasitic and bacterial causes of diarrhea followed by a discussion of optimal diagnostic tests, treatments and preventions.

Gastrointestinal (GI) signs can be the result of primary diseases of the GI system or secondary GI diseases. The secondary GI diseases are generally those of the kidneys, liver, pancreas (pancreatitis or exocrine pancreatic insufficiency [mainly dogs]), endocrine system (hyoadrenocorticism; diabetic ketoacidosis; hyperthyroidism [mainly cats]), or central nervous system.

Differential diagnoses for primary GI diseases are often grouped into obstruction (masses, foreign body, and intussusception), dietary intolerance, drugs/toxins (garbage gut), inflammatory gastric and bowel diseases, neoplasia, infectious diseases, and parasites.

The primary bacteria associated with gastrointestinal tract disease in dogs and cats include **Salmonella spp., Campylobacter spp., Clostridium perfringens, Helicobacter spp**., bacterial overgrowth syndrome, bacterial peritonitis, and bacterial cholangiohepatitis. The primary nematodes are **Ancylostoma/Uncinaria, Trichuris vulpis** (dogs), **Strongyloides, Dirofilaria immitis** (vomiting in cats), **Toxocara spp**., **Toxascaris leonina, Ollulanus tricuspis** (cats), and **Physaloptera spp**. Common enteric protozoans include **Giardia spp., Cystoisospora spp., Cryptosporidium spp**., and **Tritrichomonas foetus (blagburni).**

Occasionally, otherwise healthy dogs or cats with acute diarrhea and normal physical examination findings can be handled conservatively by withholding food for 24 hours followed by introduction of a bland food for several days. For all animals with diarrhea with no apparent cause on physical examination, I will perform a fecal flotation, fecal wet mount examination, complete blood cell count (CBC), and rectal cytology if diarrhea is present. While the CBC generally does not lead to a specific diagnosis, the presence of eosinophilia makes inflammatory bowel diseases and parasitism more likely.

I perform acid-fast staining of a fecal smear or immunofluorescence antibody staining (Merifluor Giardia/Cryptosporidium, Meridian Diagnostics) on all animals with diarrhea to assess for the presence of

Cryptosporidium spp. oocysts. A wet mount examination may aid in identifying trophozoites of **Tritrichomonas** and **Giardia**. If neutrophils or spirochetes are evident on rectal cytology I recommend fecal culture (or PCR) for **Salmonella spp**. and **Campylobacter spp**.. If spore-forming rods consistent with Clostridium perfringens are present in large numbers, fecal enterotoxin assays and PCR assays can be performed to help confirm the diagnosis. However, these tests can be positive in healthy animals as well and so has less than 100% predictive value.

There are multiple drugs used in the treatment of gastrointestinal parasitic infections. For all puppies and kittens, the strategic deworming recommendations for the control of hookworm and roundworm infections from the Centers for Disease Control and the American Association of Veterinary Parasitologists should be followed by veterinary practitioners.   (www.cdc.gov/ncidod/dpd/parasites/ascaris/prevention.htm; www.capcvet.org).

If owners are interested in more in depth information a good website is available (www.petsandparasites.com).

Giardia infections often respond clinically to the administration of metronidazole but infection is usually not eliminated. Administration of metronidazole benzoate at 25 mg/kg, q12hr, PO, for 7 days was effective in suppressing cyst shedding to below detectable limits in 26 cats. This is the maximum dose of metronidazole that should be used; CNS toxicity can be induced by overdosing or as a cumulative neurotoxin. I personally use fenbendazole at 50 mg/kg, PO, daily for at least 5 days in dogs or cats with giardiasis. Metronidazole and fenbendazole can be combined in resistant cases. Febantel containing products have been used successfully in dogs and cats and this drug is approved for the treatment of giardiasis in some countries. The empirical dog dose is the deworming dose, daily for at least 3 days. Paromomycin, ronidazole, and nitazoxanide are alternate drugs that could be tried in cases with resistant giardiasis. However, in my experience, dogs or cats with Giardia that fails to respond to metronidazole and fenbendazole have another underlying problem. Tinidazole at 30 mg/kg, PO, daily for 7 - 14 days may be effective in some dogs and cats for the treatment of giardiasis. Secnidazole at 30 mg/kg, PO, once was reported for treatment of cats with Giardia in Brazil. Additional information is needed before this protocol can be widely recommended.

Diet changes and probiotics can be tried for the management of parasitic diarrhea. Multiple drugs have been evaluated for the treatment of cats with **T. foetus** infections; until recently no drug eliminated infection and diarrhea rarely resolves during the treatment period. Recently ronidazole at 30 mg/kg, PO, q24hr, for 14 days eliminated clinical signs of disease and trophozoites

from cats infected with one strain of the organism. In another one small study, administration of metronidazole and enrofloxacin lessened diarrhea in kittens but it is unknown if the organisms infecting those cats was **T. foetus.** Tinidazole may control the diarrhea but was less likely to eliminate the infection compared to ronidazole. Some puppies have recently been shown to be infected by **T. foetus.**

Sequential administration of clindamycin followed by tylosin blocked oocyst shedding and resolved diarrhea in one cat with chronic, clinical cryptosporidiosis. Tylosin (10-15 mg/kg, PO, twice daily) has been apparently successful in lessening diarrhea and oocyst shedding in multiple other cats and dogs with diarrhea that were Cryptosporidium positive. However, infection is not eliminated. Unfortunately, tylosin is very bitter and usually has to be given to cats in capsules. Treatment duration may need to be weeks. In cats with naturally occurring cryptosporidiosis, response to azithromycin has been variable (Lappin MR, unpublished data). If tried, use 10 mg/kg, PO, weekly for at least 10 days. If responding, continue treatment for at least 1 week past clinical resolution. Nitazoxanide is a new drug being studied for the treatment of **Cryptosporidium and Giardia**. Little information is available concerning dosages, but in one dog study seemed safe and effective at 75 mg/kg, PO, on days 0 and 14. The drug Alinia® is available and is labeled for both organisms in humans. The primary side-effect to date has been vomiting and so it should be given with food if used.

Cystoisospora spp. generally respond to the administration of sulfadimethoxine, other sulfa-containing drugs, macrolides, or ponazuril (or toltrazuril). Ponazuril is superior to other drugs and should be administered at 50 mg/kg, daily for 3 days. If there are multiple puppies or kittens with diarrhea, treatment of all in contact animals should be considered.

Since many of the gastrointestinal parasites that infect dogs and cats are transmitted by carnivorism, they should not be allowed to hunt or be fed raw meats. Additionally, infection of by many parasites results from ingestion of contaminated water. Clinical disease in some parasitized animals can be lessened by eliminating stress and providing a quality diet and clean environment.

Unless signs of bacteremia are present or signs are persistent, most bacterial enteritis cases are now treated by diet change and probiotics. If antibiotics are needed, Clostridium perfringens and bacterial overgrowth generally respond to treatment with tylosin, metronidazole, ampicillin, amoxicillin, or tetracyclines. The drug of choice for campylobacteriosis is erythromycin; however, oral administration of tylosin or quinolones is often less likely to potentiate vomiting. Salmonellosis should only be treated parenterally due to rapid resistance that occurs following oral administration of antibiotics. Tylosin can be administered at either 5 mg/kg or 15 mg/kg per dose; 25 mg/kg was shown to not be needed in one study. Boxer colitis is due to E. coli and should be treated with enrofloxacin at 5 mg/kg, PO, daily for 6 – 8 weeks. Appropriate antibiotics for the empirical treatment of salmonellosis while awaiting susceptibility testing results include ampicillin or trimethoprim-sulfa; quinolones are also effective. Some animals with infectious diarrhea will respond to the administration of a probiotic. We recently showed administration of **Enterococcus faecium** SF-68 (FortiFlora, Nestle Purina PetCare) lessened diarrhea in shelter cats and to improve metronidazole responses in non-specific diarrhea in shelter dogs. The probiotic is not resistant to metronidazole and so both can be administered simultaneously.

Helicobacter spp. infections are usually treated with the combination of metronidazole, amoxicillin, and bismuth subsalicylate in dogs. Clarithromycin or azithromycin may be logical choices in cats since the species is often difficult to treat with multiple drugs. Whether to concurrently administer an antacid like famotidine is controversial but seems to lessen vomiting in some cats.

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WSV18-0129

NURSES (HILLS)

AGGRESSIVE BEHAVIOR IN PETS - WHAT TO DO?

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Aggressive Behavior in Pets-what to do?

This time, mainly aggression in dogs will be discussed. Dogs can be aggressive to humans and this behavior can be sometimes dangerous. Aggression from animals can also break human animal bond that can end up with euthanasia or abandoning the animals which will be a social and public health problem.

Aggression in dogs to the owners are most of the time due to fear, conflict, learned, and not being consistent. However, the famous myth says owner needs to "dominate" the dogs so many owners tends to scold, yell and sometimes even try to hit the dog for their aggression. Aggression from fear and anxiety cannot be fixed with positive punishment. Aggression can be worse from this myth. During this lecture, we will discuss how to diagnose canine aggression and how to treat it.

WSV18-0236

WSAVA DENTAL GUIDELINES

CURRENT CONCEPTS IN PERIODONTAL DISEASE AND THERAPY

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Current concepts of periodontal disease

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Periodontal disease is the **number one** medical condition in small animal veterinary medicine. We will begin this presentation with an overview of the current knowledge as to the pathogenesis of periodontal disease. This will allow us to properly treat the condition. Following this is a discussion of the local and systemic effects of periodontal disease. This will give attendees the ability to improve client compliance with dental recommendations. In addition, a firm grasp of the disease process will improve practitioner understanding of proper treatment modalities.

Due to the plethora of new and concerning information about this condition, treatment and prevention is the subject of significant research. This focus has resulted in numerous new products and procedures to prevent and treat periodontal disease and this presentation is designed as an introduction to these new and future therapies.

Periodontal disease overview

Periodontal disease is the number one health problem in small animal patients. By two years of age, 70% of cats and 80% of dogs have some form of periodontal disease. However, there are generally little to no outward clinical signs, and therefore therapy typically comes very late in the disease. Consequently, periodontal disease may also the most undertreated disease in our patients.

Pathogenesis:

Periodontal disease is generally described in two stages, gingivitis and periodontitis. Gingivitis is the initial, **reversible** stage in which the inflammation is confined to the gingiva. The gingival inflammation is created by plaque bacteria and may be reversed with a thorough dental prophylaxis and consistent homecare. Periodontitis is the later stage of the disease process and is defined as an inflammatory disease of the deeper supporting structures of the tooth (periodontal ligament and alveolar bone) caused by microorganisms. The inflammation results in the progressive destruction of the periodontal tissues, leading to attachment loss. This can be seen as gingival recession, periodontal pocket formation, or both. Mild to moderate periodontal pockets

may be reduced or eliminated by proper plaque and calculus removal. However, periodontal bone loss is **irreversible** (without regenerative surgery). Although bone loss is irreversible, it is possible to arrest its progression. However, it is more difficult to maintain periodontally diseased teeth in comparison to healthy teeth. Additionally, periodontal attachment loss may be present with or without active inflammation.

Periodontal disease is initiated by oral bacteria which adhere to the teeth in a substance called plaque. Plaque is a biofilm, which is made up almost entirely of oral bacteria, contained in a matrix composed of salivary glycoproteins and extracellular polysaccharides. Calculus (or tartar) is basically plaque which has secondarily become calcified by the minerals in saliva.

It is important to note that rough tooth surfaces will greatly increase the speed of plaque and calculus formation. Therefore any condition which detracts from the smooth tooth surface should be addressed. This can be as simple as a bonded sealant or in some cases can be better treated by a composite restoration. Another condition which commonly hastens the onset of periodontal disease is crowding. This will impair the not only homecare efforts, but also the natural cleaning ability of the patient.

Plaque and calculus may contain up to 100,000,000,000 bacteria per gram. More importantly, bacteria become must more resistant to antiseptics and antibiotics than their free living or "planktonic" counterparts. In fact, they are 1,000 to 1,500 times more resistant to antibiotics and antiseptic concentrations need to be up to 500,000 times that which would kill singular bacteria.

Plaque on the tooth surface is known as supragingival plaque. Once it extends under the free gingival margin and into the area known as the gingival sulcus (between the gingiva and the teeth or alveolar bone), it is called subgingival plaque. Supragingival plaque likely affects the pathogenicity of the subgingival plaque in the early stages of periodontal disease. However, once the periodontal pocket forms, the effect of the supragingival plaque and calculus is minimal. **Therefore, control of supragingival plaque alone is ineffective in controlling the progression of periodontal disease.**

Initial plaque bacteria consists of predominately non-motile, gram-positive, aerobic facultative rods and cocci. Gingivitis is initiated by an increase in the overall number of bacteria, which are primarily motile gram negative rods and **anaerobes.** The **specific plaque hypothesis** is based on the fact that these few species are seen in virtually all cases of chronic periodontal disease.

The bacteria in the subgingival plaque secrete toxins as well as metabolic products. Also produced are cytotoxins and bacterial endotoxins which can invade tissues on their own, and in turn cause inflammation to the gingival and periodontal tissues. This inflammation causes damage to the gingival tissues and initially results in gingivitis. Eventually, the inflammation can lead to periodontitis, i.e. the destruction of the attachment between the periodontal tissues and the teeth.

In addition to directly stimulating inflammation, the bacterial metabolic byproducts also elicit an inflammatory response from the animal. White blood cells and other inflammatory mediators migrate out of the periodontal soft tissues and into the periodontal space due to increased vascular permeability and increased space between the crevecular epithelial cells. White blood cells fight the infection by phagocytizing bacteria, but may also release enzymes to destroy the bacterial invaders either by design or after their death. When released into the sulcus, these enzymes will cause further inflammation of the delicate gingival and periodontal tissues. **In fact, the progression of periodontal disease is determined by the virulence of the bacteria combined with the host response.** It is the host response that often damages the periodontal tissues. However, patients with deficient immune systems typically have more severe periodontal disease than those individuals in good health.

The inflammation produced by the combination of the subgingival bacteria and the host response damages the soft tissue attachment of the tooth, and decreases the bony support via osteoclastic activity. This causes the periodontal attachment of the tooth to move apically. The end stage of periodontal disease is tooth loss; however the disease has created significant problems prior to tooth exfoliation.

Clinical Features:

It is important to be familiar with normal features in order to identify abnormal findings. Normal gingival tissues are coral pink in color (allowing for normal pigmentation), and have a thin, knife-like edge, with a smooth and regular texture. In addition, there should be no demonstrable plaque or calculus on the dentition.

The first obvious clinical sign of gingivitis is erythema followed by edema of the gingiva. However, it is now known that the FIRST evidence of gingivitis is bleeding during brushing, probing, or after chewing hard/rough toys. Therefore it is important to realize that normal appearing teeth/gums can actually be infected. If the first stages of gingivitis are not treated, it will progress into edema, spontaneous bleeding, and halitosis.

The development of halitosis in pets is almost always due to periodontal disease. As previously stated, periodontal disease is caused by an increase in anaerobic bacteria. Certain strains of these bacteria will digest protein as their energy source (generally provided by the host). Some amino acids contain sulfur and this digestion creates products called **volatile**

sulfur compounds (VSCs). One of these is hydrogen sulfide, which creates the "rotten egg smell". VSCs are proinflammatory and actually contribute to periodontal inflammation and attachment loss. Therefore, not only are the VSCs produced by periodontal disease, they also directly increase the level of periodontal disease. Consequently, control of halitosis should be part of the treatment for periodontal disease.

Gingivitis is typically associated with calculus on the involved dentition, but is primarily elicited by PLAQUE and thus can be seen in the absence of calculus. Alternatively, widespread supragingival calculus may be present with little to no gingivitis. It is critical to remember that calculus itself is essentially non-pathogenic. Therefore, the degree of gingival inflammation should be used to judge the need for professional therapy.

As gingivitis progresses to periodontitis, the oral inflammatory changes intensify. The hallmark clinical feature of established periodontitis is attachment loss. In other words, the periodontal attachment to the tooth migrates apically. As periodontitis progresses, alveolar bone is also lost. On oral exam, there are two different presentations of attachment loss. In some cases, the apical migration results in gingival recession while the sulcal depth remains the same. Consequently, tooth roots become exposed and the disease process may be identified on conscious exam. In other cases, the gingiva remains at the same height while the area of attachment moves apically, thus creating a periodontal pocket. This form is typically diagnosed only under general anesthesia with a periodontal probe. It is important to note that both presentations of attachment loss can occur in the same patient, as well as the same tooth. As attachment loss progresses, alveolar bone loss continues, until tooth exfoliation in most cases. After tooth exfoliation occurs, the area generally returns to an uninfected state, but the bone loss is permanent.

Severe local consequences:

The most common severe local consequence of periodontal disease is an **oral-nasal fistula (ONF)**. ONFs are typically seen in older, small breed dogs; however they can occur in any breed as well as felines. ONFs are created by the progression of periodontal disease up the palatal surface of the maxillary canines however; any maxillary tooth is a candidate. This results in a communication between the oral and nasal cavities, creating an infection (sinusitis). Clinical signs include chronic nasal discharge, sneezing, and occasionally anorexia and halitosis. Definitive diagnosis of an oronasal fistula often requires general anesthesia. The diagnosis is made by introducing a periodontal probe into the periodontal space on the palatal surface of the tooth. Interestingly, this condition can occur even when the remainder of the patient's periodontal tissues are relatively healthy (including other surfaces of the affected tooth). Appropriate treatment of an ONF requires extraction of the tooth and closure of the defect with a mucogingival flap. However, if a deep periodontal pocket is discovered prior to development of a fistula, periodontal surgery with guided tissue regeneration can be performed to save the tooth.

Another potential severe consequence of periodontal disease can be seen in multi-rooted teeth, and is called a **class II perio-endo abscess.** This occurs when the periodontal loss progresses apically and gains access to the endodontic system through the apical blood supply, thereby causing endodontic disease via bacterial contamination. The endodontic infection subsequently spreads though the tooth via the common pulp chamber and causes periapical infection on the other roots.

This condition is also most common in older small and toy breed dogs; however, this author has personally treated a case in a Labrador Retriever. The most common site for a class II perio-endo lesion to occur in small animal patients is the distal root of the mandibular first molars.

The third potential local consequence of severe periodontal disease is a **pathologic fracture.** These fractures typically occur in the mandible (especially the area of the canines and first molars), due to chronic periodontal loss, which weakens the bone in affected areas. This condition is again, most commonly seen in small breed dogs, mostly because their teeth (especially the mandibular first molar) are larger in proportion to their jaws as in comparison to large breed dogs. Pathologic fractures occur most commonly as a result of mild trauma, or during dental extraction procedures. Although this is typically considered a disease of older patients, this author has personally treated three cases in dogs less than three years of age.

Pathologic fractures carry a guarded prognosis for several reasons including: lack of remaining bone, low oxygen tension in the area, and difficulty in rigidly fixating the caudal mandible. There are numerous options for fixation, but the use of wires, pins or plates is generally required. Regardless of the method of fixation, the periodontally diseased root (s) MUST be extracted.

Awareness of the risk of pathologic fractures can help the practitioner to avoid problems in at risk patients during dental procedures. If one root of an affected multi-rooted tooth is periodontally healthy, there is an even greater chance of mandibular fracture due to the increased force needed to extract the healthy root. An alternate form of treatment for these cases is to section the tooth, extract the periodontally diseased root, and perform root canal therapy on the periodontally healthy root. In cases where periodontitis involving a mandibular canine or first molar is identified during a routine prophy, it is best to inform the owners of the possibility of a jaw

fracture prior to attempting extraction of the offending tooth.

The fourth local consequence of severe periodontal disease results from inflammation close to the orbit which could potentially lead to **blindness**. The proximity of the tooth root apices of the maxillary molars and fourth premolars, places the delicate optic tissues in jeopardy.

The fifth local consequence is described in recent studies which have linked chronic periodontal disease to **oral cancer**. The association in this case is likely due to the chronic inflammatory state that exists with periodontitis. In this way, periodontal inflammation acts as a 'promoter" of cancer, similar to the chronic inflammation from smoking increases the incidence of lung cancer.

The final significant local consequence of periodontal disease is chronic **osteomylitis**, which is an area of dead, infected bone. Dental disease is the number one cause of oral osteomyelitis. Furthermore, once an area of bone is necrotic, it does not respond effectively to antibiotic therapy. Therefore, definitive therapy generally requires aggressive surgical debridement.

In some cases, the bacterial infection may also result in a septicemia. In one case treated by this author, the patient presented with an entire hemi-mandible which was necrotic secondary to osteomyelitis. In this case, the patient required a complete hemi-mandibulectomy.

Severe systemic manifestations:

Systemic ramifications of periodontal disease are also well documented. The inflammation of the gingiva and periodontal tissues that allows the body's defenses to attack the invaders also allows these bacteria to gain access to the body. It is important to note that just established gingivitis (i.e. no attachment loss) is enough to create these systemic effects. In humans, the periodontal surface area comprises a surface area the size of the palm of your hand. This is a large area of infection for the body to deal with. However, if you consider the size of the mouth and teeth of a small breed dog in relation to their body, there is actually a far greater level of infection affecting these patients.

There are a plethora of studies both in the human and veterinary literature which document a link between periodontal inflammation and organ dysfunction. Affected organs include the kidneys and liver, leading to decrease in function of these vital organs over time. Furthermore, it has also been suggested that these bacteria can become attached to previously damaged heart valves (IE valvular dysplasias) and cause endocarditis, which in turn can result in intermittent infections, and potentially thromboembolic disease. Other studies have linked oral bacteremias to cerebral and myocardial infarctions and other histological changes. Additional human studies have linked periodontal disease to an increased incidence of chronic respiratory disease (COPD) as well as pneumonia. Oral bacteremias have also been linked to arthritis and adverse pregnancy affects.

There are many studies that strongly link periodontal disease to an increase in insulin resistance, resulting in poor control of diabetes mellitus as well as increased severity of diabetic complications (wound healing, microvascular disease). Additionally, it has been shown that diabetes is also a risk factor for periodontal disease. Periodontal disease and diabetes are currently viewed as having a bidirectional interrelationship where one worsens the other.

Most critically, periodontal disease is now associated with early mortality. In other words, humans with bad periodontal disease die earlier than those in good periodontal health. In fact, periodontal disease is now viewed as a higher risk factor for early death than smoking!

Conversely, proper therapy of periodontal disease has been shown to have beneficial effects on systemic maladies. The kidney, liver, and heart function have all been shown to improve when periodontal disease is properly treated. Further, glycemic control is increased in patients with good periodontal health. **periodontal therapy**

Methods and products for periodontal disease treatment and prevention can be grouped into three distinct treatment areas:
- 1)   Control the infection (pathogen control)
- 2)   Decrease the amount inflammation and/or bone destruction by the host (host modulation)
- 3)   Re-grow lost bone (guided tissue regeneration)

Pathogen control

It is well known that periodontal disease is initiated by plaque bacteria. Therefore, the basis for periodontal therapy is, and likely always will, be plaque control. Proper plaque control is a four pronged attack based on the level of disease.
- Dental prophylaxis
- Home care
- Periodontal surgery
- Extraction

A. Complete dental prophylaxis should include the following steps:

1. Pre-surgical exam
    1. Decreases "surprises" under anesthesia
2. Proper and balanced anesthesia
3. Supragingival scaling
    1. Mostly with a ultrasonic scaler
    1. Hand scaling ideally follows the ultra-sonic step
1. SUBgingival scaling
    1. Generally with a curette or SUBGINGI-VAL tip
    2. Hand instruments MUST be sharp
2. Polishing
3. Sulcal lavage
4. Oral exam and charting
5. Dental radiology

B. Homecare

Homecare is an absolutely critical part of periodontal therapy. This is because plaque forms in 24 hours, tarter in 3 days and gingivitis in 2 weeks. This means that even with annual cleaning, patients are infected 50 weeks a year. In fact, human studies show that professional cleanings without homecare are essentially worthless.

There are 2 major divisions of homecare, active and passive. Active homecare is defined that the client actually needs to perform work as opposed to feeding a diet or treat, the latter is considered passive.

Active Homecare

As far as homecare is concerned, tooth brushing is still the gold standard. Educate your clients early about the benefits and compliance will increase. Brushing is performed with a toothbrush and veterinary toothpaste. However, mechanical removal of plaque by the bris the most important part of periodontal care. The toothpastes typically only provide flavorings and anti-tartar agents, neither of which is actually helpful for control of periodontal disease.

Antiseptics such as chlorhexidine and zinc ascorbate can be good adjunct therapy for periodontal disease. However, as above, plaque bacteria are very resistant to antiseptics and therefore mechanical removal of plaque is the most important part of periodontal care.

As great as **effective** toothbrushing is, this is rarely the case. Toothbrushing needs to be performed correctly on a very regular basis. If a client stops brushing even for a short time, gingivitis will return. Since it has been shown that less than 1% of clients brush their pets daily, this is rarely a great choice. Further, it is very difficult to access the distal teeth as well as the linguo-palatal surfaces. It has been shown that brushing is effective on rostral teeth (canines and incisors) but less so on premolar and molar teeth. Chew based "passive" homecare is more effective on the chewing teeth. Therefore, a combination of the two is likely best.

Passive homecare

Passive homecare is mostly chew based removal of plaque. As far as "passive" methods of homecare are concerned, many available products have NO scientific evidence behind them. Essentially all pet store products have no studies.

Further, most studies just look at overall plaque and calculus reduction, not WHERE the reduction occurs. This may or may not indicate true effectiveness against periodontal disease. This is because the decrease is generally at only the incisal edge to middle of the tooth and does not reach to the gingival margin where the disease actually occurs. This may or may not indicate true effectiveness against periodontal disease Therefore, when you are determining what products to recommend to your clients, ideally look beyond just plaque and calculus control and determine **where** that control occurs.

Softer and more pliable products are not only safer in general, they should clean all the way to the gumline.

C. Periodontal Surgery

The other "new" form of pathogen control **should be** periodontal surgery. As discussed in the last article, pockets greater than 3-mm are pathologic and in need of therapy. All pockets between 3 and 6-mm should be treated with closed root planing and ideally the administration of a sustained release local antimicrobial. Pockets greater than 6-mm or furcation level II and III **require** periodontal flap surgery to effectively clean the root surface and allow for reattachment and infection control. These procedures can be learned by a general practitioner and require minimal investment in equipment. If this is not an option, these teeth should be extracted.

D. Extraction

While extreme, the ultimate in plaque control is extraction. This will completely remove the plaque retentive surface of the tooth. It is the actual cure for gum disease. Dental radiographs will greatly facilitate the procedure.

This author is a big believer in minimally invasive surgery. Use small, sharp luxating elevators, minimal bone removal, and envelope flaps for extractions.

BONE REGENERATION

Regenerating bone lost via periodontal disease is another weapon in the fight against periodontal disease. This is combined with periodontal flap surgery to clean and regenerate the lost bone. The technique of guided tissue regeneration (GTR) has been around for decades, but recent advances in barriers and bone grafting has markedly improved the success rates. Regardless, there are only a handful of conditions which carry a good prognosis for bone regeneration. The best prognosis is seen with 3-walled periodontal pockets (typically seen on the palatal aspect of the maxillary canine and distal aspect of the distal root of the mandibular first molar) and class II furcation lesions. Since these are quite common in small breed dogs, there are a large number of patients who would benefit from these procedures.

The theory of GTR is that the down growth of faster healing soft tissue must be prevented to allow the slower growing bone and periodontal ligament to repopulate the periodontally induced bony defect. GTR involves creating a periodontal flap and performing open root planning to create a clean root surface for healing. After this is accomplished, the defect is filled with bone augmentation and a barrier membrane placed. There are numerous products currently utilized on the human side, however currently the products of choice for most veterinary dentists are cancellous freeze-dried demineralized bone for the graft and demineralized laminar bone sheets[as the membrane.

HOST MODULATION

This is an exciting new area of periodontal therapy. It is the use of products to decrease the inflammatory response to bacterial plaque. In this way it can lessen gingivitis and in some cases decrease the amount of alveolar bone loss. Some products are drugs, but there are and increasing number of nutraceuticals in this segment.

Probiotics have been shown to be very effective at improving oral health. They can be administered orally, bit are more effective when rubbed on the gums. Additionally, they have been shown to decrease pocket depths when injected into a periodontal pocket.

Fatty acids are well known for their anti-inflammatory effect on skin and joints. They have also been shown to be effective against periodontal disease. In particular, a veterinary labelled product can be topically applied for maximum local effect, but when swallowed also provides joint support.

Other agents in this category are CoQ_{10}, antioxidants, and proper overall nutrition.

CONCLUSIONS

Periodontal disease is by far the most common disease process in small animal veterinary patients. It is particularly common in small and toy breed dogs. Not only does it create local infection and can lead to tooth loss, there are numerous negative local and systemic effects of untreated periodontal disease. In fact, on the human side periodontal disease is known as the "silent killer". Proper care of periodontal disease is critical for the overall health of the patient.

The basis for therapy of periodontal disease is plaque control. This is achieved by a combination of professional cleanings, periodontal surgery, extractions, and most critically homecare. It is critical to select therapies (particularly homecare) which are effective at and below the gumline. Recently, guided tissue regeneration and host modulation have emerged as additional options for combatting periodontal disease.

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SVA PRACTICE MANAGEMENT

HOW BUILDING A VIBRANT, ONLINE COMMUNITY ON FACEBOOK CAN HELP YOU COMBAT

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1. **SS - How building a vibrant, online community on Facebook can help you to combat Dr Google**

As vets we've all experienced that frustrating moment when a client comes into the practice armed with a pile of print-outs courtesy of Dr Google. Often the client has misunderstood the situation or found 'false news' and it can be time consuming and difficult to talk them around and regain their trust in your diagnosis and treatment plan. How can we ensure that the veterinarian's voice is heard loud and clear in this age of Dr Google?

People love talking about their animals and this gives vets a unique opportunity to use Facebook as a marketing and communication tool. Many vets' Facebook pages are not run to maximum effect and a few simple changes can make a huge difference to the number of local owners reached. We look at case studies and 5 easy steps to take your Facebook page to the next level and start making it work for you.

When owners are concerned about their pet's health their first instinct is probably not to call the vet, they are more likely to consult friends on social media, Google or watch videos before deciding what they should do next and without forward planning by the vet practice owners are unlikely to think of looking to see what their vet has to offer online. Social media offers a valuable opportunity to become part of the daily lives of our clients so that turning to their vet online becomes their first action.

Becoming part of the daily lives of clients via social media takes planning, time and budget but will be richly rewarded with brand exposure and an engaged group of local pet owners not only ready to engage with educational messages but also to provide feedback and word of mouth recommendations to their own online communities. Vet practices should be aiming for an engaged community of local pet owners with their vet practice at the centre of the community.

Building an engaged community will require:

• Regular posting

The more frequently you post the more likely it is that your content will be seen. We recommend posting daily as a minimum, the practices that achieve the best results post several times during the day. Posts should not be made closer together than every 4 hours as the most recent post will stop the previous post from being seen prematurely.

• Conversations

Social media is designed for two way conversations and if you want your content to be seen it is important to get your community to talk back to you. Avoid posting closed statements or comments that are difficult to engage with. Try using questions to give the community an easy way to engage with you.

• A genuine interest in your community

It is crucial to reply when a member of the community posts a comment or photo back. Sometimes simply clicking like can be enough but the practices that achieve the best result will reply back in a conversational way with the commenter.

Every community of pet owners is different, make a note of posts that your community has responded to well in the past and topics they seem interested in. Ask them about their pets and their lives.

• Ads

Facebook has deliberately tweaked its algorithm so that if businesses want to reach their full potential they will need to pay for Facebook Ads. Ads can be used to build likes or to ensure that everyone that has already liked the page will see the content. Ads can also be targeted to people who have visited the practice website or responded to an email.

Content planning is important to make sure that your educational messages on Facebook back up your communications offline. Start with your business aims, ask staff which topics they wish more owners understood better and think of questions that you and your staff are commonly asked by owners.

Social media really lends itself to informing and educating pet owners. Messages can be delivered in a drip feed and the same message can be delivered several times in very different ways covering all learning styles.

WSV18-0216

SVA FELINE

UPDATE ON FELINE ESOPHAGEAL DISEASES: NOT THAT RARE AFTER ALL

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Clinical manifestations of oesophageal disease can include regurgitation, dysphagia, odynophagia (pain on swallowing), ptyalism, and exaggerated swallowing. Regurgitation has been defined as the passive expulsion of ingested material (food/liquid) from the oesophagus and stimulated by local events affecting oesophageal structure/function. Attempts should be made to distinguish regurgitation from vomiting. In contrast to regurgitation, vomiting is often preceded by prodromal events such as vocalising, hypersalivation, retching, and abdominal contractions. In theory, this sounds simple; however, this can pose as a true clinical challenge for many practitioners due to their secretive lifestyles and also for the tendency of these patients to not uncommonly experience overlapping co-morbidities (e.g. 'IBD'àvomited trichobezoaràoesophageal foreign bodyàoesophagitisàregurgitation AND vomiting). To add even more complexity, anorexia, coughing, dyspnoea, and pyrexia may also be noted as a possible sequela to oesophageal disease due to aspiration of contents or even oesophageal perforation.

It's paramount for the practitioner to consider both the patient's signalment **and detailed** clinical history/examination to assist with decision making. In 2018, astute owners have beautifully utilised digital information (i.e. smart phone videos) to assist the clinician in making a preliminary diagnosis of oesophageal disease and guide diagnostic decisions.

Oesophageal Disease You'll Likely Diagnose 2-3x/year in Your Feline Career!

1) Oesophagitis

Clinical signs of oesophagitis usually develop within 1 to 3 days following an insult e.g. reflux from previous sedation/GA, access to caustic medications (e.g. doxycycline, clindamycin) [1-3] , previous local trauma (vomition of trichobezoar), or from thermal injury. Nonspecific lethargy, anorexia, salivation, and vague oral discomfort may precede the onset of regurgitation. In cats, exaggerated attempts to swallow with the head and neck extended, is often accompanied by gagging, retching, odynophagia (pain on swallowing), are often clues of oesophageal pathology.

Two published studies had evaluated the dynamics of tablet/capsule swallowing in cats and highlighted how this phenomenon occurs [4-6] In one study, tablet transit times showed that tablets and capsules made it to the stomach within 5 minutes post-administration 37% and 17% of the time, respectively. In the other study, capsules remained in the oesophagus for more than 4 minutes on 53% of occasions. It's clear that oesophageal entrapment occurs commonly after tablet and capsule administration in cats, and if the tablet/capsules (or their contents) are potentially irritating to tissues, it's easy to understand how oesophagitis occurs after these events. Interestingly, both of these studies demonstrated that the administration of a small amount of food or a small bolus of water after tablet/capsule administra-

tion was highly successful in promoting prompt and complete passage of tablet/capsules into the stomach.

In oesophagitis, survey thoracic radiographs are usually unremarkable except for the occasional presence of small amounts of air in the oesophagus. In some cases, the underlying cause of oesophagitis is identified (e.g., foreign body, hiatal hernia, or oesophageal mass. In cases of hiatal hernia, the displaced stomach may be identified as a mass in the dorsocaudal mediastinum cranial to the hiatus. Contrast oesophagrams are normal in mild cases; however, the mucosal surface may appear irregular with secondary hypomotility in severe cases. Segmental narrowing of the lumen can result from spasticity, intramural oedema, and focal indistensibility caused by inflammation, which can be difficult to differentiate from a developing stricture.

Oesophagitis is usually an endoscopic diagnosis based on mucosal abnormalities. If gastro-oesophageal reflux is the underlying cause of oesophagitis, lesions are most severe in the distal esophagus and the gastroesophageal junction may appear wide open.

Treatment of oesophagitis:

Feeding

Soft, low fat/high protein diet should as it may improve the lower oesophageal tone and minimise delays in gastric emptying. Elevated feeding may reduce episodes. For severe cases PEG tube may be required.

Mucosal protectants
- Sucralfate suspension given PO: 100-200 mg/kg BID/TID

Gastric antacids

To reduce gastric acidity and thereby help prevent further esophageal damage during reflux
- Omeprazole: 1mg/kg PO BID (this is preferable to less potent H2 blockers)
- Ranitidine: 1-2 mg/kg PO bid/tid
- Famotidine: 0.5 mg/kg sid/bid

Other drugs
- Analgesia (e.g. buprenorphine)
- Motility modifying agents (e.g. cisapride) can be of value to increase lower oesophageal sphincter tone and enhance gastric emptying, thereby reducing gastric reflux

2) Oesophageal Stricture

An oesophageal stricture should be suspected when regurgitation develops 1-4 weeks post potential suspect oesophageal injury. Clinical signs are similar to, and often impossible to differentiate from, oesophagitis. The appetite is often good, or even considered ravenous.

Strictures may be single or multiple. Oesophagoscopy +/- fluoroscopy are the most reliable method(s) for diagnosing oesophageal stricture(s) and also for determining the lumenal diameter, stricture length, and presence of associated oesophagitis. Single strictures are found in 80% of patients, but diffuse oesophagitis can result in 2 or 3 strictured areas in some cases.

Management can include endoscopically guided balloon dilatation[7,] or rarely, a stent[8]. The balloon catheter can be passed through the lubricated operating channel of many endoscopes or guided alongside the endoscope to visualize the breaking down of fibrous tissue in a controlled manner. The procedure is repeated at 3 to 5 day intervals for a minimum of three treatments. The total number of dilatations is variable (averaging four

times, ranging three to 10) and is determined by the severity of the stricture and the clinical response.

Prognosis of oesophageal stricture:

Although some severe cases require prolonged therapy, a majority of cases seem to achieve good and acceptable clinical response to balloon dilatation. Some cats will experience complete clinical whilst others may be plagued with only partial response.

3) Oesophageal Foreign Body

Oesophageal foreign bodies are an occasional problem caused by trichobezoars, string, needles, fishhooks, pins, **hairballs,** and very occasionally, bones (especially V-shaped avian bones). Oesophageal FBs usually lodge at the thoracic inlet, the base of the heart, or the hiatus of the diaphragm because of the constricting effect of surrounding soft tissue structures in these areas. The extent of secondary oesophageal damage depends on the type of object, its size and shape, and the duration of time in contact with the mucosa.

Oesophageal Disease You'll Likely Diagnose 2-3x in Your Feline Career!

4) Oesophageal Hypomotility (Megaoesophagus)[9]

Oesophageal hypomotility refers to a decrease in oesophageal peristalsis. Megaoesophagus is a flaccid dilated oesophagus resulting from a severe diffuse motility disorder.

Both congenital and acquired forms of megaoesophagus occur in cats. A hereditary form of megaoesophagus has been suspected in young cats, especially Siamese. Siamese cats with megaoesophagus frequently have a concurrent gastric emptying disorder with the underlying cause of acquired megaoesophagus being unknown. Occasionally, systemic neuromuscular disease is recognised as a cause of megaoesophagus, for example, myasthenia gravis (secondary to thymoma), tick paralysis, or even dysautonomia/Key Gaskell.

Symptomatic treatment for megaesophagus:

- Elevated feeding with small frequent meals of varying consistencies to see which is best tolerated

- Feeding high quality, calorie-dense foods

- Promotility agents (metoclopramide, cisapride 1mg/kg PO q 8h or 1.5mg/kg PO q 12h) can be trialed as it increases motility of the oesophageal smooth muscle, but generally the response to these agents seems to be poor since most of the oesophagus in the cat is composed of skeletal muscle and thus it's efficacy in megaoesophagus remains questionable.

5) Oesophageal Neoplasms

Primary oesophageal neoplasms are considered 'rare'; however, this may be an underrepresentation. Squamous cell carcinoma is the most common primary oesophageal neoplasm in elderly cats. Oesophageal neoplasia causes chronic progressive signs of oesophageal disease and presents similarly to the aforementioned conditions.. Survey radiographs may be normal or may reveal a soft tissue mass in the region of the oesophagus. Endoscopically, neoplasia usually appears as a focal proliferative mass, which may partially or completely occlude the lumen, often with an accumulation of hair preceding the mass. Surgical resection is not usually feasible or successful in the long term.

6) Periesophageal Masses

Mass lesions arising from peri-oesophageal tissues may cause regurgitation due to compression of the oesophagus with partial or complete obstruction. Mediastinal lymphoma is most common, although any large tumour or abscess arising from mediastinal structures (e.g., thymus, lymph node, lung) could potentially cause secondary oesophageal compression. Lymphoma is mostly in young cats, whereas thymoma occurs in elderly cats. Survey and/or contrast thoracic radiography usually identifies the mass. Lymphoma is diagnosed by fluid or FNA cytology.

In Conclusion:

Feline practitioners must remain astute when assessing cats whose owner report vomiting, ptyalism, 'painful mouth', and weight loss. A structured approach based around the key questions of defining the problems/system/location/lesion provides a robust framework for the practitioner to ensure all relevant diagnostic clues have been considered. Clinical thinking/reasoning skills, once developed, will allow for time efficient clinical assessment and to allow the practitioner to make diagnostically relevant decisions which will enhance client communication and improve the welfare of the cat.

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ORTHOPEDIC SURGERY

FEMORAL HEAD EXCISION: A PRACTICAL APPROACH TO SIMPLIFYING THIS SURGERY AND IMPROVING OUTCOMES

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Learning Objectives

At the end of this session you will be able to:

- Identify the surgical landmarks for correct excision of the femoral head and neck
- Position the limb correctly to ensure the correct plane of excision

The dog is positioned in lateral recumbency with the affected leg uppermost. The procedure is simplified if the leg is free-draped so that the stifle and hock joints are within the sterile field and can be manipulated during the surgery by a scrubbed-in assistant.

An incision is made immediately cranial to the greater trochanter. The incision is centered at the level of the trochanter and extends distally about 1/5 of the femoral length and the same amount proximally.

The subcutaneous tissue is dissected on the same line. The fascia between the biceps femoris caudally and the tensor fascia cranially is incised along the same line from the trochanter distally. The cranial edge of the superficial gluteal muscle is incised and separated from the tensor fascia muscle.

The division between the middle gluteal dorsally and the tensor fascia lata muscle ventrally is developed. The line of this division is the ventral edge of the ilium – this will be at right angles to the midpoint of your initial incision.

If you are struggling to find the division between the middle gluteal and the tensor fascia lata due to fat or hemorrhage or edema from trauma palpate the wing of the ilium and draw a line from the ventral edge of the ilial wing to the greater trochanter. This is the line that the ventral edge of the middle gluteal muscle lies on.

The middle gluteal is retracted dorsally to expose the deep gluteal. Both insert on the greater trochanter. The middle gluteal has a muscular insertion onto the greater trochanter while the deep gluteal has a white tendinous insertion. The middle gluteal is much thicker than the deep gluteal.

Blunt dissect between the middle and deep gluteal muscles just cranial to the greater trochanter. Use a Langenbeck or similar blade retractor to retract the deep gluteal dorsally to visualize the tendon of the deep gluteal.

A partial tenotomy of the deep gluteal tendon is then made. The deep gluteal tendon is cut transversely at right angles to the direction of the tendon for ½-2/3 of its width about 5mm from its insertion on the greater trochanter.

A cut is then made from the dorsal edge of the transverse tenotomy in a cranial direction running parallel with the muscle fibers. This will also be the line of incision along the femoral neck into the joint capsule.

The deep surface of the deep gluteal muscle is loosely attached to the joint capsule of the hip. The deep surface of the deep gluteal muscle is blunt dissected away from the joint capsule.

The Langenbeck retractor is now placed more deeply to retract both the middle and deep gluteal muscles.

You should now be looking at the joint capsule of the hip joint and be able to see the thin capsularis coxae muscle running over the joint capsule.

This is the point where people often get "lost" in the approach. You are looking at the joint capsule covering the femoral head and neck however it is not as easy to see as it appears in some of the surgical approaches texts.

Externally rotate the leg and palpate the femoral head. You can feel a curved "groove" which is the dorsal acetabular rim.

Now incise the capsule directly along the head and neck. There are 3 ways you can identify the line for this incision.

The first and easiest way is that the capsular incision is on the same line as the longitudinal incision through the deep gluteal tendon.

The second is that it is at the most dorsal part of the femoral head and neck. The third is that it runs on the same line as the thin capsularis coxae muscle. Use a scalpel blade and make this capsular incision cutting down on the femoral head and neck.

Continue the capsular incision along the femoral head and neck laterally along the cranial surface of the proximal femur through the origin of the vastus muscles.

Combine sharp (scalpel) and blunt (periosteal elevator) dissection to reflect the joint capsule and associated vastus muscles from the cranial aspect of the proximal femur. The capsular tissue will not elevate and will need sharp dissection with a scalpel blade to elevate it. The vastus muscle will elevate easily with a periosteal elevator if you push at 45 degrees to the bone surface and find the subperiosteal plane.

Reflect the vastus muscles from the cranial surface sufficiently so that you will be able to eventually make the femoral neck cut on a **line connecting the medial**

edge of the greater trochanter with the dorsal edge of the lesser trochanter.

A **Hatt spoon curette** is used to cut the round ligament and luxate the head. This is the best instrument by far for this part of the surgery as it combines leverage of the femoral head with a cutting tip. The Hatt spoon curette is relatively cheap and much better and easier to use than a "hip disarticulator".

Cutting the round ligament requires careful force and is made simpler if the surgical assistant is externally rotating the femoral head to tension the ligament as it is being cut. The curette is used like an ice cream scoop.

When the ligament is completely cut the leg will be able to be externally rotated (supinated) 90 degrees so that the stifle joint and hock joint are perpendicular to the table and the femoral head will "pop out" out of the acetabulum (move laterally).

If you have not completely cut the round ligament you will not be able to externally rotate the leg 90 degrees and the femoral head will remain partly tethered to or within the acetabulum.

The femur is externally rotated so that the stifle is at 90 degrees to normal. Continue to blunt and sharp dissect the joint capsule / vastus muscles so that you can palpate the lesser trochanter. Elevation on the **medial** aspect of the femur is important to achieve this and now the femur can be externally rotated 90 degrees this is much easier than before the ligament was cut. Keeping the scalpel blade inside the joint capsule and in contact with the bone as you elevate the medial capsular tissue prevents any iatrogenic damage.

Push a blunt elevator or other blunt instrument ventral to the gluteal tendon insertion on the greater trochanter to identify where the medial edge of the greater trochanter is. Make a mark on the bone at this point.

The lesser trochanter is on the caudal edge of the medial aspect of the femur and is the point of insertion of the iliopsoas muscle. Palpate either the lesser trochanter or the tubular insertion of the iliopsoas muscle on the trochanter so that you can make a mark on the cranial surface of the femur at the dorsal edge of the lesser trochanter. In some dogs you can visualize the tendon of insertion.

If you cant palpate either the tendon or the lesser trochanter it is probably because you have not elevated the medial capsular tissue far enough ventrally. Progress this elevation if necessary until you can confidently identify the lesser trochanter.

Mark a line on the cranial surface of the femur connecting the medial aspect of the greater trochanter with the dorsal aspect of the lesser trochanter. Ensure the capsule and vastus is reflected sufficiently to achieve this.

Ensure your assistant is holding the femur at 90 degrees external rotation (supination). **This is critical to achieving the correct plane of excision** and limiting the amount of rasping you need to do after the excision.

The assistant can ensure the leg is externally rotated 90 degrees by using the stifle joint and hock joint as "handles" and ensuring that they are both held perpendicular to the table / floor.

The assistant needs to focus on this while you complete the excision. The most common mistake is for the assistant to allow some internal rotation of the limb during the cut. This inevitably leads to insufficient neck being removed with an angular piece of bone remaining on the caudal aspect of the neck which then needs to be removed. It is your responsibility because they are working under your direction. J

A **sagittal saw with a fine sharp blade is the best instrument to make the cut in the femoral neck.** It has the advantage of accuracy, lack of propagation into fracture of the caudal part of the neck and it leaves a smooth surface. Provided the cut plane is correct there is no need to rasp an ostectomy made with a sagittal saw. A good sagittal saw is one of the best pieces of equipment to simplify this surgery.

The other alternative if a sagittal saw is not available is an **osteotome.** This is not as good as a sagittal saw as it creates a rougher cut necessitating rasping to a smooth surface. It also has the tendency to cause small fractures of the caudal surface of the femoral neck. This is less likely with a sharp osteotome that.

It is important to note the difference between an osteotome and a chisel. **Chisels are not suitable** for bone surgery. Osteotomes have a symmetric or equilateral triangular tip and cut in the direction that the shaft is aimed. Chisels have an

asymmetric tip or right angle triangular tip and do **not** cut in the direction the shaft is aimed.

The importance of using a sharp osteotome of an appropriate size for the animal can't be understated.

What about Gigli wire? This needs to be properly placed without entrapping caudal soft tissues and has the disadvantage that when it is placed under tension to make the cut it tends to migrate medially to the narrowest part of the neck which is usually the mid part of the neck. In doing so this leaves a significant part of the femoral neck that is not removed. Insufficient or partial removal of the femoral neck is one of the main causes of postoperative morbidity and poor long-term function after excision arthroplasty.

Confirm that your assistant has the leg externally rotated at 90 degrees – the stifle and hock joints should be

perpendicular to the table.

Place the sagittal saw or osteotome on the line marked on the cranial femur between the greater and lesser trochanters.

Ensure that the saw or osteotome blade are perfectly vertical / perpendicular to the table. By doing this you are cutting through the femoral neck in the direction of the acetabulum. The acetabulum is medial to the femoral neck and so is protecting the neurovascular structures around the hip joint from the blade.

Commence the cut and **concentrate on keeping the sagittal saw blade or osteotome blade "vertical to the world" / perpendicular to the table and on the line you marked between the medial edge of the greater trochanter and the dorsal edge of the lesser trochanter.** Provided that you stay perfectly vertical and, on the line, and that your assistant keeps the leg externally rotated at 90 degrees, you will have made the cut in the correct location removing all of the femoral neck and the femoral head.

An excision arthroplasty rasp, which is designed for this task, is used to smooth the ostectomy if necessary. Typically this is unnecessary with a sagittal saw but is usually necessary with an osteotome.

Take care to completely close the joint capsule over the acetabulum as this is important in interposing thick fibrous tissue between the acetabulum and the femur. The rest of the soft tissues are closed as described in standard surgical texts.

APPROACH TO ELAPID ENVENOMATION

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Acute gastric distension is a common cause of abdominal pain in emergency medicine, though it can sometimes be difficult to appreciate on physical examination. Findings on abdominal radiographs may also, at times, be ambiguous. This talk will cover the approach to common causes of acute gastric distension; including gaseous gastric dilation (GD), gastric dilation and volvulus (GDV), food engorgement and fluid distension.

Gaseous distension

Gas dilation of the stomach typically falls into two different categories; gaseous dilation related to the syndrome of GDV and gas distension secondary to aerophagia. Aerophagia, secondary to respiratory distress, usually only causes mild distension. However, sometimes the gaseous distension can impede caudal displacement of the diaphragm and reduce tidal volume, contributing to respiratory distress. In these patients, decompressing the stomach may relieve some of the respiratory distress. Often these patients have such severe respiratory distress that sedation and intubation is imminent. If the patient is already anaesthetized, then it is prudent to decompress the stomach via orogastric intubation (OGI) if distension is evident. If the patient is conscious, then careful nasogastric intubation (NGI) can be attempted, however the procedure must be aborted if it starts to compromise the patient. Gastric trocarisation is not usually indicated in these patients as the distension is not typically severe or compromising perfusion, and it carries the risk of trauma to abdominal organs.

Gaseous gastric dilation, or dilatation, in a large breed, deep chested dog is a problem that is inevitably situated on the continuum to GDV. Typical history is similar to GDV and includes acute retching, hypersalivation, a distended abdomen and agitation. These signs often occur in the early morning or evening, related to meal times. Physical examination findings are often similar to that of a GDV in the early stages, which are predominantly tachycardia, abdominal pain and tympanic abdominal distension. Some dogs do not have obvious tympanic abdominal distension due to their stomach being mostly under the rib cage. Any signs of vasoconstrictive shock (pale mucous membranes, decreased pulse amplitude, delayed capillary refill time, cool extremities) usually indicate that volvulus is present, but can still occur with severe gaseous dilation. Some dogs with early GD or GDV can present with signs of

vasodilation (injected or red mucous membranes, fast capillary refill time). Regardless, any compromise to perfusion prompts swift intervention.

The four essential steps for a dog with GD or GDV are opioid analgesia, blood volume expansion, abdominal radiographs and gastric decompression. Pre-treatment electrolyte and blood gas analysis is also useful. Acid base abnormalities usually parallel a lactic acidosis, however, some dogs display high lactate with a standardised base excess that does not reflect the degree of lactic acidosis.(1, 2) This may be due to pyloric outflow obstruction soon before the GDV occurred, causing preceding metabolic alkalosis, and some dogs are known to have either over-eating or a gastric foreign body as a feature of their GDV presentation.

Electrocardiographic monitoring is advised if a pulse irregularity is detected; pre-surgical ventricular arrhythmias have been associated with a higher incidence of gastric necrosis and a higher mortality rate.(3, 4) However, these arrhythmias usually involve isolated ventricular premature complexes and usually do not require treatment. An intravenous catheter should be placed promptly and any signs of shock treated with fluid therapy. Gastric dilation, with or without volvulus, causes obstructive shock by compressing the major veins in the cranial abdomen, and reducing venous return to the heart. Expanding blood volume increases perfusion pressure, and therefore venous return. This helps to address hypoperfusion quickly while giving more time to address the cause of obstruction. Crystalloid fluid therapy, such as Lactated ringer's solution, is often the most appropriate choice as these dogs can have some degree of dehydration. The use of synthetic colloid fluids in the pre-surgical patient or a patient at risk of Systemic Inflammatory Response Syndrome, without a strong indication, is controversial due to possible adverse effects and should not be routinely administered.

Blood volume expansion and radiography can sometimes be done concurrently if the treatment area is alongside diagnostic imaging facilities. It is ideal to perform radiographs before decompression in order to make the diagnosis clear. However, if the dog is in moderate to severe shock, decompression should be performed before taking radiographs. Decompression may result in resolution of the volvulus,(5, 6) or make it more difficult to appreciate volvulus on radiographs, but should confirm that it was indeed gaseous gastric dilation causing the problem, even if somewhat deflated. Some would debate that if there were no evidence of volvulus on radiographs, then emergency surgery is not indicated. However, if a dog presents with signs of shock on physical examination then chances are that there was some degree of volvulus present. Delaying surgery puts the dog at risk of complications from gastric necrosis or gastric rupture, ongoing hemorrhage from any ruptured

blood vessels or repeat volvulus soon thereafter.(5, 7)

One of the more difficult decisions is surgical planning for large-breed dogs that have simple gastric gaseous dilation on radiographs and **no evidence of shock**. It is still advisable to perform a gastropexy in this patient to prevent future GDV, however, it may not be urgently required. Sometimes these dogs can wait until the next day to either have a laparotomy or laparoscopy performed. The decision to delay surgery needs to be weighed up carefully in terms of how closely the dog can be monitored until the procedure is performed and whether or not the dog shows any signs of repeat gastric dilation, after the initial decompression, or abdominal pain.

There are two main methods for stomach decompression for gas; gastric trocarisation and orogastric intubation. There are positives and negatives to each procedure and no one method has been shown to be superior over the other.(8) Orogastric intubation (OGI) should be attempted first as it is less traumatic to the stomach. However, if it is difficult to decompress the stomach by OGI or the patient requires general anaesthesia to perform OGI due to non-compliance or the distension is severe accompanied by severe shock, then trocarisation is preferred. Sometimes all it takes is a little decompression via trocarisation to facilitate moving the OGI tube through the fundus. Trocarisation is a procedure that should be done carefully, with appreciation for the location of the spleen and being sure not to leave the stylette in place while the gas is being evacuated. Laceration of the spleen or liver is possible with this procedure.

As emergency laparotomy is usually a large expense, and these dogs typically require a high level of care post-operatively, many owners wish to know the pre-surgical risk of their dog dying before committing to the investment. Researchers have attempted to aid this process by assessing the ability of many pre-surgical factors to predict outcome. Clinical signs for >5-6 hours, hypothermia at admission and the presence of gastric necrosis combined with the need for splenectomy have been associated with a increased mortality. Pre-surgical factors that have shown some association with gastric necrosis or a higher rate of complications include high lactate on admission,(1, 9) high lactate post-fluids,(10) and ventricular arrhythmias(3, 4). However, care must be taken in applying results of these studies to individual dogs; the published mortality rates are often from small studies and some are older studies not reflecting today's standard of care. Also, each individual is unique for it's own risk, which can't be well predicted. Overall, for dogs that are taken to surgery, the discharge rate is usually above 90% if appropriate supportive post-operative care is given.

If owners decline surgery, then there are only two

options; euthanasia and conservative management. Conservative management is not an appealing option. If decompression successfully repositions the stomach, gastric necrosis and perforation, and repeat GDV can occur within the next 24 hours, causing the patient to suffer. For those dogs that survive the initial GDV, most studies support that repeat GDV will occur in nearly all dogs within a year and, most, sooner.(5, 6)

Food engorgement

Food engorgement, or food bloat, can present with similar history and clinical signs to that of GDV.(2) On physical examination, there may be tachycardia, a distended painful abdomen that can be tympanic and hypersalivation. It is prudent to approach these patients in a similar fashion to a GDV case. Opioid analgesia should be given promptly and if the tachycardia does not resolve in response to analgesia, then a small crystalloid fluid bolus should be given. Gastric distension due to food engorgement can be marked on radiographs, however, if there is no gaseous distension and the stomach is correctly positioned, then there is no indication for surgery. Some clinicians induce emesis, which can be productive and reduce stomach size, however this may also increase abdominal pain due to stomach cramping and places the dog at risk of aspiration.

Fluid therapy, analgesia and time are usually all that is required. It is important to monitor electrolyte and acid-base status, as food engorgement can cause a mild free water deficit (hypernatraemia), and metabolic alkalosis due to third spacing of gastric fluid into the food mass. Close monitoring for any development of signs of GDV also is important. Most dogs improve after 12 hours of hospitalisation, with a decrease in abdominal distension, and can go home with instructions for rest and small meals. Mild diarrhoea or soft stool in the 3 days after engorgement is common.

Fluid distension

Acute fluid distension of the stomach is usually either due to gastric stasis or pyloric outflow obstruction. The degree of gastric distension does not usually cause obvious abdominal distension. Gastric stasis is usually associated with moderate fluid distension whereas pyloric outflow obstruction usually only causes distension if there is a component of decreased gastric motility. Gastric stasis is common in critically ill patients, especially in those suffering from Systemic Inflammatory Response Syndrome or recovering from abdominal surgery. Regional peritonitis, for example, due to pancreatitis, is also a common cause. Gastric stasis should be suspected in any critically ill patients that continue to vomit or regurgitate despite antiemetic and prokinetic drugs. Abdominal ultrasonography is useful to confirm suspicions, whereby a large fluid filled hypomotile stomach can be identified.

Fluid distension of the stomach can contribute to dehydration and electrolyte abnormalities via loss through vomiting or regurgitation. The nature of this loss will depend on whether there is duodenal reflux into the stomach and administration of antacid therapy. If there is no pyloric outflow obstruction, often the vomitus has a neutral to mildly acidic pH, as it is a mixture of stomach and duodenal fluid. If there is either a functional or mechanical pyloric outflow obstruction, or gastric hypersecretory disorder, then the majority of the loss will be hydrochloric acid, promoting a metabolic alkalosis in the patient. Potassium will also be lost in vomiting and regurgitation. As the effects on acid-base and electrolytes varies with the type of loss, it is important to monitor these parameters.

Gastric distension secondary to gastric stasis can be uncomfortable for the patient, and promote vomiting or regurgitation. If there is no response to prokinetic therapy, such as metoclopramide or erythromycin, it may be useful to place an NGT and remove the majority of fluid from the stomach. This helps to relieve some of the discomfort and often helps to control the vomiting or regurgitation. It also allows for gastric pH monitoring in order to assess efficacy of any antacids administered and allows the administration of enteral nutrition. Small-volume microenteral nutrition stimulates gastric motility, can improve lower esophageal sphincter tone and supplies essential amino acids to the gut, helping it to repair.

Acute fluid distension may also be due to ingestion of large volumes of water, such as in near-drowning cases. This usually causes vomiting and reduction in stomach size prior to presentation. However, if a patient presents with gastric distension due to water ingestion and requires a general anesthetic, it would be best to decompress the stomach via OGI soon after induction, while keeping the patient in sternal recumbency. This may avoid regurgitation and aspiration while providing mechanical ventilation.

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NAVC SHORT TOPICS FROM EXPERTS

HOW I TREAT HYPOTHYROID DOGS

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how I treat hypothyroid dogs

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Canine hypothyroidism is the most commonly diagnosed endocrinopathy in the dog. There is a large incidence of false diagnoses and unnecessary supplementation. This presentation will discuss the difficulty in correctly diagnosing the disease, recognition of clinical signs, and treatment.

Etiology

Hypothyroidism caused by primary disease at the level of the thyroid is most commonly recognized in dogs. Lymphocytic thyroiditis probably caused by immune-mediated mechanisms is the most common pathologic finding. In these cases, the thyroid gland is infiltrated with lymphocytes, plasma cells, and macrophages, and there is progressive destruction of the follicles. It may need years to have complete destruction of the gland, and that is why hypothyroidism is usually seen in young adults. Clinical signs occur when 75% of the gland is destroyed. There is probably a genetic component of the disease since it appears to be inherited by polygenic mode in colony-raised beagles. Antibodies against the thyroid have been a source of controversy in diagnosing thyroid disease. In some dogs thyroid antibody titers may rise as antigens are released into the circulation from thyroid gland damage. These may be measured and in some cases may indicate progressive disease. Certain breeds may have increased frequency of circulating antibody. Antibodies against thyroglobulin have been associated with routine vaccination in dogs; however, it is not known whether this is associated with the development of hypothyroid disease.Other less common causes of hypothyroidism include thyroid atrophy that occurs when the thyroid parenchyma is replaced by adipose tissue with no inflammatory cells. This is an idiopathic change or may signal endstage lymphocytic thyroiditis. Neoplastic destruction of thyroid tissue also can result in hypothyroidism. Congenital thyroid agenesis or dysgenesis may occur rarely. Trimethoprim-sulfadiazine-induced hypothyroidism has been reported as an example of drug-induced disease and has been postulated to directly interfere with thyroid peroxidase activity and thus directly inhibit thyroid hormone synthesis. Secondary disease, with hypopituitarism, is rarely reported.

Clinical Signs

Hypothyroidism most often occurs in middle-aged dogs (4-10 years). Large breed dogs appear to be predisposed including Golden Retrievers, Doberman Pinschers, and Labrador Retrievers (not documented). Spayed females and castrated male dogs may be at increased risk.

Clinical signs are often subtle and have a gradual onset. Metabolic signs reflect a decreased cellular metabolism including lethargy, exercise intolerance, heat seeking, weight gain, mental dullness, decreased appetite, and constipation. Dermatologic signs are often presenting complaints and include bilateral truncal alopecia that is nonpuritic, alopecia of the caudal thighs and a "rat" tail, loss of guard hairs (puppy coat), seborrhea, chronic otitis, hyperpigmentation, failure of hair growth, secondary pyoderma, or myxedema. Neuromuscular signs including profound lethargy and muscle weakness, peripheral nerve paralysis, slow nerve conduction velocities, type II myofiber atrophy, and loss of peripheral vestibular disease are also reported. Reproductive signs include prolonged interestrus intervals, failure to cycle, and inappropriate galactorrhea in the female. Congenital hypothyroidism results in growth retardation, mental retardation, and disproportionate dwarfism (retarded epiphyseal growth) in puppies. Dilated cardiomyopathy that was responsive to thyroid supplementation has been reported in two Great Danes.

These animals are also predisposed to development of polyglandular endocrine gland destruction and may also develop hypoadrenococticism, diabetes mellitus, and/or hypoparathyroidism. Some hypothyroid animals can also present comatose with cerebral myxedema. Physical examination findings include a mildly overweight to severely obese animal, although some patients may be of normal body condition. Profound lethargy may be present, as well as, hypothermia, bradycardia, skin disease, myxedema of skin, mostly on head, or stunted growth.

A biochemical database should be performed on every animal suspected of having hypothyroidism. This is necessary to identify concurrent disease and determine the metabolic status of the animal before potential therapy is initiated. A CBC may reveal mild/moderate normocytic, normochromic, nonregenerative anemia, potentially from decreased erythropoiesis. Findings on a routine serum chemistry include fasting hypercholesterolemia that may be severely elevated. Hypercholesterolemia is seen in approximately 80% of hypothyroid dogs and can help you identify the disease. Other findings may include fasting hypertriglyceridemia and mild elevations in liver enzymes. Exercise-induced hyperkalemia has been reported in some dogs. A urinalysis is usually normal. ECG abnormalities include a sinus bradycardia and decreased amplitude of the P and R waves.

Diagnosis

Hypothyroidism is an overdiagnosed disease! Baseline hormone measurements can be very helpful in identifying this disease, but are often not interpreted correctly leading to misdiagnosis. T_4 is the major thyroid hormone secreted into the bloodstream and, therefore, would be thought to be the hormone to measure. However, measurement of serum T_4 has consistently confused the diagnosis of the disease because > 99% of T_4 is protein bound in plasma. The amounts and affinity of binding proteins can change by many physiologic and pharmacologic factors and artificially lower the T_4 measurement while thyroid status is actually normal. Measurement of T_4 is useful as a screening test. If the result is in the middle-high normal range, hypothyroidism would be placed lower on the list of differential diagnoses. T_4 in serum is relatively stable and can be sent to outside laboratories for measurement by RIA. Interference by anti-T_4 antibodies can cause spuriously high readings. The practitioner must remember that certain breeds of dogs, notably sighthounds, have lower T_4 than other dogs. The correct breed-associated reference range should be used to interpret serum T_4 measurements.

FreeT_4 (fT_4) measurement is that in which the free hormone is separated by equilibrium dialysis and measured by RIA. This factors out influence of nonthyroidal illness and drugs that lower the concentration of T_4 serum binding proteins and subsequently decrease the total T_4. It is more expensive and takes longer than T_4 measurement; however, it more accurately diagnoses the disease. In one study, 98% of hypothyroid dogs had low fT_4, although a small percentage of euthyroid dogs with concurrent nonthyroidal illness have low serum fT_4.TSH measurement (cTSH) is used to diagnose hypothyroidism in people and takes advantage of measuring the negative feedback (or lack thereof) of fT_4 and fT_3 on pituitary secretion of TSH. As fT_4 and fT_3 decrease in primary hypothyroidism, TSH levels will increase due to lack of negative feedback. In dogs and cats,this test has been hampered by availability of good assay reagents. As is stands now it is not valid as a test on its own. Studies show that 25-38% of hypothyroid dogs had normal TSH levels. In euthyroid dogs with concurrent illness, only 12% had elevated TSH levels. This test has low sensitivity, but can be very specific when combined with fT_4 or T_4 measurement.

TSH-stimulation test is the gold standard of testing for hypothyroidism. It tests for reserve in a hypofunctioning gland. In normal dogs, cats, and birds serum T_4 rises in response to TSH administration. Hypothyroid dogs have a blunted response. Injectible bovine source TSH is available as a research grade compound which may cause anaphylaxis. Human recombinant TSH is available

and has been shown to be effective in dogs. However, its high cost makes routine use prohibitive.

Thyroid biopsy is another way to diagnose thyroid disease. It gives histologic evaluation of gland, but no information on function. These glands are usually atrophied and a surgical biopsy is required. Usually it is not necessary in order to obtain a diagnosis.

Thyroid autoantibodies are circulating antibodies to T_3, T_4, and thyroglobulin in the serum. Elevations of these antibodies may indicate early immune-mediated destruction of the gland. However, there are problems with specificity of antibody production. In a recent large study, 6.3% of samples submitted from dogs with clinical signs of hypothyroidism were positive for thyroid hormone antibodies.

Problems with the diagnosis of hypothyroidism occur when concurrent illness results in euthyroid sick syndrome. This is a syndrome defined in humans. It is a physiological adaption to decrease cell metabolism during periods of illness. Serum levels of T_4 and T_3 are depressed, although the animal is euthyroid at the cellular level. Levels of fT_3 and fT_4 appear to be affected less, but may also be decreased. In addition this can be due to changes in thyroid hormone binding proteins, changes in activation of the 5-deiodinase or 5'-deiodinase enzymes, changes in TSH secretion, or increased metabolism of thyroid hormones. Things that can cause this in dogs include hyperadrenocorticism, exogenous glucocorticoid treatment, diabetes mellitus, starvation, systemic diseases, and pyoderma. These animals are euthyroid and do not need to be supplemented with thyroid hormone. Other factors that can affect thyroid hormone measurement include drugs (especially glucocorticoids, phenobarbitol, and clomipramine) and fasting.

So how does the clinician diagnose hypothyroidism. It can be difficult and confusing. First the clinician should have a clinical index of suspicion. Basal T_4, fT_4, T_4 autoantibodies, and TSH can be measured at the same time. Alternatively, measurement of TSH and T_4 or fT_4 is quicker and gives the same specificity, although the antibody status of the animal is not determined. If clinical index of suspicion is high and initial thyroid testing comes back as normal, the animal should be retested in 1-2 weeks. Animals should be off thyroid supplementation for 6-8 weeks before thyroid testing is attempted.

Treatment

In an emergency situation with myxedema stupor or coma, treat with intravenous L-thyroxine, and supportive care. Oral sodium levothyroxine (L-thyroxine) is the treatment of choice for longterm management of hypothyroidism. The dose is 0.01-0.02 mg/kg PO q 12 -24 hours. Start at a lower dose for dogs with cardiac illness, severly debilitated dogs, or geriatric patients and raise the dose slowly if necessary. Monitor and adjust dose by measuring T_4 4-8 hours post-pill; dogs should be in the high-normal to slightly-high range at that time if on the correct dose. Monitor for signs of hyperthyroidism. Side effects are few. Levothyroxine (0.02 mg/kg) given once per day may be sufficient for most dogs after they are initially controlled. Obtain a pre-pill T4 to determine whether once/day dosing is appropriate for this patient.

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SVA FELINE

LATEST TIPS FOR MANAGEMENT OF FELINE HYPERTENSION

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Introduction

Pathophysiology and incidence of systemic hypertension

Systemic hypertension (SH) occurs secondary to a number of diseases, including chronic kidney disease (CKD), hyperthyroidism, primary hyperaldosteronism, hyperadrenocorticism and phaeochromocytoma. Of these, CKD is the most commonly detected cause of SH, followed by pre- and post-treatment cases of hyperthyroidism. In up to 20% of cats with SH no underlying cause is identifiable and hypertension is classified as idiopathic[1]. Cats with idiopathic SH are geriatric (> 12 years of age) and subclinical renal disease is suspected to be the cause of hypertension in at least some of these cases.

SH is diagnosed in up to 40% of cats with CKD seen in primary care clinics in up to 65% of CKD cases in the referral practice setting. Most cats with CKD and SH have creatinine concentrations of < 300 µmol/L (3.4 mg/dL) (IRIS stage I-III). In humans with CKD, SH is associated with impaired renal sodium handling, excessive activation of the renin–angiotensin–aldosterone system (RAAS) and sympathetic nervous system, structural changes to arterioles, endothelial dysfunction, oxidative stress and genetic factors. Investigations into RAAS activation in cats with SH and CKD have yielded heterogeneous results, and the pathogenesis of SH is yet to be fully elucidated [2].

Measuring blood pressure in clinics

The two indirect methods of blood pressure recommended for diagnosis of SH in conscious cats in clinics are Doppler sphygmomanometry and High Definition Oscillometry (HDO). However, only systolic blood pressure (SBP) readings are accurate using either of these methods. Thus, in clinical practice diagnosis of SH in cats is on the basis of elevated SBP.

In the largest single study to measure SBP in 780 apparently healthy cats[3] using the Doppler method, the median SBP was 120.6 (110.4–132.4) mmHg. Factors independently associated with a higher SBP were increasing age category, being more nervous during assessment, being male, being neutered, and being a stray.

Minimisation of stress is critical to prevent stress-related "white-coat" hypertension in the clinic environment, which can result in large transient increases in BP. Standardised protocols minimise stress and help ensure accurate and consistent BP measurements are readily available[1], and should include time for the cat to acclimatise in a stress free environment, minimal use of restraint and use of a cuff width 30-40% of the circumference of the limb/tail where it is used. The International Renal Interest Society has standardised definitions of hypertension (http://www.iris-kidney.com/guidelines/staging.html) for cats with CKD that can also be applied to other causes of hypertension in cats (**Table 1**). The International Society of Feline Medicine (ISFM) consensus guidelines on hypertension include recommendations for frequency of BP monitoring in cats (Table 2). It should be noted that

Table 1. International Renal Interest Society (IRIS) Classification of Blood Pressure Sub-stages

Systolic blood pressure (mm Hg)	Blood Pressure Substage	Risk of Future Target Organ Damage (TOD)
< 150	Normotensive	Minimal
150-159	Borderline hypertensive	Low
160-179	Hypertensive	Moderate
≥ 180	Severely hypertensive	High

Table 2 ISFM Panel Recommendations for monitoring of Systemic Blood Pressure (SBP)[1]

Category	Frequency of SBP monitoring
Healthy cats 7 – 10 years of age	At least every 12 months
Healthy cats ≥ 11 years of age	At least every 6 – 12 months
Risk factors present including: - underlying diseases that cause secondary hypertension, e.g. CKD, hyperthyroidism - evidence of target organ damage (TOD), e.g. ocular changes, arrhythmia/gallop/heart murmur, CKD, ocular signs, CNS signs	When risk factor first detected and at least every 3 – 6 months

Treatment protocols

For cats with CKD treatment for hypertension is usually initiated when the SBP is 160-179 mmHg and ocular/cardiac/neurological TOD is detected or, in the absence of TOD if SBP is persistently160-179 mmHg (as measured on > 1 occasion on separate visits ideally measured over one to two months), if SBP is ≥ 180 mmHg and TOD is present or, in the

absence of TOD if SBB is persistently ≥ 180 mmHg (as measured on > 1 occasion on separate visits measured over one to two weeks).**Amlodipine besylate** a second-generation dihydropyridine that blocks L-type calcium channels in vascular smooth muscle is the first-choice antihypertensive agent in cats. It is. Due to its long plasma half-life and slow receptor binding and dissociation that results in a relatively slow onset and waning of effect, the drug can be administered once daily orally. To manage SH it is recommended to aim for a target SBP of < 160 mmHg and to recheck SBP one to two weeks after commencing treatment.

The recommended dose rate for cats is 0.125 – 0.5 mg/kg once daily orally, and most cats are administered 0.625 mg to 1.25 mg/cat at diagnosis. A recent study investigating factors influencing the dose of amlodipine required to change SBP in cats with SH identified that cats with a higher SBP at presentation needed a higher dose of amlodipine to decrease SBP to target levels[4]. Plasma amlodipine concentrations were directly related to the dose of amlodipine administered. The decrease in SBP was directly and independently associated with the SBP at diagnosis and the plasma amlodipine concentration. Based on these results the authors proposed a starting dose for cats with SBP of ≥200 mmHg of 1.25 mg per cat once a day[4].

For cats with SH that have SBP ≥ 160 mmHg after > 1 week of amlodipine therapy, treatment options include doubling the dose of amlodipine (from 0.25 – 0.5 mg/kg once daily) or combining amlodipine with a RAAS inhibitor, either an angiotensin-converting enzyme inhibitor (ACEi), e.g. benazepril, or an angiotensin II receptor blocker (ARBs), e.g. telmisartan. The antihypertensive effect of benazepril in cats is relatively mild, thus benazepril is not recommended for first-line therapy of SH in cats.

Telmisartan selectively antagonizes the angiotensin II, subtype 1 (AT1) receptor, the latter which mediates the adverse effects of angiotensin II on the cardiovascular system and kidneys. A field trial evaluating the effect of telmisartan on SB in healthy conscious cats found that doses of telmisartan of 1 – 3 mg/kg/day administered orally as a single dose or split into two equal doses resulted in a significant reduction of SBP and was well tolerated [5]. In cats administered placebo Mean ± SBP was 131 ± 15 mmHg at baseline and 124 ± 14 mmHg after 14d. For cats administered 1 mg/kg telmisartan once daily mean baseline SBP was 130 ± 15 mmHg and after 14 d was 105 ± 10 mmHg, while for cats administered 1 mg/kg telmisartan twice daily baseline SBP was 131 ± 19 mmHg and after 14 d was 91 ± 18 mmHg. Further studies are required to determine dose and efficacy of telmisartan for treatment of feline SH.

Gingival hyperplasia is a dose-dependent adverse event associated with amlodipine administration in humans. Recently, it was described in a cat administered 2.5 mg of amlodipine daily, and resolved after the drug was withdrawn[6].

Cats with SH should be monitored after stabilisation at least every three months. Drug dosages should be decreased in cats showing clinical signs of hypotension (e.g weakness) or if SBP < 120 mmHg.

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ANIMAL WELLNESS & WELFARE

MAXIMIZING QUALTY OF LIFE OF ILL PATIENTS

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MAXIMIZING QUALITY OF LIFE OF ILL PATIENTS

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WHAT DO WE MEAN BY QUALITY OF LIFE?

Despite the strong sense that we understand what QOL is, the term currently defies precise description. This is because QOL is a personal, private, subjective experience, has no 'normal,' 'average,' or any other frame of reference, lacks any units of measurement, and means different things to different people.In animals, QOL is not restricted to what kind of housing the animal has, the type of food he gets, the luxuriousness of her bed, the number of walks he gets per day, what size of yard she has to play in, whether he goes to doggie day care or stays home alone all day, or whether she has animal companions to play with. **And most important, it is not restricted to—or equivalent to—his health status**. It is a compilation of all of these factors and more, and the animal's reaction to and feelings **about** them. So what do we mean by quality of life in animals? It can best be understood as one's level of **enjoyment of life**. In this way, we can view QOL in many ways as very similar (though not quite identical) to **happiness.THE FEELINGS OF QUALITY OF LIFE**

Quality of life in animals appears to be comprised of the balance between pleasant and unpleasant feeling states – known as the affect balance model. In this view, QOL may be seen as a scales, with pleasant feelings on one side and unpleasant on the other. The direction of tipping of the scales represents the individual's QOL. Quality of life increases when the balance tips toward the pleasant feelings, and declines when the balance tips toward unpleasant feelings. A key feature of the affect blanace model of QOL is that it becomes very clear as to which factors in life contribute to QOL. Anything which tips the QOL scales—in either direction—plays a role in the animal's QOL, but those things that do **not** tip the scales do not affect the animal's QOL. MAJOR CONTRIBUTING FACTORS TO QUALITY OF LIFE

Several factors contribute to QOL, all having their influence through their associated pleasant and unpleasant feelings. Those with the greatest influence include:

l **Social relationships—**Social bonds are promoted and enforced by pleasant and unpleasant emotions. Positive social affiliations and companionship elicit pleasant feelings, and separation and isolation elicit unpleasant feelings.

l **Mental stimulation**—Monotonous, unchanging environments elicit highly unpleasant feelings of boredom. Conversely, pleasant feelings are elicited by stimulation, challenges, and mental engagement.

l **Health**—Compromised health involves a wide array of unpleasant feelings. Physical disabilities limit one's opportunities for experiencing pleasurable feeling states.

l **Food intake**—The pleasant taste of food and the unpleasant feeling of hunger both motivate consumption of nutrition to support life, and both may contribute the animal's QOL.

l **"Stress"**—As a contributing factor to QOL, stress refers broadlt to specific unpleasant emotions such as fear, anxiety, loneliness, boredom, and anger. Its influence on QOL is through the feelings associated with these emotions.

l **Control**—In animals and humans, one of the strongest predictors of well-being is the perception of control over meaningful aspects of one's life. The opposite—a sense of lack of control—is associated with feelings of helplessness and depression, and lowered enjoyment of life.

MAXIMIZING QUALITY OF LIFE – GENERAL PRINCIPLES

Maximizing QOL can be summarized by a single principle: **Tip the QOL scales as far toward the pleasant side as possible**. Based on the balance model of QOL, this may be achieved by minimizing unpleasant feelings, promoting pleasant feelings, or a combination of the two. This basic principle applies to all animals, healthy and ill. For animals with a health disorder the main effort is to restore a diminished QOL by alleviating the unpleasant feelings associated with the disease. For animals with disabilities, this most often means restoring or replacing the impaired function in order to regain lost pleasures in life. For healthy animals, the main emphasis is promoting pleasures. In all cases, QOL rises as the scales tip increasingly toward the positive direction.

MAXIMIZING QUALITY OF LIFE IN THE ILL ANIMAL

In the ill animal the QOL balance is tipped toward the unpleasant side because of (1) the increase in unpleasant feelings associated with the disease state, which may consist of a single high-intensity discomfort or multiple low-intensity discomforts; (2) a diminished ability to enjoy pleasant feelings and experiences due to the tendency of unpleasant feelings to focus attention progressively more on the discomfort and progressively less on pleasant feelings; and (3) the impaired opportunities to experience pleasure due to the disabilities associated with the medical disorder. Because of the powerful

effects of the unpleasant feelings, the emphasis of maximizing QOL in the presence of disease is directed toward the alleviation of the discomforts associated with the disease. Restoration of health is the most effective means to regain the diminished QOL, but also effective is alleviation of unpleasant feelings when cure is not attainable. Numerous interventions may help achieve this objective, for example, medications and oxygen supplementation to aide oxygenation, analgesics, antiemetics, laxatives, anxiolytics, antihistamines, corticosteroids, chemotherapy agents, and gentle and soothing human contact such as stroking, petting, and talking to the animal, which research suggests can attenuate feelings of pain, anxiety, fear, and loneliness. Attention should be prioritized according to the distress potential of the specific unpleasant feelings.

It is also crucially important to include mental health and well-being in the spectrum of animal health disorders. Emotional illnesses, such as phobias and separation anxiety, elicit unpleasant feelings as distressing as physical illness. Although the primary focus for QOL in ill animals is the alleviation of unpleasant feelings, the vastly underappreciated potential for QOL maximization—a key element to tipping the QOL scales toward the pleasant side—is the promotion of pleasant feelings. Providing the ill animal with more pleasurable experiences will enhance QOL (this is what "pampering" is—an effort to flood the animal with pleasurable feelings). Sources of pleasurable feelings include social interaction and companionship (with humans and other animals), mentally stimulating and engaging activities (variety, challenges, play, chase-and-pounce games, fetch games, hunting for hidden objects and food treats, outings, interactive toys, leash walks outside, a continuous supply of novel objects to investigate and explore such as cardboard boxes, tree branches, objects), taste pleasures (palatable foods, snacks), human contact (petting, massage, laying in lap), climbing, digging up things, lounging in sunlight, and enjoyable sights, sounds, and smells. Because of the individual nature of QOL, the type and quantity of pleasure-eliciting stimuli must be individualized for each animal. Accordingly, the person who is most familiar with the animal's unique personality and nature is best suited to compile the list of pleasures to be used in the QOL maximization program.It is critically important to be sure that attempts to offer pleasant activities are suitable for the specific disease or disability. For example, when an animal (or human) is very ill and simply desires rest, trying to provide a lot of social interaction or mental stimulation might be unappreciated and possibly even harmful to the healing process. However, in some cases activities which elicit unpleasant feelings may be beneficial to QOL, as long as the net effect is to tip the QOL scales toward the pleasant side. For example, if going on walks leads a dog to feel some discomfort of arthritis, but the walks are highly pleasurable and desired, then continuing the walks would be expected to result in a net improvement to QOL.

CONCLUSION

The paramount objective in veterinary care is to maximize QOL. This goal is accomplished, in both ill and healthy animals, by the dual effort of minimizing unpleasant feelings and promoting pleasurable feelings. This keeps the QOL scales tipped as far toward the pleasant side as possible, giving the animal the greatest possible emotional pleasantness in life. By expanding medicine's focus to include the promotion of pleasant feelings in addition to the traditional medical objective of treating disease, the veterinary clinician's ability to improve QOL in ill animals is greatly enhanced. Moreover, by including all aspects of life as contributors to an animal's QOL – not just those directly related to the medical disorder – the ability for us to enhance QOL increases. Lastly, by including mental health and well-being in our calculation of QOL we can expand enhance even further our ability to maximize the animal's QOL. Pet owners and veterinarians should work as a team to improve each animal's QOL, paying attention to the individual nature of every animal. As research continues to elucidate the emotions, feelings, and diseases of animals, our ability to assess and maximize QOL will steadily improve. As it does, the potential for animals with medical disorders to lead the most enjoyable lives will be greatly enhanced.

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DIAGNOSTIC IMAGING

TIPS AND TRICKS TO OPTIMIZE URINARY BLADDER ULTRASOUND IMAGING

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HOW CAN I GET PROFICIENT AT ULTRASOUND-GUIDED FINE NEEDLE ASPIRATION?

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Introduction

Diagnostic ultrasound is widely available in veterinary practice. Point of care ultrasound, also called FAST scanning is a valuable method for initial assessment of a patient presented to the emergency room in order to determine if free fluid is present or not. If fluid is detected, sampling is indicated in most cases and ultrasound guidance is very helpful, in some cases necessary. Ultrasound in general is a very sensitive method to detect pathology, but unfortunately in most instances it is also not specific enough to diagnose specific disease, or even to decide if a benign or malignant disease process is present. Tissue sampling is therefore often used to further characterize a lesion or an organ where disease is suspected. Ultrasound can also aid in performing cystocentesis especially in patients that are obese or have a small urinary bladder. Diagnostic ultrasound in dogs and cats is enhanced by the opportunity to directly obtain tissue samples but there are risks associated with the procedure such as hemorrhage, leakage of fluid from a cystic structure, or inadvertent puncture of gastrointestinal tract. It is therefore extremely important for ultrasonographers who do fine needle aspiration (FNA) to have the expertise and experience to perform the sampling procedure safely.

Patient preparation

Preparing the patient properly is the first step in ensuring success of the procedure. First of all, the lesion identified has to be determined to be accessible with ultrasound-guidance. This means that there has to be a path between the lesion and skin surface where there are no other organ systems or vascular structures that have to be perforated to reach the target. The target lesion has to be superficial enough for the length of the needled used. Doppler ultrasound should be used to determine how vascular a lesion is and if there is a safe approach without large vessels that may represent a risk for hemorrhage. Sedation is recommended for

fine needle aspiration except for cystocentesis and abdominocentesis. FNA can be performed in the awake animal or with local anesthesia, but unexpected movement is more common and the pain and discomfort of multiple aspirates for the patient should be avoided. Sedation protocols vary and should be selected based on the patient, and also based on accessibility and estimated risk of the procedure. For example, FNA or a large subcutaneous mass is much less risky and challenging than aspiration of the gallbladder or a lymph node. In some patients, general anesthesia may be necessary to completely avoid motion and to provide control of the respiratory movement. Respiratory movement particularly increases difficulty of fine needle aspirates in the cranial abdomen (liver, gallbladder).

Fine needle aspiration techniques

Two basic techniques are available: free-hand technique and usage of a needle guide. A needle guide is a sleeve attached to the ultrasound probe that fixates the syringe in a specific position, and the needle enters the image in a predictable path that is often indicated by a line in the ultrasound image depending on the system. The main limitation of this system is that the angle of needle entry is difficult to modify and very superficial or very deep structures can be hard to reach. Free-hand technique is associated with more of a learning curve but is more flexible as the angle of entry can be adjusted depending on the location of the target. Practice is of utmost importance for ultrasonographers who perform fine needle aspirates, for above mentioned reasons. There are many ways to perform ultrasound-guided FNA. What all have in common is that the needle and ultrasound probe have to be aligned perfectly to allow visibility of the needle as it enters the tissue and this requires practice. Slight malalignment will lead to the needle leaving the image plane, and if the sonographer is not sure where the tip of the needle is located there is no way of knowing if structures other than the target are accidentally punctured. FNA can be performed right or left handed, depending on preference of the ultrasonographer. Care should be taken to always place the needle close to the ultrasound probe in order to enter the field of view, but far enough to not accidentally insert the needle into the rubber covering of the probe which may cause expensive damage to the equipment.

Proficiency in ultrasound-guided FNA

Practice is the key to success. How to go about practice when first starting out? Initial attempts at FNA can easily be made using phantoms. There are commercially available ultrasound phantoms that have "lesions" inserted that are visible in the ultrasound image. Ultrasound phantoms can also be home-made using a plastic container filled with gelatin. "Lesions" can be hidden in the semi-hardened gelatin phantoms using fruits and vegetables or any other small item that is not

gas-filled or made out of mineral/metal. Cut-off fingers of plastic gloves filled with water and knotted tightly make excellent cystic targets to practice cystocentesis. Once the ultrasonographer is comfortable reliably reaching targets in a phantom and to always fully visualize the needle tip throughout the procedure, the move to a live patient is justified. Keeping in mind that it is much easier to see the needle in a phantom than in a real patient where heterogeneity of the subcutaneous fat partially obscures the needle signal in some cases. Easy targets should be chosen first such as cystocentesis, or fine needle aspirates of a large spleen or a large superficial mass that is not well perfused (check with Doppler ultrasound). The most challenging types of FNA should only be attempted when the ultrasonographer is very comfortable with needle guidance. Lymph nodes can be challenging as they are always closely associated with vascular structures. Additionally, they tend to move away from the needle as they are relatively loosely attached in the surrounding fat. Gallbladder aspirates carry the risk of bile peritonitis if leakage occurs after aspiration and to make things more difficult the gallbladder is located very cranially and affected by respiratory motion even in sedated patients. Other higher risk aspirates include lung aspirates where there is a risk of pneumothorax, and aspirates of highly perfused lesions such as thyroid carcinomas. Finally, prior to performing fine needle aspirates a risk – benefit analysis should always be made keeping in mind possible complications and the sensitivity and specificity of FNA in different organ systems.

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INFECTIOUS DISEASES AND GASTROENTEROLOGY (TRANSLATED INTO MANDARIN CHINESE)

PRACTICAL APPROACH TO ACUTE HEMORRHAGIC DIARRHEA SYNDROME IN DOGS

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INTRODUCTION

Acute Hemorrhagic Diarrhea Syndrome (AHDS) is a new name proposed to describe the syndrome known for decades as Hemorrhagic Gastroenteritis (HGE). The new name defines the disease better since a studies out of Germany have demonstrated that affected dogs do not display any gastric inflammation. The syndrome can affect dogs of all breeds and all ages, although middle aged dogs of small and toy breeds seem to be predisposed.

ETIOLOGY

The etiology of AHDS is not known, although abundant **Clostridium perfringens** were shown to be present in the duodenum of affected dogs. Other hypotheses about the etiology include dietary or microbial toxins, and severe dietary indiscretion. It is unclear if clostridial proliferation is a cause or a consequence of the disease. **C. perfringens** enterotoxins and **C. difficile** toxins A/B do not appear to be involved in the pathogenesis. However, a newly discovered pore-forming cytotoxic toxin (NetF) produced by Type A **C. perfringens** has been co-localized with severe necrotic intestinal lesions in affected dogs. In addition, acute enteritis strongly impacts the intestinal microbiota, particularly with respect to species diversity, and causes acute dysbiosis.

CLINICAL PRESENTATION

Most affected dogs are small breed and middle-aged. A large German study reports a predisposition in Yorkshire terriers, miniature pinscher, miniature schnauzer, and Maltese, and a mean age of 5 years. Dogs with AHDS are typically presented with acute onset bloody diarrhea, possibly with vomiting and anorexia. They are usually very dehydrated and in various stages of hypovolemic and/or distributive shock. Lethargy and abdominal pain are also common.

DIAGNOSTIC APPROACH

Dogs with AHDS typically have a high PCV (often above 65%) and a normal or low serum concentration of total solids (or total proteins, albumin, or globulins). White blood cell counts may be normal, high or low depending on the severity of disease. Mild thrombocytopenia is common. Other serum biochemistry changes may include pre-renal azotemia, increased liver enzymes, hypoglycemia, and electrolyte abnormalities. Metabolic

acidosis is often present. Abdominal radiographs are usually of limited diagnostic utility and show an enteritis pattern with fluid and gas-filled small intestinal loops.

DIFFERENTIAL DIAGNOSES

All steps should be taken to rule out diseases with similar clinical presentation (hemorrhagic diarrhea and vomiting) that may come in consideration based on the dog's signalment, environment, history and physical exam. These include parvovirus infection, bacterial infections (Salmonella, Campylobacter, **Clostridium perfringens**, **Clostridium difficile**), severe parasitic infestations, dietary indiscretion, toxicosis (e.g. mushrooms, vitamin K antagonist rodenticides, etc.), intestinal volvulus or intussusception, acute necrotizing pancreatitis, acute liver disease, hypoadrenocorticism, sepsis, and immune-mediated thrombocytopenia.

MANAGEMENT

Management consists in aggressive fluid therapy. Intravenous boluses of isotonic crystalloid solutions (10-20 mL/kg) should be used to treat hypovolemic shock. Perfusion and cardiovascular status should be reassessed every 15 minutes, and further boluses administered as required until normal blood pressure is restored. Fluid deficits should be replaced over a 6-12 h period with crystalloid solutions, adding the maintenance requirements and estimated ongoing losses due to continuing diarrhea. In severe cases with distributive shock that do not respond to fluid boluses, vasopressors should be used. Electrolytes deficits such as hypokalemia should also be corrected. Other symptomatic treatment modalities include antiemetic drugs such as maropitant 1 mg/kg IV or SC, and possibly gastric antacids if the gastric mucosa is compromised due to hypoperfusion (e.g. pantoprazole 1mg/kg q12h IV, omeprazole 1 mg/kg q12h PO, as a less efficacious 2nd choice famotidine 1 mg/kg IV q12h). The PCV generally normalizes in response to aggressive fluid therapy, and the serum protein concentration usually drops to levels indicative of hypoproteinemia with both low serum albumin and globulin concentrations.

Use of antibiotics is not recommended in most cases. A recent study showed that cases of mild to moderate severity do not appear to benefit from antimicrobial treatment when endpoints such as time to resolution of diarrhea and length of hospital stay are compared between dogs given amoxicillin and clavulanic acid and those receiving placebo. Broad-spectrum antibiotics should be administered intravenously only to severe cases with existing or impending sepsis, and those showing mucosal sloughing.

Dogs with HGE / AHDS should be fasted for max. 12-24 h and then offered small quantities of easily digestible food frequently (boiled chicken and rice, adequate commercial prescription diets) in order to support the intestinal mucosal barrier.

Probiotics have the potential to be helpful in the long-term treatment of AHDS. They may modulate intestinal immune function, promote epithelial cell homeostasis, exert neuromodulatory effects, block the effects of pathogenic bacteria, and have nutritional benefits. Probiotics designed for use in dogs and cats such as those manufactured by reputable pharmaceutical or pet food companies are preferred, as over the counter products have been shown not to be as reliable. Some products contain one bacterial strain while others consist multiple strains. They have been shown to shorten the duration of acute diarrhea in shelter cats and in decrease the time to first normal feces in dogs with acute enteritis. Probiotics should be administered for 2-4 weeks to animals with acute enteritis. It may be preferable to delay the initiation of probiotic treatment in dogs with bloody diarrhea and compromised intestinal mucosal barrier until the hemorrhagic diarrhea has resolved.

PROGNOSIS

The prognosis of AHDS is good when dogs are presented early in the course of the disease, and aggressive supportive treatment can be initiated promptly. Most dogs can be discharged after a median hospital stay of 3 days (range 1-7 days). Serious complications may include DIC, sepsis, and aspiration pneumonia in vomiting animals. AHDS may be fatal if the emergent needs of the patient are addressed too late.

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NURSES (HILLS)

PAIN IDENTIFICATION AND MANAGEMENT

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PAIN IDENTIFICATION AND MANAGEMENT

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Pain types in dogs

Behavioural sings

Posture	Changes in behaviour	Facial expressions	Other
Hunched posture Guarding painful area	Withdrawn or uninterested in surroundings Aggressive Clingy Reluctance to move Restlessness Unusual licking or chewing at an area Reduced appetite Aversion to being touched Nausea	May have a 'worried' look above the eyesMay look pinched around the mouth.May show whites of eyes or make darting movements with eyes	Lameness or abnormal gait Vocalisation Muscle atrophy may occur if pain has been chronic

Physiological

- Rapid breathing rate
- Rapid heart rate
- Elevated temperature
- Increased blood pressure
- Increased blood cortisol level
- Increased blood glucose levels
- Salivation
- Weight loss if pain has been chronic

Signs of pain in cats

Behavioural

Posture	Changes in behaviour	Facial expressions
Hunched posture. Tense muscles.	Very quiet. May stop grooming or start over-groom. Unwillingness to move. Lameness or abnormal gait. Muscle atrophy if pain has been chronic. Appetite loss. Hiding.	Half closed or squinting eyes

Physiological

- Rapid breathing rate
- Rapid heart rate
- Elevated temperature
- Increased blood pressure
- Increased blood cortisol level
- Increased blood glucose levels
- Salivation
- Weight loss if pain has been chronic

Common techniques nurses may use to manage pain.

- Provide clean, dry and comfortable bedding at all times.
- Keep the patient clean and dry.
- Gentle handling when moving the patient.
- Patting and talking to the patient.
- Massage may be beneficial. Check this with the vet.
- Toys or bedding from home
- Cats may like a box to hide in or part of the cage door covered for privacy.
- Provide a quiet environment.
- Provide warmth, either localised (e.g. covered hot water bottle) or general (heated room).
- Keep out of draughts.
- Minimal disturbance (e.g. do TPR when medicating)
- Don't wake sleeping patients.
- Regular toilet walks if the patient's condition allows.
- Rotate the patient at least 4 times daily if they are unable to turn themselves.
- Assess wounds and bandages regularly.
- Assess pain levels regularly and discuss the patient's condition with the vet.

Pain scoring

Simple unit-dimensional scale

Type of uni-dimensional scale	Description of scale	Advantages	Disadvantages
Simple descriptive scale (SDS)	3- or 5- point scale where scorer assigns a description based upon their own un-structured assessment of the animal. For example; no pain/mild pain/moderate pain/severe pain.	Very simple to use and understand	No validated Non-linear Definitions of the descriptive words is open to interpretation (very subjective)
Numerical rating scale (NRS)	A score from 0 to 10 is given based upon scorer's own un-structured assessment of the animal, with 0 being no pain and 10 the maximum pain possible	Simple to use More sensitive and reliable than SDS or VAS	Not validated Non-linear (e.g. animal with a score of 8 is not necessarily four times as painful as one with a score of 2) Subjective
Visual analogue scale (VAS)	Based upon the scorer's own un-structured assessment of the animal, a mark is made on a 100m line with the 0mm end representing no pain and 100mm end representing the worst possible pain.	Simple to use	Significant inter-individual variability Subjective

Pain relief medication

Analgesia is a state of reduced sensibility to pain. Analgesics interrupt the ascending pain pathway at various points and therefore suppress the sensation of pain.

Types of analgesics

Various types of analgesics are commonly used in veterinary medicine. They include:

- **NSAIDS**: Non-steroidal anti-inflammatory drugs
- **Local:** Local anaesthetic preparations
- **Opioids:** Narcotic analgesics
- **Glucocorticoids:** Steroidal anti-inflammatory drugs

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NSAIDs

Examples include: Meloxicam, carprofen (Rimadyl ®), ketoprofen, phenylbutazone, aspirin, acetaminophen (paracetamol), robenacoxib (Onsior ®), firocoxib (Previcox ®), derocoxib (Deramaxx ®)

NSAIDs act peripherally as a group inhibiting the production of the enzyme cyclo-oxygenase and reduce the level of prostaglandins in the tissue. These actions reduce the inflammation around the injured tissue and produce direct analgesia at the site of pain.

The cyclo-oxygenase is an enzyme that is responsible for formation of inflammatory mediators, particularly prostaglandins. It can be divided into 2 groups:

- **COX 1**, a 'house-keeping' enzyme

- **COX 2**, an enzyme responsible for inflammation and pain

Older NSAIDs tended to inhibit both COX-1 and COX-2. More recent drugs are being refined to only inhibit COX-2, as COX-1 is a 'good' enzyme.

The prostaglandin production that is inhibited is also not 'specific'; therefore, some 'good' prostaglandins are also inhibited. This includes the prostaglandins that:

- Maintain gastrointestinal mucosal integrity

- Maintain normal platelet function

- Maintain normal renal perfusion

Due to this inhibition of prostaglandins, care must be taken when administering NSAIDS. They are contraindicated in certain situations, including:

- Geriatric patients

- Trauma patients

- Patients with active bleeding or bleeding disorders

- Renal failure patients

- Patients with liver disease

NSAIDs must never be administered together with corticosteroids.

Side-effects of NSAIDS:

- **Gastric irritation -** This can manifest as vomiting, diarrhoea, gastric haemorrhage.

- **Renal Compromise-** Prostaglandins maintain and regulate the haemodynamic and blood flow of the kidneys.

- **Platelet dysfunction-** Prostaglandins are also involved in the induction of aggregation (unification) of platelets. Therefore, NSAIDs prolong bleeding times.

Local anaesthetics

Examples include: Lignocaine, Bupivicaine, Marcaine and Prilocaine.

Local anaesthetics produce reversible block of nerve impulse conduction peripherally and in the spine (epidural), depending on site of administration of drug. This produces a loss of sensation and function to the blocked area.

Area	Application	Example
Nerve/Regional Blocks	Local anaesthetic is injected into or around the nerves of a specific area	Thoracotomy – intercostal nerve infiltration Dental surgery Distal limb surgery
Epidural	Local anaesthetic is injected into the area around the spinal cord	Stifle surgery Large animal caesarean
Topical	Applied directly on the skin/area of pain	

Opioids

Opioids prevent the message from being received in the brain. All opioids have slightly different properties and the choice of drug used can be dependent upon several factors including:

- Patient presentation/disease/injury

- Type of pain

- Potency of drugs

- Side effects that may occur

- Length of duration

- Route of elimination

- Veterinarian preference

Opioids bind to specific opioid receptors in the central nervous system to produce analgesia. Due to each drug having different properties some will stimulate and some will inhibit the various receptors. Therefore, combination of opioids must be used with great care as one can disrupt the effects of the other.

Types of Opioids

Drug	Use	Duration	Other	Side-effects
Morphine	Severe pain	~4hrs	Action is dose dependent Higher doses or repeated does will increase the analgesic effect	Vomiting Constipation Respiratory depression
Methadone	Severe pain	~4hrs	Synthetic opioid Similar to morphine Quicker onset than morphine	Less side effects than morphine No vomiting
Fentanyl	Severe pain	3-5hrs	100x potency of morphine Peak effect 5mins after I/V	Bradycardia
Pethidine	Mild pain	1-2hrs	1/10th potency of morphine	Can cause hypotension/histamine release if given fast I/V
Buprenorphine (E.g. temgesic ®)	Mild – moderate pain	8 hrs	Slow onset of action Overdose will cause no analgesia	Slight cardiovascular effects
Butorphanol (E.g. torbugesic®)	Mild, visceral or somatic pain	~2hrs	Potent anti-tussive	Slight respiratory effects

Summary

- Assess level of pain. Discuss your assessment with the attending veterinarian.

- Provide a clean and dry cage at all times. Patient should be away from draughts.

- Provide soft bedding to reduce pressure sores. Rotate animal if it is not able to move a particular body part at least 4 times a day.

- Provide cats with a nest such as a cardboard box with padding for them to hide.

- Assess periodically if wound and sutures are intact.

- Assess if any unexpected bleeding or seepage from wounds or other sites.

- Check bowels and bladder at least twice per day.

- Ensure bandage is intact, dry and providing the protection and support required.

References

The Animal Industries Resource Centre Course materials – Certificate IV in Veterinary Nursing

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WSAVA DENTAL GUIDELINES

DENTAL RADIOLOGY TECHNIQUES AND BASIC INTERPRETATION

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Learning objective:

Obtaining diagnostic radiograph is necessary to interpret it. Correct projections and exposing techniques are necessary too get the good quality radiograph. The correct way of reading dental radiographs will be presented with presentation of most commonly radiopaque and radiolucent lesions.

Evaluation of dental radiographs starts with appropriate orientation of the image according to established standards.

The key to properly identifying the imaged teeth on standard (analog films) radiographs is the embossed dot, which is near one corner of the film. When exposing a radiograph on standard radiographic films, the convex surface points towards the radiographic tube head when the film is properly positioned. It is not possible to obtain a diagnostic radiograph with the film in backwards, because of the lead sheet on the back side of the film. Therefore, when exposing the film, the embossed dot must be facing out of the mouth.

Interpreting dental radiographs starts with the appropriate orientation. First, place the convex side of the dot towards you. This means you are looking at the teeth as if your eyes are the x-ray beam. This step is done for you on most digital systems. The dot should always be located in such a way that it is not superimposed on structures being imaged. When chemical development is performed, place the clip to hold the film adjacent to the dot. This will provide an area of interest free of interfering artifacts. Next, rotate the film so that the roots are in their natural position (pointing up on maxillary views and down on mandibular). When this is done, it is necessary to determine if it is the left or right side of the patient. For lateral oblique projections (canine, premolar, and maxillary molar teeth) or parallel projections (mandibular molar teeth), the side of the film where the more mesial teeth are located indicates the side that was imaged. In other words, if the mesial teeth are on the right side of the film, it is an image of the right side of the patient. With other projections, such as dorsoventral (DV) or ventrodorsal (VD) images (i.e incisors or canines), the right side of the mouth is on the left side of the film and vice versa for the left side of the mouth. This is similar to a VD image of the abdomen.

To distinguish between mandibular and maxillary images, certain landmarks should be evaluated.

For mandible the presence of the mandibular canal, mental foramina, mandibular symphysis and ventral mandibular margin (cortex).The most rostral mental foramen is located in the second incisor area, the middle at the level of apex of the second premolar, and the caudal is at the level of the third premolar. In dogs, the mandibular second, third, and fourth premolars and the first and second molars should have two roots. In cats there are normally only three teeth caudal to the canine. There are obviously exceptions to these rules (e.g. third root in a molar, fused roots or the presence of the second premolar in cats, and supernumerary teeth)

In maxilla the presence of palatine fissures, incisive canal; the conchal crest rostrally and pterygopalatine fossa caudally. The radiopaque line running across the canine root and just dorsally to the roots of the premolars and molars is the nasal surface of the alveolar process of the maxilla. Nasal structures are visible above the conceal crest with symmetric turbinate details. Typical structures for the nasal cavity are the palatine fissures and incisive foramen. In dogs, the fourth premolar as well as two maxillary molars normally have three roots; however, the second molar often has fused roots. In cats, the zygomatic arch is typically superimposed on the maxillary cheek teeth.

Normal radiographic anatomy. There are numerous structures within the oral cavity that mimic pathologic states depending on the projection. Knowledge of normal radiographic anatomy will help avoid over interpretation.

Normal alveolar bone will appear gray and relatively uniform throughout the arcade. It is slightly more radiopaque "darker" than tooth roots. In addition, it appears slightly but regularly mottled. Alveolar bone should completely fill the area between the roots (furcation) and end at the cementoenamel junction (CEJ). The root canals should all be the same width; allowing for differences in the diameters of the root. There should be no radiolucent areas in teeth or bone. A regular thin dark line (periodontal ligament) should be visualized around the roots.

Periodontal disease. Periodontal bone loss results from the combination of bacterial induced inflammation and host response creating osteoclastic resorption of bone. This resorption will result in crestal bone loss to a level below the cementoenamel junction. This decrease in bone height may also create furcational exposure. Horizontal bone loss is the most common pattern in veterinary patients is horizontal. This appears as generalized bone loss of a similar level across all or part of an arcade. The other pattern is angular (vertical) bone loss. The radiographic appearance of angular bone loss is one area of recession below the surrounding bone. The surrounding bone may be normal or be undergoing horizontal bone loss. Therefore it is common to have a

combination of the two types in the same arcade. Bone loss does not become radiographically evident until 30-50% of the mineralization is lost. Therefore, radiographic findings will always **underestimate** bone loss. In addition, bone loss on only on surface (i.e. lingual, palatal, or facial) may be hidden by superimposition of bone or tooth. This may resulting in a non-diagnosed bony pocket. Always interpret radiographs in light of the complete oral examination findings.

Endodontic disease. Endodontic disease may be demonstrated radiographically in several ways. An individual tooth may have one, some, or all of the different changes listed below. However, only one need be present to establish a presumptive diagnosis of endodontic disease. Radiographic changes can be broken into two major classifications: 1) changes in the surrounding bone, or 2) changes within the tooth itself.

Tooth Resorption. Physiologically, tooth resorption occurs during changing of dentition from deciduous to permanent teeth. The erupting permanent tooth causes resorption of the deciduous tooth root. Persistent deciduous dentition teeth very often undergo resorption even without permanent tooth eruption,and therefore the lifespan and time of functionality of these teeth is often very limited.

The radiographic appearance of different types of resorption does not always relate to the type of disease, however replacement resorption has some typical features. In addition, localization of the lesion also could be linked to the specific type. For example PIRR is often located at the cervical area of the tooth as the consequence of damaged cervical root surface and therefore was previously called a"neck lesion".

The importance of dental radiography in TR cases cannot be overstated. Type 1 lesions typically retain a viable root canal system, and will result in pain and endodontic infection if the roots are not completely extracted. However, the concurrent presence of a normal periodontal ligament makes these extractions routine. With type 2 lesions, there are areas lacking a normal periodontal ligament (ankylosis) which also demonstrate varying degrees of root resorption, which makes extraction by conventional elevation difficult to impossible. The continued resorption in type 2 teeth is the basis for crown amputation therapy It is this authors opinion that teeth with an identifiable root canal on dental radiographs MUST be extracted completely, while teeth with no discernable root canal may be treated with crown amputation. If there is any question, always err on the side of complete extraction.

Neoplasia. Neoplasia is defined as the abnormal growth of cells that is not responsive to normal growth control. Neoplasms can be further classified by their biologic behavior as benign or malignant. Benign masses:

Most benign neoplastic growths will have no boney involvement on dental radiographs. If bone involvement does occur with a benign growth it will be expansive, resulting in the bone "pulling away" from the advancing tumor leaving a decalcified soft tissue filled space in the tumor site. Bony margins are usually distinct. Finally, this expansive growth will typically result in tooth movement.

Malignant neoplasia: Malignant oral neoplasms typically invade bone early in the course of disease, resulting in irregular, ragged bone destruction. Initially, the bone will have a mottled "moth eaten" appearance, but radiographs late in the disease course will reveal a complete loss of bone (the teeth will appear to float in space). If the cortex is involved, an irregular periosteal reaction will be seen.

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SVA PRACTICE MANAGEMENT

IMPROVE YOUR PRACTICE WEBSITE IN 5 EASY STEPS

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Improve your Practice website in 5 easy steps

The practice website is your shop window, your chance to show owners why they should choose your veterinary practice, it can also serve important functions: education, appointment booking, email address gathering. Is your website working hard enough for you? Working out where your website needs to improve or getting a new website made can be a daunting task but a little knowledge and a few tools will ensure you can take charge of the process and create a website that works for your practice. Case studies will be used to show the huge difference a good website can make to your practice and the veterinary business.

1. Brand

Make sure this comes across very quickly on your website. You have a few seconds to convince people to stay on your website and look more into your practice. Make sure they get the message about what makes your practice different and special immediately

2. Avoid Stock Photography

Stock photographs will make your website look bland and corporate, often in stark contrast to the practice itself. Your practice is a gold mine of fabulous photos of pets, owners and staff who come through your door each day. Write a photographer's brief and Invest the budget in professional photography

3. Search Engine Optimisation (Getting to the top of Google)

Most web developers will not see it as 'their responsibility' to ensure that the website is set up in the optimal way for search engines. Make sure you have this covered either in house or with an agency that have an SEO specialist

4. Make your website into a resource

You are the local expert on pet health offline but do people see you in this role online. Make sure you can say to your clients 'I understand you want to look things up on the internet please visit our website'. Online appointment booking, new client registration forms and repeat prescription order forms are very easy to set up and improve the client experience

5. Social Proof

If you were a local pet owner who knew nothing about the veterinary practice how would they know that you are trusted and valued by thousands of clients with their beloved pets. Independent reviews have been shown to increase the conversion to call rate by up to 20% and are a must on any website. Clear social media links can also help with this.

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ORTHOPEDIC SURGERY

TOP TIPS IN HOW TO REDUCE AND ALIGN FRACTURES: MINIMAL EFFORT FOR MAXIMUM OUTCOME

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Top Tips How to Reduce and Align Fractures: Minimal Effort for Maximum Outcome

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DVM - Specialist in Veterinary Surgery

Reduction is the process of either reconstructing the fractured bone to its normal anatomy by restoring the correct position of the fragments or restoring the normal alignment of the limb.

Conceptually reduction of a fracture reverses the process which created the fracture displacement during the injury and needs the application of forces and moments opposite to those that produced the fracture.

Consequently an accurate analysis of the level of displacement and degree of deformation represents a fundamental help in planning the strategies needed to achieve reduction.

This concepts apply despite of the reduction method applied, be this open or closed, manual or by skeletal traction, with or without instruments.

When a bone is fractured, the continuous tension generated by the muscles contraction, provoke shortening and overriding of the bone ends. The initial muscle spasm is aggravated by the concomitant injury of the soft tissues of the affected area. Post fracture oedema and haematoma will form in within few hours post injury and will fill the interstitial spaces and create fluid filled voids along tissue planes around the fractured area. The oedema generate hydrostatic forces that create additional shortening of the fractured extremity and an additional impediment to fracture reduction. After several days the inflammatory reaction in the area accompanied by proliferating changes, leads to contraction of more permanent nature making the reduction attempts even more difficult.

OBJECTIVES OF FRACTURE REDUCTION

In diaphyseal and metaphyseal fractures the aim is to restore as precisely as possible the length, the axial and rotational alignments of the bone. Axial bending in the saggital plane is usually well tolerated unless is generating excessive limb shortening. A moderate degree of varus angulation of the distal segment, is generally better tolerated than valgus angulation that usually leads to significant functional problems.

Articular fractures demand anatomical reduction and absolute stability to enhance the healing of the articular cartilage and make early motion possible.

Residual displacement should not be tolerated in order to avoid the development of post traumatic osteoarthrosis. Unfortunately this is not always possible and a better reduction can not be achieved without additional soft tissue damage, an extended or combined surgical approach, with consequently increased surgical time, all this increasing the risk factors and morbidity.

In this cases we have to consider that "better is enemy of good" and a less perfect reduction can be acceptable for the sake of respecting the local biology.

The secret of reduction is the application of continual steady traction over time. This will fatigue the muscles allowing reduction.

Methods of fracture reduction include open or direct reduction and closed or indirect reduction.

CLOSED REDUCTION

Reduction is obtained by application of traction, countertraction and manipulation of the limb. The fracture is not directly exposed and the fracture area remains covered by the surrounding tissues.

This method is ideal provided that it can be accomplished with minimal tissue trauma.

Traction can be applied either manually or by gravity.

To apply manual traction, the patient, under general anaesthesia, is positioned on a table on lateral recumbence on the side opposite to the fractured limb. A soft rope loop or a padded belt is placed around the axillary or groin region and is secured to the edge of the table near the patient's back. A second rope loop is placed in the carpal or tarsal area and slow, continuous traction is applied. Manual traction is quite demanding and is difficult for the surgeon to be able to apply a slow and progressively increasing traction over the 10 to 30 minutes that are usually necessary to fatigue the muscle. The Gordon's extender is a mechanical device that allows traction to be exerted without the surgeon exerting as much force.

The animal weight can be used to advantage when using gravity to obtain traction. The patient is placed on dorsal recumbence and a piece of tape, gauze or soft rope is placed around the metacarpal or metatarsal area of the affected limb. The attaching material is connected to an infusion stand or to a secure bolt in the ceiling and is pulled enough to raise the animal slightly off the table so that part of the body weight is producing traction on the suspended limb. Traction can also be modulate by temporary lowering of the table.

When adequate traction is obtained, usually after 10

to 30 minutes, is possible to reduce the fracture by manipulation of the more movable fragment.

When performing manipulation always be alert to the possibility of laceration, perforation or compression of neurovascular structures.

OPEN REDUCTION

In open reduction the fracture lines are exposed surgically, and the bone fragments are reduced under direct vision with instruments applied directly to each fragment, usually near the fracture side.

Open reduction is usually indicated for articular fractures, fractures that are unstable and complicated, fractures of several days of duration and those fractures that require internal fixation.

Reduction techniques must be gentle and atraumatic.

A good preoperative planning include a good knowledge of the anatomy and surgical approach to the area.

The surgeon should do any effort to respect any remaining vascularity and to avoid soft tissue stripping in order to preserve the biologic healing potential. Keep in mind that increased injury trauma requires decreased surgical trauma.

Strict haemostasis should be obtained by conscious use of electrocoagulation. A combination of frequent, copious irrigation with Ringer's solution and gentle suction helps to maintain a clean area allowing a good visualization of the operative field. The fragments are handled carefully during exposure and reduction, in order to preserve any soft tissue attachment.

If anatomical reconstruction is possible, the fragments are fixed in place with lag screws, wires or Kirschner wires. Fragments that are to small for internal fixation should be left untouched to preserve their blood supply.

As a general rule, all fragments must be kept weather or not they have soft tissue attachments as they will function as an autogenous bone graft.

Instruments and Methods of Open Reduction.

Bone Holding Forceps:

The fracture can be reduced with application of direct force using bone holding forceps on one or more bone fragments. (FIG 1a -b)
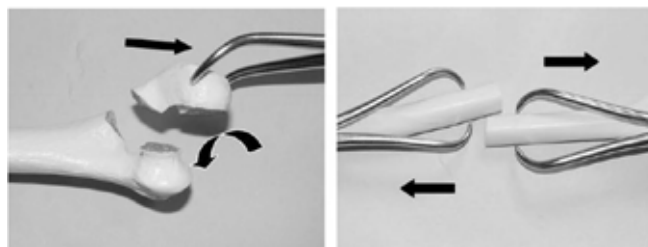
FIG 1-a   FIG 1-b

An oblique fracture overriding can be reduced with a bone holding forceps grasping obliquely each main fragment. With some pressure and simultaneous rotation of the handles the bone is lengthened and the fragments are reduced. (FIG 2a -b)
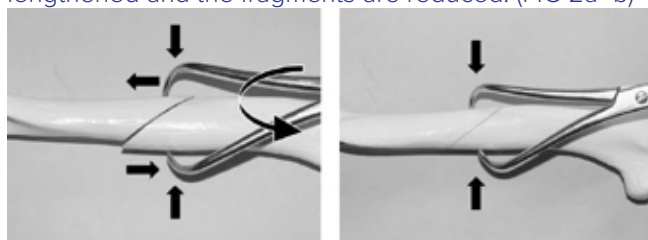
FIG 2-a   FIG 2-b

The disadvantage of this technique is the tendency of the forceps to slip on the bone surface, adding further damage to the periosteal sleeve.

One other application consists of grasping each of the two main fragments of a transverse fracture with pointed reduction forceps, lengthening is achieved by manual distraction while proper axial alignment can be controlled with the forceps. (FIG 3a)
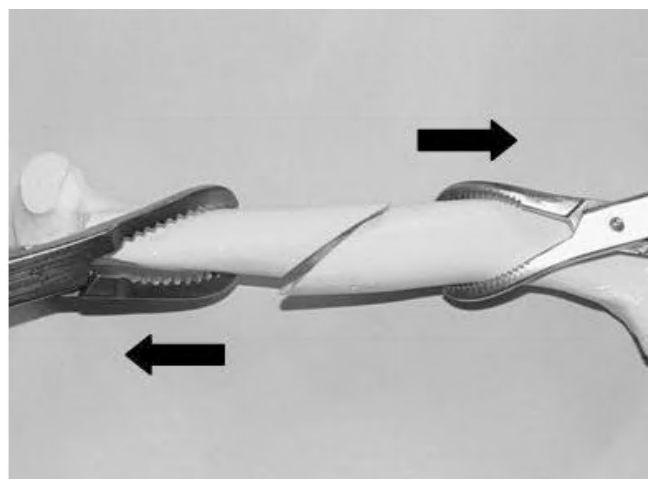
FIG 3-a

In an oblique diaphyseal fracture, reduction is secured with the application of another pointed reduction forceps applied more or less perpendicular to the fracture plane. Fig 3 b
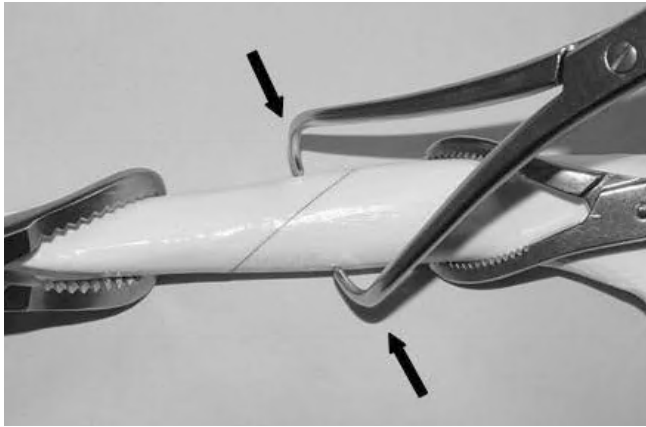
FIG 3-b

In a simple transverse fracture the primary intrinsic stability ensues, allowing removal of the clamps without loss of reduction.

The reduction can be facilitated by the combined levering action of a Hohmann retractor or a periosteal elevator. FIG 3 c
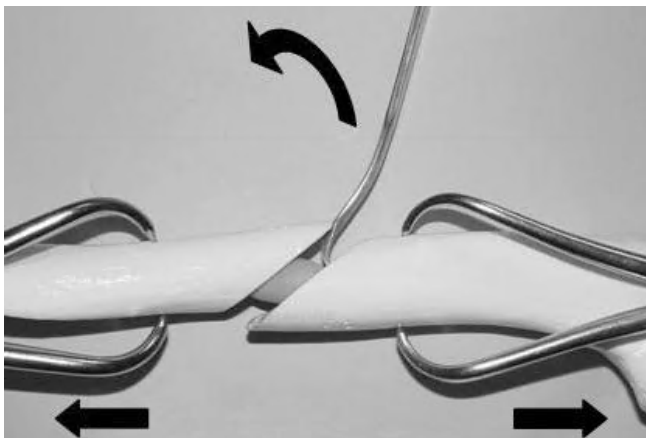
FIG 3-c

All reduction maneuvers with use of bone clamps, depend on the friction between the bone forceps and the bone being greater than that between the bone fragments.

In order to avoid complications related to the use of bone clamps, few guidelines should be followed:

Some of the clamps (e.g. Lohman clamp) cause extensive periosteal stripping. The pointed reduction forceps (Weber tenaculum) are the list atraumatic as they are more gentle to the periosteal sleeve.

Bone fragments should be carefully inspected for fissure fracture lines; application of pressure in a fissured bone fragment or accidental insertion of the tip of a pointed reduction forceps in a fracture line can increase the level of comminution. Weak bone segments can be supported by application of cerclage wire before attempting reduction. Use bone clamps cautiously in young patients,

as it is possible to crush the bone in the attempt of achieving adequate friction between bone and the forceps.

Bone clamps are also useful to correct a residual axial malallignment in the saggital plane when plating a long bone fracture. After the fracture has been grossly aligned, a plate is applied and secured to one fragment, one tip of the clamp (usually a Verbrugen self centering clamp) grasps the anterior or posterior aspect of the bone while the other tip engage on the plate's border. By closing the clamp, is possible to create a shift of the bone fragments and correct the malallignment.

Special reduction clamps are available to overcome the most difficult situations. The Farabeuf clamp and the pelvic reduction forceps (Jungbluth clamp) are designed to grasp screw heads introduced in both sides of a fracture line. This clamps allow application of compression, limited lateral displacement and in particular the Jungbluth clamp permits a certain amount of distraction. This clamps are extremely useful for reduction of pelvic fractures.

Hohmann Retractor

A small tipped Hohmann retractor is an extremely useful instrument that can be used to apply levering or as a pusher to achieve reduction. A classic application of this instrument is in the reduction of simple diaphyseal fractures. The tip of the Hohmann retractor is gently inserted in the fracture gap between the two cortices, is then turned 180 degrees to engage with its tip the cortex of the opposite fragment. Gradual and gentle application of bending force on the retractor allows reduction of the fracture. FIG 4 a- b
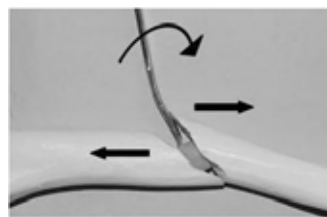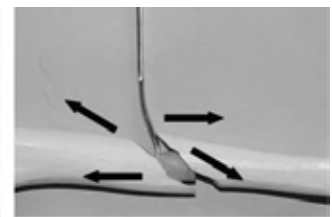
FIG 4-a   FIG 4-b

To correct minimal lateral displacement is possible to turn the retractor so that the fragments can slide on each other. When reduction is satisfactory the retractor is removed by turning and bending to decrease the leverage and sliding it out. Another common application of the Hohmann retractor is in the reduction of fractures of the ileum following the same modalities described above. Other instruments such as small osteotome, a periosteal elevator or a scalpel handle can be used to apply leverage in the same fashion of the Hohmann retractor.

Use of Implants for Reduction

Implants can contribute to the reduction as well as stabilization of a fracture.

An example of this is reduction obtained with a Steinmann pin used as a fracture distractor. This simple method can be applied to fractures of the humerus, femur and tibia. The pin diameter should be about 50% of the medullary canal and is inserted in a normograde manner. As the nail crosses the fracture line and is driven into the distal fragment reduction must occur. Cutting the tip of the pin before its insertion into the distal fragment limit penetration of the pin in the metaphyseal bone.

Reduction is facilitated by the use of bone clamps and by gentle hammering of the pin further distally avoiding rotational action. In a reconstructable fracture, once a satisfactory reduction has been achieved, is possible to secure it with bone clamps, cerclage wire or lag screws and gently withdrawn the pin before plate application. In comminuted fractures where the bone column is not reconstructable, the pin is let in place after optimal alignment and length are achieved and a plate is applied in a splinting fashion.

Application of a plate on a straight portion of the diaphysis help restoring alignment while the plate acts as a splint maintaining reduction before definitive fixation.

In a short oblique diaphyseal fracture, is possible to obtain reduction with the aid of a plate in the following manner. The plate is secured on one main fragment and an independent screw is inserted on the opposite fragment. By pushing and pulling with a laminar spreader placed between the end of the plate and the screw and with reduction forceps is possible to distract and reduce the fracture. Fig 5
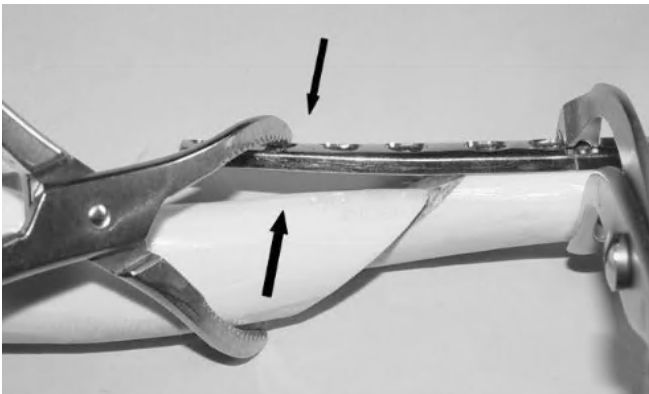
FIG 5

For ileal body fractures distal radius and tibia fractures, a plate can be precisely contoured and secured with screws on one bone segment, a bone clamp placed between the plate and the second bone segment allow gradual reduction of the fracture. When the fracture is reduced the bone clamp is tightened and the plate is fastened with screws.

To correct small displacement and angulation of an oblique metaphyseal fracture, is possible to use a properly contoured plate in an anti-glide function. The plate is applied to one fragment and tightening the screws forces the opposite fragment to glide down the oblique fracture plane, obtaining reduction and maintaining stability at the same time.

Joystick Reduction

It is a very simple technique that allows reduction of small fragments in metaphyseal or articular fractures. Insertion of small threaded K. wires allows manipulation of the bone fragments with or without direct view.

Instrumental reduction has its advantages but come to a price to the fracture biology. The surgeon should keep in mind that "The repeated use of bone clamps and other reduction tools or implants may completely devitalize the fragments in the comminuted area, which may have disastrous consequences for the healing process, including delayed union, non-union, infection or implant failure."

Fracture Distractor

The fracture distractor (Synthes, Ltd., Paoli, Pa, USA; Jorgensen Laboratories, Loveland, Colo) is an instrument that allows distraction of the fracture by means of two pins inserted throw both cortices in the proximal and distal metaphysis of the fractured bone. After insertion, the fixation pins are attached to the distractor with finger nuts. The distraction is applied by turning the wing nuts on the connecting threaded rod. As there is an inherent tendency of curved bones to straighten during distraction, angular and rotational corrections are usually necessary before definitive fixation. The mechanical advantage of the distractor allows easy distraction of the fragments but when is under load alignment corrections can be difficult. The fracture distractor can be used either in open reduction and closed/indirect reduction.

INDIRECT REDUCTION

Increased understanding of fracture healing, brought the concept of "biological internal fixation: that represents one of the major conceptual changes of the last decade.

The principle consists of minimizing the biological damage from the surgical approach and the implant contact. This principle is achieved at expenses of less precise reduction and less rigid fixation. The method of absolute stability by compression fixation is supplemented by the method of relative stability by splint fixation that results in a flexible fixation that stimulates callus formation.

The biological internal fixation come together with the technique of indirect reduction that greatly reduce the surgical trauma and help to keep bone fragments vital.

In indirect reduction the fracture lines are not directly

exposed, the fractured area remains covered by the surrounding soft tissues. Reduction is accomplished by using instruments or implants that are introduced away from the fractured zone. The technique is indicated for mutifragmentary diaphyseal and metaphyseal fractures. The aim of indirect reduction is to restore the overall length of the bone, as well as the axial and rotational alignments.

The technique is more demanding than direct reduction and require an accurate preoperative assessment and meticulous planning. Reduction is controlled by help of image intensifier, clinical checks for the alignment or intraoperative x-ray. The techniques for fixation are: Interlocking nail, external fixation, splinting with conventional plates, plate and rod, bridging the fracture with a locked internal fixator as the LCP (Locking Compression Plates).

CONCLUSIONS

In conclusion either direct and indirect reduction are useful techniques and have their place in the repertoire of the orthopaedic surgeon. A clear understanding of the role of this techniques, together with an informed assessment of the relationship between fracture pattern and soft tissue trauma, lead to correct decisions on strategies and choice of implants compatible with the biological demanding of the fracture.

New principles and methods will continue o develop however some principle will never change:

"There is danger inherent in the mechanical efficiency of our modern methods. Less the craftsman forget that union cannot be imposed but may have to be encouraged for the bone is a plant with its roots in the soft tissues.

When the vascular connections are damaged, it often requires not the technique of a cabinetmaker but rather the patient care and understanding of a gardener."

Dr. Girdlestone (Orthopaedic influence on the treatment of fractures, a clinical study. Oxford 1943)

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VECCS

INTRAVENOUS LIPID EMULSION THERAPY FOR ACUTE TOXICOSES

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There has been much interest in the use of intravenous lipid emulsion (IVLE) therapy for many different types of small animal toxicoses in the last 10 years. Although there is some evidence for its efficacy in specific toxicoses, its unbridled use in clinical toxicology is still controversial.

What is IVLE?

Intravenous lipid emulsion therapy involves injection of a fairly large dose of lipid emulsion over a short period of time in order to counteract the effects of particular toxins. It is usually provided as a sterile 20% solution of soybean oil, egg phospholipids and glycerine suspended in water. It is traditionally used for providing a fat source in parenteral nutrition formulations but in recent times, certainly in our hospital, it is more frequently used as poison antidote therapy. Once the bottle is punctured, it should be discarded within 24 hours due to risk of bacterial growth in the solution. It is ideal to consider each bottle to be single-use only. The IV catheter should be checked for patency before started the infusion and the catheter should be flushed well after use. You do not need a dedicated IV line as it is delivered over a short period of time. However, if using as a continuous rate infusion (CRIs) for more than 1 hour, then attention should be paid to the level of the sterility of the catheter that is used. When considering CRIs, bear in mind that there is no evidence for efficacy of this approach. In general, it is recommended to stop administering IVLE if the serum/plasma is grossly lipaemic.

Mechanism of action

The use of IVLE for toxicoses had its beginnings as an antidote for local anaesthetic overdose. Initial experimental studies showed improved survival after IVLE in dogs and rats that received a bupivacaine overdose.(1, 2) Since then, infusion of IVLE during resuscitation for bupivacaine has been reported to be successful in multiple human case reports.(3) Bupivicaine decreases cardiac adenosine triphosphate (ATP) synthesis. Lipid emulsion is thought to reverse the cardio-toxicity by providing fatty acids to the myocardium and increasing ATP production, thus improving myocardial contractility. It may also increase intracellular myocyte calcium, also assisting positive inotropy. Since this discovery, the use of IVLE for resuscitation of local anaesthetic overdose has been become routine in human medicine.

The second proposed mechanism is creation of a drug-scavenging 'lipid sink', into which lipophilic drugs or toxins are absorbed. In theory, absorption of the drug into an intravascular lipid compartment draws the molecule away from its site of action, giving the body more time to metabolise and excrete the perpetrator. The toxin needs to be lipophilic enough, however, for it to be attracted to the lipid compartment. It is difficult to determine what is lipophilic enough. The lipophilicity of any chemical is described as its ability to partition into octanol, expressed as the partition coefficient or log P. In general, a log P greater than 1.0 means that a chemical is lipophilic, and above ~3.0, it is highly lipophilic. However, there is some variability in the reported log P (depending on how it is measured) and the behaviour of the chemical **in vivo** is affected by it lipophilicity at blood pH, its volume of distribution and other aspects of pharmacokinetics.(4) Therefore, it is difficult to predict if IVLE will be efficacious for drug-scavenging any particular toxin based solely on log P. Generally, the greatest success has been reported for toxins whereby the log P is greater than 5.0.

Previous reports of utility in dogs and cats

There have been many case reports published in dogs and cats reporting the use of IVLE in toxicoses (Table 1). It is difficult to establish the efficacy of IVLE from case reports as often multiple other drugs are administered at the same time, and it is unknown whether or not the treatment actually altered the time course of the toxicoses for an individual patient. There is also likely a publication bias whereby instances of perceived efficacy are more likely to be published than lack of efficacy.

The types of toxins that IVLE has been used for generally fall into two categories; neurotoxins and cardiotoxins. Most case reports comment on use of IVLE to treat neurological signs due to permethrin or macrocyclic lactone toxicoses. Perceived response to treatment include various types of neurological improvement, such as reduced tremors, improved mentation or ability to walk, or reduction in sedative drugs needed. There are also some case reports of use of IVLE in toxicoses that have cardiovascular effects, such as lidocaine and diltiazem. However, the information is too limited to comment on perceived benefits.

There is one study worth mentioning in isolation as the only prospective randomised clinical trial on the use of IVLE in toxicoses.(5) Thirty-four cats were randomised to receive either IVLE or saline placebo (non-blinded) and then clinically staged for signs of toxicoses over the remainder of hospitalisation. The study found that cats that received IVLE showed faster resolution of clinical signs.

The dose administered in most reports consists of a small bolus (usually 1.5mL/kg) followed by 8-15mL/kg over 60 minutes. The use of a bolus was originally designed for resuscitation purposes (i.e. local anaesthetic overdose) and is probably only useful for cardiotoxicity. Use of a bolus for drug-scavenging purposes (i.e. 'lipid sink') is questionable and probably unnecessary. The timing of 'response' to treatment is usually within several hours of the first dose. Many authors comment on repeating the dose or extending the time of CRI; usually in cases where no initial response was seen.

Table 1. Case reports in dogs and cats(6)

Toxin/drug (number of reports)	Mechanism of action	Log P*	Species (number of cases)	Perceived benefit?
Permethrin (7)	Voltage-gated sodium channel blocker	6.5	Cat (14)	Yes (14)
Ivermectin (9)	Glutamate-gated chloride channel blocker	5.83	Dog (10) Cat (22)	Yes (5 dogs, 2 cats) No (5 dogs)(ABCB-1Δ +) Unclear (20 cats)
Moxidectin (4)	GABA and glutamate-gated chloride channel blocker	5.3	Dog (4) Cat (1)	Yes (5)
Monensin (1)	Ionophore	5.43	Dog (1)	No (1)
Minoxidil (1)	ATP-sensitive potassium channel opener	1.33	Dog (1)	Unclear (1)
Baclofen (5)	Substance P inhibitor, GABA agonist	1.3	Dog (8) Cat (1)	Yes (8) No (1 dog)
Naproxen (1)	Non-selective COX inhibitor	3.18	Dog (3)	Yes (3)
Ibuprofen (1)	Non-selective COX inhibitor	3.97	Dog (1)	Yes (1)
Lidocaine (1)	Fast voltage-gated sodium channel blocker	2.26	Cat (1)	Yes (1)
Diltiazem (1)	Calcium channel blocker	2.8	Dog (1)	No (1)
Marijuana (1)	Cannabinoid receptor agonist	5.24 - 6.97	Dog (3)	Unknown (3)

*Retrieved from either pubchem.ncbi.nlm.nih.gov or www.drugbank.ca

Adverse events

The actual rate of adverse events has not been well established as the evidence relies mostly on case reports. There have been two case reports of corneal lipidosis in cats after IVLE treatment, which resolved within a week, and one report of facial pruritis. (5, 7, 8) Other adverse events reported in human medicine include fat overload syndrome, pancreatitis, cholestasis, acute kidney injury, acute lung injury, venous thromboembolism, hypersensitivity and increased susceptibility to infection.(9)

Should I use IVLE?

The benefits versus risk need to be weighed carefully when deciding on whether or not to administer IVLE. In cases where the patient is severely affected by a neurotoxin or cardiotoxin AND there is theoretical benefit based on IVLE's supposed mechanisms of action, then the possible benefit of a single dose of IVLE likely outweighs the risk of adverse effects. There should be some estimation as to whether or not the toxin is likely to still be in circulation, rather than bound to tissue. A repeated dose may be used if there is no response

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or there is clinical regression, however, the benefit of prolonged CRIs is unknown. In hospitals that regularly use regional anaesthesia (especially bupivacaine) then IVLE should be added to the arsenal of emergency drugs available, in case of overdose or adverse reaction.

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NAVC SHORT TOPICS FROM EXPERTS

HOW I TREAT STOMATITIS IN CATS

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The history will generally include anorexia, drooling, gagging, and pain during mastication. Physical exam will typically include a thin pet with unkempt fur. The oral exam will reveal severe stomatitis usually over all teeth. The inflammation will most commonly be worse on cheek teeth than canines and incisors. However, faucitis is the key clinical finding. Severe hyperplastic inflammation to the gingiva can result from periodontal disease, however faucitis will not be present.

A pre-operative blood panel will generally show a marked elevation in globulins (Polyclonal gammopathy) and total protein.

Medical Therapy: Most medical therapies will work for a while, however in general resistance will start within a year or less. In addition, most therapies have side effects worse than the disease process in and of itself. In general, medical therapy is very frustrating to the practitioner and client.

Corticosteroids are the mainstay of most medical therapy today. It is generally very effective at first and is relatively inexpensive for the client. In my experience, injectable (depomedrol 10 mg IM) is much more effective than oral preparations in my experience. However, they will typically loose effectiveness after a year or so requiring higher and higher doses at shorter increments. This generally results in significant deleterious effects. About 10% of stomatitis cases we treat are already diabetic!!!

Antibiotics are safer than steroids but much less effective, especially in long term therapy. They are generally disappointing in their success. Metronidazole and clindamycin are the mainstays of therapy; however Clavamox and amoxicillin can be used as well. Metronidazole may be the antibiotic of choice due to its anti-inflammatory effect.

Other immune suppressive such as Imuran, Cytoxan, Gold Salts, Cyclosporine have been used. However, they are all very expensive with numerous adverse side effects (mylosuppression). Cyclosporine is currently the most commonly prescribed immune modulatory drug (other than steroids) for this disease process. However, its chronic use is somewhat expensive and has been implicated in severe fungal and protozoal infections.

Starting dose is 5-10 mg/kg. Look for a trough level of about 500 ng/ml on regular basis. In most dentists opinion it is only really effective AFTER teeth are removed. However, it has shown promise in resistant cases.

Laser therapy is not proven at all, most clients and RDVM's are very unhappy with the long term results. It is very expensive and short term relief only.

Surgical Therapy: Extraction is currently the ONLY effective long term treatment for this disease process in cats. In our experience, the sooner this is done, the better that cats do both post-operatively as well as long term.

For extractions to be successful, the teeth must be COMPLETELY removed. Therefore post-operative radiographic confirmation of complete extraction of the tooth roots is recommended. Following the insurance of complete removal of the teeth, perform aveloplasty to remove the periodontal ligament and smooth rough bony edges. This is typically performed do this with a rough diamond bur.

Studies report a 60% success rate when all teeth caudal to the canines are extracted, however our experience has not been as good. However, whole mouth extractions have a success rate of approximately **90-95%** for clinical remission. Slight faucitis may remain, but pets are comfortable. In addition, the rare cases that don't completely respond are generally much more responsive to medical therapy.

If there is NO inflammation to the canines or incisors (which is rare), then the owner is given the option of leaving the canines. However, if these are inflamed, all teeth should be extracted.

Resistant Cases

In the rare cases where the teeth have been fully extracted but inflammation and pain continues, other therapies are needed. The current treatment of choice in the USA is cyclosporine. Another option, which appears to work better in Europe is feline interferon. Finally, UC Davis has had some success with Stem Cell Therapy.

HOW I APPROACH A CAT WITH PLEURAL EFFUSION

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How I approach a cat with pleural effusion

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Cats with pleural effusion often have severe respiratory compromise at the time of presentation. Cats with respiratory compromise should be identified early, handled as little as possible and stabilized. Fortunately, most or all of the information required to localise the dyspnoea can be obtained from observation of the patient and a minimally invasive physical examination. The clinician has to juggle stabilization, localisation and owner communication while remaining vigilant for clues which allow ranking of the differentials to formulate a diagnostic plan.

Stabilisation

Triage at reception is important to identify dyspnoeic cats early. All sick cats should be observed in their carriers on arrival at the clinic regardless of the owner's description of the presenting complaint. This is because dyspnoea may be subtle or absent prior to travelling. Such is the propensity of the cat to compensate for gradual onset respiratory compromise by reduced activity that signs may not be seen by even the most observant owner. It is important that all members of the care-giving team, including reception staff, are trained to be vigilant for dyspnoeic emergencies and to take appropriate action.

Where respiratory distress is noted or suspected, immediate stabilisation is indicated. The techniques used and the order in which they are carried out depends on assessment of the individual patient.

- Reduce oxygen requirements by placing the patient in a cool, quiet environment and minimising handling to reduce oxygen demand.
- Supplemental oxygen Options for short term oxygen delivery to the dyspnoeic cat include oxygen chamber, mask and flow-by. The method that is best tolerated by the patient should be used. Struggling must be avoided. An oxygen chamber or cage is a useful way to deliver oxygen without the need for restraint.
- Intravenous access should be achieved at the earliest opportunity
- Light sedation may be beneficial for dyspnoeic cats to reduce anxiety (eg butorphanol).
- Therapeutic thoracocentesis is carried out after im-

aging except where suspicion for pleural effusion is high and the dyspnoea is severe. In this case unguided needle thoracocentesis can be life-saving and the risk of causing significant harm is low.

- Monitoring of respiratory rate and depth should be initiated early so that changes can be readily appreciated.
- Where hypoventilation cannot be controlled by other means, the patient can be anaesthetised, intubated and ventilated.

Presenting signs of pleural effusion

Observation Cats with significant pleural space disease adopt a sternal position with abducted elbows. A restrictive respiratory pattern with increased inspiratory effort is typical. A restrictive respiratory pattern is **rapid** and **shallow.** Conditions which prevent the lungs from fully expanding cause a restrictive respiratory pattern. An increased respiratory rate and decreased inspiratory volume minimises respiratory effort in non-compliant lungs. Another major cause of restrictive respiratory patterns in cats is pulmonary parenchymal disease (eg, pulmonary oedema, pneumonia)

Auscultation helps to distinguish pleural space disease from pulmonary parenchymal disease. Breath sounds are decreased or absent with pleural space disease. Differential diagnoses then include pleural effusion, pneumothorax, intrathoracic mass or diaphragmatic hernia. Where there is effusion, the reduction in breath sounds is more pronounced ventrally and a fluid line may be appreciated on auscultation or percussion. Concurrent pulmonary oedema may contribute to the dyspnoea in left-sided heart failure and produce pulmonary crackles dorsally. Pleural effusion or pericardial effusion can cause muffled heart sounds.

Imaging Pleural effusion can be confirmed with radiography (a single DV view, if patient permits) or thoracic ultrasonography.

Major differential diagnoses for pleural effusion in the cat

congestive heart failure

neoplasia

pyothorax

feline infectious peritonitis

idiopathic chylothorax

These are not the only differentials but they are by the most common causes of pleural effusion of sufficient volume to cause dyspnoea. Data obtained from signalment, history and physical examination can be used to rank this list of common differentials.

On clinical examination of cats with suspected pleural space disease, the following should be noted:
- Jugular veins - are they distended and pulsating? This occurs with increased right heart filling pressures (congestive heart failure, high output failure e.g. hyperthyroidism) or with pulmonary hypertension.
- Are the jugular veins distended without pulsation? This occurs with occlusion of right heart inflow (cranial mediastinal mass, large non-cardiogenic pleural effusion)
- Lung sounds - pleural effusion will often result in reduced lung sounds, particularly ventrally. It may be possible to determine a fluid line on auscultation.
- Heart sounds are they muffled? Consider a pleural exudate, eg pyothorax. Transudates and modified transudates contribute less to muffling of heart sounds.
- Is the cardiac apex beat displaced? An intrathoracic mass or focal accumulation of fluid may displace the cardiac apex beat. (e.g. a left-sided intrathoracic mass would displace the cardiac apex beat resulting in an abnormally loud apex beat on the right and an absent or muffled apex beat on the left.).

Ranking of differential diagnoses is further informed by the gross characteristics (smell, colour, turbidity) of fluid obtained at thoracocentesis. Thoracocentesis is both a diagnostic and therapeutic procedure. A butterfly needle attached to extension tubing, a 3 way tap and a 50 ml syringe. Local anaesthetic can be used. The ventral third of the 7th-9th intercostal space avoiding the caudal rib margins can be used for blind thoracentesis.

Diagnostic samples collected in EDTA and plain tubes and direct smears can be submitted for total protein, total and differential cell count and cytology. Additional testing such will be directed by clinical data and gross evaluation of the fluid. Once the patient is stable additional testing such as repeat imaging (radiographs or CT), NT-proBNP, echocardiography may be indicated.

Indwelling chest drains suitable for cats include

http://www.milainternational.com/index.php/products/guidewire-inserted-chest-tube.html

A video of placement is available at this site.

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HOW CAN I GET PROFICIENT AT ULTRASOUND-GUIDED FINE NEEDLE ASPIRATION?

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TIPS AND TRICKS TO OPTIMIZE URINARY BLADDER ULTRASOUND

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Introduction

The urinary bladder is often thought to be one of the easier organs to evaluate with ultrasound. However, there are many pitfalls for bladder ultrasound interpretation because of artifacts which are more common and more apparent in the urinary bladder, and because of mobility of intraluminal structures as well as the potentially intrapelvic location of the caudal aspect of the urinary bladder and urethra. Knowledge of ultrasound artifacts, how they can be avoided or interpreted, and good scanning technique are very important when interpreting ultrasound examinations of the urinary bladder.

Scanning technique

As with any other ultrasound examination it is important to prepare the skin properly by clipping the haircoat and applying alcohol and ultrasound gel. The urinary bladder, depending on body composition and degree of filling typically has a very superficial location along the abdominal wall. High frequency linear transducers are ideal to evaluate the wall thickness and wall layering in most animals. Curvilinear, microcurved or sector probes may be necessary to evaluate the urethra and prostate due to deeper location and better access to the pelvic inlet with a smaller footprint probe. It is important to adjust the focal zone during evaluation of the urinary bladder. For the superficially located ventral or ventrolateral (depending on patient position) bladder wall the focal zone should be moved into a superficial position whereas it should be moved further down when evaluating the far bladder wall. Since the urine in the bladder lumen is poorly sound attenuating there is fair amount of distal acoustic enhancement which can be corrected by adjusting the time gain compensation to a curvilinear shape, decreasing the amount of intensification of the deeper echoes. The bladder can be examined with the patient in lateral or dorsal position, keeping in mind that bladder position relative to midline can vary with degree of filling and presence of gastrointestinal contents. The bladder should be examined in long axis and in transverse image planes, and care should be taken to follow the urethra as far as possible into the pelvic canal to determine if there are mural or intraluminal lesions. For a complete evaluation of the urinary bladder the regional lymph nodes, ureters and kidneys should be examined as well. The ureterovesicular junctions are located in the caudodorsal bladder wall at the level of the trigone and are variable in their visibility and appearance. If visible, they present as focal tissue thickening or protrusion into the urinary bladder lumen. The ureters are typically poorly visible unless dilated or thickened. Examination of the kidneys is important to determine presence of obstructive lesions, infection, nodules or masses and other lesions that could result from or manifest in urinary bladder disease. The sublumbar lymph center including the medial and internal iliac lymph nodes is the closest draining lymph center and is closely associated with the bifurcation of the aorta and caudal vena cava.

Common artifacts

As mentioned above, distal acoustic enhancement is a common artifact associated with the urinary bladder and can easily be corrected by adjusting time gain compensation. Also common is the occurrence of "pseudosludge". This is characterized by increased echogenicity in the far field over the urinary bladder with a curvilinear surface and could be confused with real urinary sediment. The underlying cause is a combination of slice thickness artifact where a portion of the curved echogenic urinary bladder wall is averaged with the hypoechoic bladder lumen resulting in an intermediate echogenicity along the bladder wall. The second component is called side lobe artifact. This artifact is caused by sound beams originating from the transducer that travel in a slightly different direction from the main beam. If reflected back to the transducer by a highly reflective surface such as the descending colon, some of these sound waves may return to the transducer and result in creation of an image along the main axis of the beam again mimicking urinary bladder sediment. This artifact is not unique to the urinary bladder but can happen anywhere in the abdomen, the difference is that these stray or side lobe echoes are visible in the otherwise anechoic urinary bladder whereas they blend in with the abdominal contents elsewhere. Near field artifacts are unavoidable and are encountered throughout the abdomen as well. They are particularly disrupting in the urinary bladder though since the thin bladder wall is in a very superficial position and is difficult to evaluate when obscured by these artifacts. Linear transducers help with improving the near field, and as mentioned above adjustment of the focal zone, use of harmonic imaging or image compounding can help as well. A very useful artifact in the urinary bladder

is distal acoustic shadowing caused by mineralized structures such as urinary calculi. This artifact is caused by complete absorption of sound waves by the mineralized structure with creation of a signal void or shadow distal to the surface of the calculus. If shadowing is present then the conclusion can be drawn that there is mineralized material in the urinary bladder. Another artifact that can be used to determine presence of mineralized calculi is the twinkle artifact. When color Doppler is placed on a mineralized structure with a highly irregular surface it will create Doppler signal without any actual flow present.

A few helpful tips and tricks

Urinary bladder ultrasound is a very dynamic examination due to the mobile nature of the bladder contents. Repositioning the patient is therefore very helpful and commonly used. Turning the patient from one side to the other or from lateral to dorsal recumbency or vice versa has several effects. It moves different portions of the bladder wall to the surface and areas that were previously obscured by near field artifacts or artifacts originating from the colon can now be assessed. It can also be used to determine if any observed lesions are intraluminal and mobile or if they are sessile and associated with the bladder wall. Sediment can be moved and stirred up that way to differentiate from artifacts as described above. Can't see the trigone because the bladder is in a too caudal location? Try having someone do a rectal examination and have them try to push intrapelvic structures such as the prostate cranially. Not sure if a shadowing structure is a calculus or an aggregation of mineralized sludge? This question is sometimes difficult to answer. Repositioning the patient or rolling them from side to side a few times often stirs up sediment and results in altered shape or dissolution of an aggregate of sludge whereas a calculus may change its location but not its size and shape. Palpation of the urinary bladder to move intraluminal contents is another good option. Doppler ultrasound is useful not only to elicit the above-mentioned twinkle artifact but is essential to determine if a mural mass is perfused or not (tumor vs. hematoma). It is also often used to improve visibility of ureteral jets. Presence of a visible urine jet through both ureterovesicular junctions into the urinary bladder is proof of patency (for example in presence of trigonal urinary bladder masses) and location of the ureteral junction in order to rule out ectopic ureters in dogs with urinary incontinence. To increase the frequency and visibility of ureteral jets a diuretic can be administered intravenously or subcutaneously which will result in increased urine production within a few minutes.

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SVA PRACTICE MANAGEMENT

RECRUITING THE RIGHT STAFF, AND KEEPING THEM

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RECRUITING THE RIGHT STAFF AND KEEPING THEM

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People are the key to the success of any organization which is why recruiting and retaining the right staff for your clinic is critical to its future development.

Preparation

Before commencing the recruitment process there are a number of key processes that need to be complete first

Your Clinic's Vision and Values

You must be able to describe to any prospective member of the practice staff an inspiring vision for the future of your practice that will encourages them to join you in the journey, and to describe the values that you will uphold whilst you are on the journey.

The vision and values are key to attracting applicants and helping them to understand the type of clinic they will be joining, whilst also providing you with a benchmark to determine whether a candidate will be a good "fit" for your clinic. Failing to live up to a clinic's values is the most common reason why well qualified candidates do not work out as anticipated.

The Job Descriptions

Recruiting a new member for the team is a good opportunity to review the job description of the role, even if they are replacing an existing member of staff. Developing job descriptions helps you to reflect on the key tasks the individual is to perform, and will allow both you and the candidate to judge whether they are suitable for the role.

The Person Specification

This should define the type of person for whom this role would be suited. This should include the obvious technical qualifications that might be required, however it is really about the personal attributes that are required. These should reflect the clinic's values in terms of personal character (empathetic, conscientious, self awareness, willingness to help others, etc.)

The Job advert

The level of detail you may wish to include in the job advert is usually dictated by the cost of the medium used. It should include the Job role, and then the key points from your vision, values, job description and person specification as space allows. Providing a contact point to allow access to further detail is always helpful, and this is simple where the supporting documents are already available

Invite candidates to provide a covering letter, setting out why they should be the next member of your team. Putting the key points in the advert allows them to direct their answer accordingly

Reviewing Applications

Remember, the best candidate may not have the best CV! Often long serving staff are prompted to explore job vacancies because of some recent event. As such they will often not have had the time to prepare a "polished" CV, so a little bit of digging can often unearth a little gem.

Create a list of the key requirements (technical and more importantly personal) and judge each CV against these in as objective a way as possible. Don't make the list too long, it is better to focus on 6 to 10 key areas and look for evidence in each case. Assign a weighting score to each questions so that you can "grade" experience (say 3 for lots, 1 for some) Ensure that your key requirements don't breach local laws regarding discrimination.

The interview process

The interview is an opportunity for you to explore the candidates background and experience, and for the candidate to find out more about your clinic. As such the way in which you prepare for and conduct the interview process will be a key factor in both attracting good staff and identifying future problems. Before each interview review the score sheet from the CV review, and identify any areas of concern that you specifically wish to explore. Use the same list to score each candidate after the interview. It is a good idea to have identified a question or scenario that you can use to allow candidates to demonstrate their suitability against each point. Asking each candidate the same questions will give you a much better impression of their relative strengths and weaknesses. This is important, because often we find that we don't have an "ideal" candidate.

Effective Questioning

Use as few questions as possible (prepare them beforehand

Use open ended questions

Tell the listener why the questions are important

Ask every question as if it were being asked for the first time

Ask questions at the relevant time

Effective Listening

Care about the other person's point of view

Maintain concentration

Take time to weigh up what is being said

Use "acknowledgements" to give you "thinking time"

Develop questions to fill in the "gaps" in your understanding

Induction and probation

Prepare and induction program before the successful candidate arrives. Try to give them time to get to know your clinic and the key process that they will use before we "chuck them in at the deep end". It is worth investing a short period of time at the start to avoid problems later on.

Set up periodic reviews to touch base and to allow you to deal with any emerging problems quickly.

Set up more formal reviews at 3 and 6 months to review progress and to record successes and concerns.

These reviews should be a meeting on equal terms, and which use a positive reflection of the past and open ended questions to draw on the employee's own evaluation of past performance and to direct their skills towards new accomplishments. The outcome should be an agreed list of SMART objectives.

43RD WORLD SMALL ANIMAL VETERINARY ASSOCIATION CONGRESS AND 9TH FASAVA CONGRESS

WSV18-0229

ORTHOPEDIC SURGERY

THE ENLARGED JOINT: HOW TO USE JOINT TAPS AS A SIMPLE DIAGNOSTIC TOOL IN SOLVING JOINT PROBLEMS

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Learning Objectives

At the end of this session you will be able to:

- Recognise non-inflammatory joint fluid from its gross characteristics
- Use simple cytological interpretation of synovial fluid to differentiate inflammatory and non-inflammatory fluid

The Role of Joint Fluid Analysis in Diagnosis of Joint Diseases

Synovial fluid is a dialysate of plasma that contains glycoproteins from the synovial membrane Type B cells. Polysulphated glycosaminoglycans (PGAGs) contribute to the viscosity of the synovial fluid. Disease processes of the synovial membrane or articular cartilage can alter the characteristics of synovial fluid.

Osteoarthritis (OA) is by far the most common type of joint disease in dogs. Inflammatory joint disease, infection and immune-mediated diseases, are an infrequent but significant cause of joint pain and lameness.

Synovial fluid analysis can been used to differentiate between:

- inflammatory and non-inflammatory arthropathies
- infectious and immune-mediated inflammatory arthropathies
- traumatic arthropathies
- acute and chronic inflammation

Since the treatment of OA is very different from that used for inflammatory joint disease an accurate diagnosis is important. The single most useful test in distinguishing between OA and inflammatory joint disease is synovial fluid analysis.

Arthrocentesis for synovial fluid analysis is a relatively simple procedure to perform though it remains a surprisingly under-utilised tool in veterinary medicine.

What is needed for Arthrocentesis?

- Needles 20 – 22 gauge. Length depends on the distance of the joint cavity from the skin surface and the size of the animal. For the majority of joints in most small – large dogs 40mm / 1.5" length is ideal. 25mm is sufficient for the carpus and hock joints while for the shoulder and hip joint in giant breeds >40mm / 1.5" may be necessary.
- Syringes   2-5 ml syringes
- Microscope slides
- EDTA tubes
- (Heparin tubes +/- - only necessary if a mucin clot test is to be performed)
- sterile gloves
- Bottle of blood culture medium if synovial fluid is to be cultured – this requires ideally 5ml of synovial fluid. This is incubated for 24 hours before plating to blood agar plates. Culture of a small amount of fluid is unreliable.

Collection Technique

General anesthesia or sedation is recommended. The sites for arthrocentesis should be clipped and surgically prepared. Draping is not necessary however the use of sterile surgical gloves is recommended.

Successful arthrocentesis is easier if the operator has a good "mind's eye" picture of the local joint anatomy when making the needle puncture.

The needle puncture should be made with the needle attached to the syringe. Apply gentle negative pressure to the syringe. After collection, release the negative pressure, **disconnect the syringe from the needle** to prevent sucking blood into the syringe when you withdraw the needle through the joint capsule, and then prepare the fluid as described below.

If blood appears in the needle hub disconnect the syringe before it contaminates the fluid in the syringe if possible as contamination with peripheral blood will alter the synovial fluid analysis. If this is not possible and blood contamination has occurred make a note of this so that it may be taken into account when interpreting the results.

Iatrogenic contamination with blood may be differentiated from hemorrhagic synovial fluid. Hemorrhagic fluid is usually uniformly discolored whereas iatrogenic contamination can be seen as discrete blood swirling in the fluid.

Shoulder joint

A lateral or a cranial approach is possible. The lateral approach is made by inserting the needle just distal to the acromion process of the scapula and immediately caudal to the greater tubercle.

The cranial approach is made by inserting the needle medial to the greater tubercle of the humerus and directing it caudally, ventral to the supraglenoid tubercle of the scapula

Elbow

Many options exist for arthrocentesis of the elbow joint. The easiest approach is the caudolateral approach. The elbow is flexed to a normal standing angle.

The needle is inserted just medial to the lateral epicondylar ridge and directed cranially, medially and

slightly distally into the olecranon fossa (towards the tip of the anconeal process). This is the largest joint space in the elbow.

Carpus

This is probably the easiest joint to sample. There are three joint levels in the carpus;

the antebrachiocarpal (ABC) joint, the middle carpal joint and the carpometacarpal joint. The ABC joint is the most proximal of the carpal joints located between the radius and the radial and ulna carpal bones. The middle carpal joint lies between the radial and ulna carpal bones and the numbered carpal bones. The carpometacarpal joint is the most distal of the three joint levels.

The middle carpal joint and the carpometacarpal joint communicate with each other while the ABC joint is separate. Despite this, sampling is routinely made from the ABC joint as it is the largest of the carpal joint spaces and it is rare that joint disease should be localised specifically to the distal two joint levels.

The carpus is flexed and the needle is inserted into the craniomedial part of the ABC joint. A depression can be visualised and palpated at the joint space. The accessory cephalic vein is also easily visualised in this location and avoided.

Coxofemoral joint

The coxofemoral joint is more difficult to sample and is not routinely sampled unless specific indications exist. A lateral or a ventral approach is possible.

For the lateral approach the femur is abducted and slightly externally rotated (supinated). The needle is inserted craniodorsal to the greater trochanter and directed medially and slightly ventrally to penetrate the joint space.

For the ventral approach the animal should be positioned in dorsal recumbency with the stifle joint fully abducted and the femur perpendicular to the long axis of the spine. The insertion of the pectineus muscle on the ileopectineal eminence of the pubis is palpated. The needle is inserted caudolateral to this eminence and directed cranially and dorsally into the joint.

Stifle joint

When a palpable joint effusion exists the easiest collection site in the stifle is the femoropatella joint pouch, which is the largest of the joint pouches in the stifle joint. It should be noted that this is a potential space so it is only suitable for collection **when an effusion is present**.

The needle is inserted immediately lateral to the patella and trochlea ridge at the level of the distal pole of the patella elevated about 30^0 fro the skin surface. The needle is directed dorsally towards the non-articular part of the lateral femoral condyle.

In chronic disease with thickened joint capsule you will feel a decrease in resistance when the needle tip passes through the capsule into the joint. If the condyle is contacted by the needle (this area is not covered in articular cartilage so no damage is done), the needle is withdrawn slightly and the collection made.

The femorotibial joint space is the other option for collection from the stifle joint. It is a permanent space and so is easy to penetrate even when no effusion is present however collection of fluid can be complicated by interference by the fat pad.

The needle is inserted immediately lateral to the straight patellar ligament midway between the patella and the tibial tuberosity. The needle is directed medially and caudally through the fat pad into the intercondylar space.

Hock joint

The hock joint can be penetrated medially or laterally and either cranial or caudal to the malleoli.

What to do with the synovial fluid on collection?

What you do with the synovial fluid will depend on the volume collected.

If small volumes (<0.2ml) are obtained these should be assessed in the syringe for color, clarity and viscosity prior to preparation of a direct smear. In these cases there is not enough fluid to submit a sample in EDTA for total white blood cell count so the sample is assessed visually for color and clarity, a smear is made, and the viscosity is subjectively assessed.

Smears should be made as soon as possible after collection to reduce the artefactual vacuolation and nuclear degeneration of large mononuclear cells. Smears are simply made by placing a drop on a slide and performing a "squash" preparation where a spreader slide is laid flat on the sample slide and the two slides are drawn slowly apart. Thin smears are easier to interpret and can be made by slowly pulling the two slides apart.

If larger volumes are collected the fluid should be put into anticoagulant tubes. Unless a mucin clot test for viscosity is to be performed place a drop on a slide as described above for a smear and put 1ml into an EDTA tube.

Normal synovial fluid will not clot, as it does not contain fibrinogen. However if there has been blood contamination during collection or intra-articular haemorrhage or protein exudation has occurred, clotting may occur.

EDTA is the preferred anticoagulant for cytological examination while heparin is preferred for the mucin clot test and viscosity measurement. EDTA will cause

degradation of hyaluronic acid and thereby affect viscosity. Both EDTA and heparin are suitable for assessment of color and clarity and measurement of protein.

If a septic arthropathy is suspected synovial fluid should be submitted for aerobic culture in a blood culture bottle or if anaerobic culture is required then anaerobic transport media is needed.
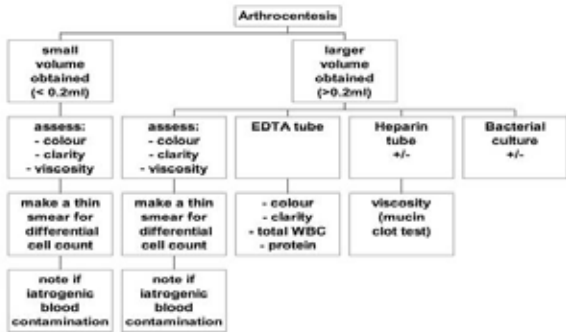
Synovial fluid analysis

Synovial fluid is assessed grossly for volume, color, clarity and viscosity, and a total and differential white cell count and protein content performed. Mucin clot test is usually not indicated.

Normal values are listed below:

Volume (ml)	Colour	Clarity	Viscosity	Mucin Clot	Cell Count (x 10⁹/L)	Mononuclear Cells (%)	Neutrophil (%)
0.01-1.0	None to light yellow	Transparent	Good	Good	< 3.0	90 - 100	0 - 10

Volume

The volume of synovial fluid collected from a normal joint varies and ranges from 0. 1ml to 0.5ml. Joint effusion is usually apparent clinically +/- radiographically depending on the joint.

Color

Normal synovial fluid should be colorless. A change in color suggests hemorrhage or inflammation. A yellow-tinge indicates that hemorrhage into the synovial membrane has occurred and hemoglobin breakdown products are being released into the fluid. Iatrogenic contamination with blood is apparent from incomplete mixing of blood as evidenced in a clear synovial fluid sample that becomes streaked and blood-tinged during collection. Should iatrogenic contamination occur this should be taken in to consideration when assessing total and differential white cell counts and protein levels as blood will falsely elevate all of these.

Clarity

The clarity of synovial fluid is affected by the degree of cellularity. Normal synovial fluid is usually completely transparent. Synovial fluid may be graded as transparent, transluscent or opaque. Transparent synovial fluid allows the print on the syringe to be read through it. The print appears as an area of darkness if the fluid is transluscent and is indiscernible if the fluid is opaque.

Inflammatory fluid has a high white cell count and the fluid is usually opaque.

Viscosity

Viscosity may be assessed both subjectively and objectively. Viscosity is a function of the concentration and quality of hyaluronic acid and becomes poorer as the degree of joint inflammation increases due to depolymerisation of the hyaluronic acid by bacterial or inflammatory proteases.

Subjective assessment of viscosity is performed at the time of fluid collection by observing the length of the synovial fluid strand created by dropping some fluid from the collecting syringe to a slide or alternatively by placing a drop of fluid between your thumb and forefinger. The viscosity is normal if a strand of greater than 2.5 cm

is achieved before breaking (although much longer strands are typical). Fluid of normal viscosity will not run off a microscope slide when held vertical.

Normal synovial fluid has viscosity like oil. A "thin" fluid of poor viscosity is common in inflamed joints, however it may also be seen in non-inflammatory arthropathies.

Objective assessment of viscosity by the mucin clot test may be performed on synovial fluid collected into either plain or heparinised tubes although is not routinely performed. EDTA is unsuitable for the mucin clot test as it tends to degrade hyaluronic acid.

Total Cell Count

Total and differential cell counts of synovial fluid are important parameters of arthropathies. Reported total cell counts from normal canine joints can vary but are generally **less than 3.0 x 10⁹/L**. Being a dialysate of plasma there are only small numbers of nucleated cells and an absence of red blood cells.

If there is only sufficient fluid to prepare a smear then a total cell count estimate should be. The body of a smear should contain one to three nucleated cells per 400x magnification field. Cell count estimates are graded as normal, or slightly, moderately or grossly increased.

Total white cell counts in non inflammatory arthropathies are typically normal to slightly elevated – usually < 5.0 x 10⁹/L.

Total white cell counts in inflammatory arthropathies are typically moderately to markedly increased - > 10.0 x 10⁹/L.

Differential Cell Count

Normal synovial fluid contains > 90% large mononuclear cells and lymphocytes (small mononuclear cells). Lymphocyte numbers reported in normal synovial fluid range from 11% to 44% with the large mononuclear cells representing the majority of normal cells.

Normal joint fluid contains very low numbers of neutrophils. Typically neutrophils are <10% of the total white cell count.

Elevation of the relative proportion or absolute number of neutrophils in synovial fluid indicates either inflammation of the synovial membrane or contamination with peripheral blood. Elevation of the neutrophil percentage >10% regardless of the total white cell count is significant.

It is also important to assess the morphology of the neutrophils. It is widely reported that neutrophils from a septic inflammatory arthropathy will usually show evidence of toxic change or degeneration whereas those from an immune-mediated arthropathy will appear more normal. This however has been shown to be unreliable. Joint infection is commonly seen without degenerate neutrophils being present. The presence of intracellular bacteria is indicative of infection.

A case series of dogs with septic arthritis (Marchevsky and Read 1998), would suggest that neutrophils in septic arthropathies are more typically non-degenerate. In a series of 19 dogs with septic arthritis only one dog (5%) had degenerate neutrophils present in a synovial fluid smear. Only 7 of 13 dogs had bacteria visible on cytology.

Protein

As synovial fluid is a dialysate of plasma the protein level in normal synovial fluid is usually low. Normal synovial protein levels in the dog are 20 to 25g/L. Protein levels can be measured by either a refractometer or biochemical assay. Synovial fluid protein levels are a function of the local vascular permeability, the molecular size of the protein and its plasma concentration.

Synovial fluid protein levels will increase proportional to the degree of inflammation and may approach serum protein levels. Synovial fluid samples with very high protein levels may clot. (This can also happen in the presence of peripheral blood contamination.)
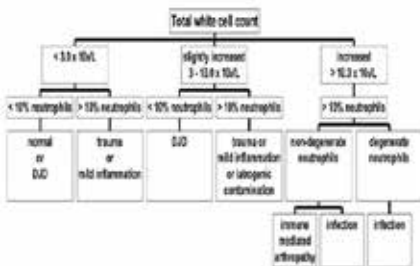
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NAVC SHORT TOPICS FROM EXPERTS

HOW I DIAGNOSE CANINE COLLAPSING AIRWAY DISEASES

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HOW I DIAGNOSE CANINE COLLAPSING AIRWAY DISEASE

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Introduction

Upper and lower airway disease is common in dogs and cats, which can present with similar signs regardless of the location. Following stabilization of the patient with oxygen, radiography plays a very valuable role in determining the cause of airway distress. In addition to radiography, computed tomography (CT) is a cross-sectional modality for examining pathology when radiography is equivocal. This lecture will provide the participant an overview of how to differentiate upper from lower airway disease in the coughing dog and discuss how to arrive at a practical diagnosis with many case examples.**Techniques in Radiography**

If the animal is stable enough with its respiratory rate and effort, I advocate a 4-view thoracic radiograph (R and L lateral, VD and DV). A dorsoventral radiograph instead of a ventrodorsal radiograph is often beneficial in dyspneic patients as they are often more comfortable resting on their sternum. If diseases of the upper airways are suspected, a lateral projection of the larynx/pharynx area and cervical trachea is mandatory. A foreign body or mass in this region can be immediately recognized. Thoracic studies should always be obtained during inspiration. An additional exception to the rule is the comparison of diameters of the trachea and main stem bronchi in patients with tracheal collapse, which requires both respiratory phases and even fluoroscopy.**Upper Airway Diseases**

Larynx und Trachea

Lateral views of the laryngeal and cervical region are usually sufficient. Typical changes in dogs are increasing mineralization of the laryngeal and tracheal cartilages with age. Mineralization can already be present in dogs as young as 2 years of age, especially in chondrodystrophic breeds. The most commonly diagnosed diseases of the larynx are larynx paralysis, eventration of the ventricle and larynx collapse. These can usually be diagnosed by oral inspection. Radiographic findings are often

unremarkable; however, decreased diameter of the air-filled oro- and nasopharynx may be detected. In brachycephalic syndrome, elongation of the soft palate, tracheal hypoplasia and increased soft tissues of the larynx can be identified radiographically. The normal ratio of tracheal diameter to thoracic inlet diameter is .07-.21 in bulldogs and averages 0.16 in non-bulldog brachycephalic breeds. Non-brachycephalic breeds have a ratio of 0.2. Note: the trachea will appear narrower prior to one year of age in many breeds, especially brachycephalic ones.

In cats the pharyngeal area should be scrutinized for soft tissue masses associated with nasopharyngeal polyps.

The rigidity of the trachea decreases with age in small breed dogs and has an affect on the diameter during inspiration and expiration, which can be shown radiographically. This makes the diagnosis of collapsing trachea in small breed dogs sometimes difficult. The difference between normal and pathologic is not always clear. Therefore, dynamic views of the trachea can be very helpful for a more definitive diagnosis. This is best performed with fluoroscopy so that the change in diameter of the cervical and thoracic trachea can be observed during deep inspiration and expiration as well as when the dog coughs. Unfortunately, this is only available at referral centers. Static radiographs, however, are a minimum data base and should be performed if the survey radiographs are negative. Both in inspiration and expiration have to be compared to detect many types of tracheal collapse. Typicallly, the extrathoracic trachea will collapse during inspiration, the intrathoracic trachea and stem bronchi during expiration. Collapse may also occur focally at the thoracic inlet. The significance of a soft tissue opaque shadowing of the dorsal tracheal border is subject to discussions. One possible explanation for the appearance is a redundant tracheal membrane, meaning the protrusion into the tracheal lumen.

Small breed dogs with large livers and a cranially displaced diaphragm can have direct compression of the stem bronchi which can have a similar affect as an enlarged left atrium in dogs with left heart failure. Reduction in the diameter of the trachea may also occur due to compression by mediastinal masses, enlarged tracheobronchial lymph nodes or an enlarged left atrium. Collapse of the stem bronchi is commonly the cause of coughing in small breed dogs with mitral insufficiency due to endocardiosis and enlargement of the left atrium. Tracheal collapse and compression should be differentiated from stenosis secondary to space-occupying lesions. Granulomas (foreign body, parasites) are rare. Neoplasia is also very uncommon. Tumors such as osteochondroma, osteosarcoma and chondrosarcoma appear as soft-tissue opaque lesions that contrast with the air-filled tracheal lumen. Inhaled foreign bodies such as small pebbles are not uncommon, especially in cats.

Foreign bodies are usually readily visible due to contrast with the air-filled lumen. Stenosis can occur due to prior foreign body or aspiration of gastric juices. They present radiographically as a focal narrowing of the tracheal lumen and can be present anywhere along the length of the trachea.

Tip: for squirming small breed dogs where you are trying to get inspiratory and expiratory views in lateral recumbency and respect radiation safety at the same time, make a doggie burrito: swaddle the tiny dog in a towel with its legs pulled forward and it won't struggle on its side.**Lower Airway Diseases**

Peribronchial infiltrates and edema, narrowing of the bronchial lumen due to either thickening of the bronchial wall or build up of secretions as well as enlargement of the bronchi are common consequences of dogs and cats with lower airway disease.

Radiographic findings of lower airway disease are rings with a relatively small air-filled lumen represent transverse sections of the affected bronchi along with an increased number of linear structures throughout the lung. The lung will appear to have a diffusely increased opacity due to the presence of thickened bronchi, bronchial secretions or peribronchial infiltrates. The difficulty lies in differentiating disease from age related changes of the bronchial tree, which can appear similar. Mineralized bronchial walls due to age appear thin and finely mineral opaque and sharply delineated. Thickening of the bronchial walls leading to "doughnuts" and "tramlines" is a sign of chronic bronchial inflammation. Primary differential diagnoses are chronic bronchitis, eosinophilic infiltrates or parasitic infections. Thickening of the peribronchial tissues (bronchial cuffing) due to edema or inflammation can mimic bronchial wall thickening but belong to another list of differentials (bronchopneumonia, cardiogenic edema in large breed dogs, or allergic reactions).

Chronic bronchitis is an exclusion diagnosis. Thickened bronchial walls and their increased visibility is a reliable sign of chronic bronchitis in dogs. In severe cases the bronchi can be completely opacified by mucus and can be confounded with vessels or even small nodules. In cases of acute bronchitis the thoracic study may be inconspicuous or resemble chronic cases.

Bronchiectasis is much less common and appears as widened, irregularly shaped bronchial branches with a thickened wall. This represents end-stage bronchial disease, usually following chronic bronchitis. Soft tissue opacities may also be present in the periphery of the lung due to secretion build up and appears as a peripheral alveolar pattern, either in one or multiple lobes. Bronchiectasis may only be evident in one or two lung lobes or can be generalized.

Acute bronchitis may appear unremarkable

radiographically. Bronchitis is an interstitial disease, so the radiographic findings are often mixed. It can result in air trapping and emphysema, which will result in an increased air volume in the lung which appears hyperlucent. Atelectasis of the right middle lung lobe may occur in cats and appears as a consolidation and decreased volume of that lung lobe (best seen in VD view).

A relatively common cause of cough is inhaled foreign bodies such as grass awns, wooden pieces and the like. The radiographic appearance depends on the degree of bronchial obstruction and if the foreign body has irritating properties.

The most common form of pneumonia is unspecific bacterial infection und often follows primary lung disease such as hemorrhage, viral infection or chronic bronchitis. Patients suffering immunodeficiency or having reduced ciliary apparatus motility are prone to pneumonia. In cases of pneumonia, three different projections are recommended to visualize all parts of the lung field. The most common radiographic sign is an alveolar pattern affecting an entire lobe or just its tips ventrally. The most often affected areas are the cranioventral parts of the lung and the right middle lobe. An asymmetric distribution is also possible. Aspiration pneumonia most often affects the cranioventral regions, whereas inhaled, high speed foreign bodies tend to lodge in the caudodorsal region.

Neoplasia of the bronchial walls is relatively rare. The most common tumor type is bronchial wall carcinoma. Also in neoplasia the radiographic appearance may be manifold (solitary nodules, military to alveolar consolidation). The accumulation with cells and fluids in the interstitium may lead to an increased opacity without complete obliteration of the air containing spaces. Therefore the vessels, caudal vena cava and the cardiac silhouette may still be visible but ill defined.

Canine Tracheobronchomalacia

Tracheal chondromalacia and collapse are common in middle-aged and older small and toy breed dogs. Collapse can be static or dynamic and can occur anywhere from mid-trachea to the stem bronchi and can even involve the entire trachea. Extrathoracic tracheal collapse is most pronounced during inspiration while intrathoracic collapse is most pronounced during expiration. Therefore, both inspiratory and expiratory radiographic images should be performed that include the entire length of the trachea. Ideally, video fluoroscopy should be performed if screening inspiratory and expiratory static radiographs fail to detect collapse.

Static tracheal collapse

This is identified radiographically as a static dorsoventral narrowing of the tracheal lumen. An undulating appearance of the dorsal wall or non-uniform diameter of the tracheal lumen is also suggestive of chondromalacia and indicates the need for dynamic examination.

Inspiratory and Expiratory Radiography

Dynamic radiography to diagnose tracheal collapse takes patience and effort. Small dogs with breathing difficulty are stressed and quiet surroundings and attention to their comfort is necessary for a successful outcome. Radiographs can be exposed during induced coughing after applying pressure to the trachea. With the dog in lateral recumbency, the trachea is palpated until coughing is induced. An attempt should be made to make exposures as the dog breaths deeply in and then again on deep expiration. However, this can be difficult to do and often times small dogs are not cooperative or stressed during the procedure. A good alternative is to calmly cover the nose and mouth while talking to it and this will gradually lead to deeper and deeper inspiration and consequently expiration so that exposures can be made at these moments.

Confirming a Diagnosis of Collapse

Less than 25% decreased luminal diameter is generally considered insignificant in chondrodrystrophic breed dogs and ususaly doesn't correlate to collapse fluoroscopically. Furthermore, intrathoracic collapse can be detected fluoroscopically when dynamically performed radiographs are negative. Decreased luminal diameters should be further classified as 50%, 75% or 90-100% during inspiration and expiration and whether or not the collapse is cervical, intrathoracic, static or dynamic. However, in another study, bronchomalacia and sublobar airway collapse in the absence of cervical tracheal collapse were common in medium and large breed dogs examined, providing further evidence for the utility of bronchoscopy in the diagnostic evaluation of dogs with cough. This underscores the necessity for including bronchoscopy in coughing dogs, even if the tracheal collapse is not evident radiographically.

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SVA FELINE

CURRENT APPROACH AND MANAGEMENT OF DISEASES OF THE NASAL CAVITY IN CATS

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Introduction

Presentation for chronic nasal discharge and sneezing is common in cats of all ages. Chronic rhinosinusitis (CRS) is the most common cause of this presentation in clinical practice. Diagnosis requires **exclusion** of other causes of nasal discharge and sneezing (Table 1):

Table 1: Causes of nasal cavity disease in cats

Inflammatory and Infectious Causes
- Chronic rhinosinusitis
- Nasopharyngeal or nasal polyps
- Nasopharyngeal stenosis
- Dental disease – secondary bacterial tooth root infections
- Foreign bodies
- Fungal rhinitis – cryptococcosis, aspergillosis

Neoplastic Causes
- Lymphoma
- Adenocarcinoma
- Squamous cell carcinoma
- Undifferentiated carcinoma
- Fibrosarcoma
- Osteosarcoma

Chronic rhinosinusitis (CRS)

Cats with CRS typically present with a history of > 4 weeks mucopurulent nasal discharge and sneezing. Feline herpes virus-1 (FHV) is thought to have an initiating role, resulting in severe mucosal damage and turbinate lysis, compounded by an exuberant immune-response, possible cycles of FHV reactivation and recurrent secondary bacterial infection.

Fungal rhinosinusitis

Aspergillosis occurs in two forms; sino-nasal aspergillosis (SNA) and sino-orbital aspergillosis (SOA). Both infections arise in the nasal cavity after inhalation of fungal spores (conidia). In SOA, which is more common (65% cases), infection spreads to involve the orbit and paranasal tissues. Brachycephalic breeds, especially Persians and Himalayans are predisposed to SNA and SOA (40% of cases). SNA is most commonly caused by *A. fumigatus* and *A. niger*, while SOA is most commonly caused by *A. felis* and by *A. udagawae*.

Because the fungi that cause aspergillosis are ubiquitous, and since *C. neoformans* has a worldwide distribution and *C. gattii* is endemic in many regions of the world cryptococcosis and aspergillosis should be considered in any cat with chronic URT signs.

Diagnosis

Findings at presentation can help rank differential diagnoses, but clinical signs overlap with infectious, inflammatory and neoplastic causes. Exophthalmos is a common sign in cats with fungal or neoplastic lesions invading from the nasal cavity into the orbit. A mass in the orbit causes loss of retropulsion of the globe, prolapse and inflammation of the nictitating membrane, conjunctival discharge and central corneal ulceration. Orbital masses often invade into the oral cavity, detected as a submucosal or ulcerated mass in the pterygopalatine fossa adjacent the most caudal molar tooth. Seizures and other CNS signs occur in neoplasia and fungal infection due to extension of disease.

Investigation

- Serology. Detection of cryptococcal antigen in the blood using a latex cryptococcal antigen agglutination or cryptococcal antigen lateral flow assay has high sensitivity and specificity for diagnosis of cryptococcosis. Detection of *Aspergillus*-specific IgG has high sensitivity and specificity for diagnosis of aspergillosis, but commercial assays are lacking. Assays to detect precipitins, such as agar immunodiffusion assays, are commercially available but have poor sensitivity.

- Oropharyngeal/conjunctival and nasal swabs – PCR testing or viral isolation for FHV-1 is usually unrewarding in cats with CRS since viral shedding has ceased; Bacterial culture of superficial nasal swabs is equally unrewarding since commensals will be cultured.

- Dental probing to identify deep periodontal pockets, oronasal fistulae or palatine defects is performed along with imaging, rhinoscopy and biopsy under general anaesthesia.

- Diagnostic imaging – CT is superior to radiography for evaluation of nasal cavity and paranasal sinuses. Images should be evaluated for evidence of tooth-root infection. Imaging features of CRS, neoplasia and fungal rhinitis overlap including severe turbinate lysis and soft-tissue attenuation within the nasal cavity and sinuses. Obstruction of the Eustachian tubes, common with neoplasia and inflammatory disease, results in opacification of the tympanic bullae.

- Diagnostic nasal lavage – is performed to collect diagnostic material for bacterial culture for cases of CRS, although results may represent normal flora. A 6 to 8 F sterile catheter is inserted into the nasal cavity, ending rostral to the medial canthus of the eye. The nasopharynx is occluded using digital dorsal pressure on the soft palate. Approx. 2 - 3 mL of sterile saline is flushed into the catheter and aspirated back. The collected sample can be used

for aerobic and anaerobic culture, fungal culture, or PCR (e.g. for *Mycoplasma* or *Bordetella*).

- Endoscopic examinations are performed next with the anaesthetised cat in sternal recumbency. Avoid mouth gags that maximally open the mouth as these occlude maxillary artery blood flow and can cause post-anaesthetic cortical blindness. Nasopharyngoscopy using a retroflexed bronchoscope or similar scope is performed to detect choanal mass lesions, stenotic lesions or foreign bodies. Mass lesions can be biopsied. Rigid antegrade rhinoscopy is performed next.

- Nasal lavage and nasal biopsy. Guided endoscopic or unguided nasal biopsies are collected for culture and histology. Therapeutic nasal lavage alleviates congestion in cats with CRS and is performed in the intubated cat (cuffed tube) with the pharynx packed with gauze. A 10 ml aliquot of sterile saline is flushed into the nasal cavity through one naris while the other naris is occluded with digital pressure. The procedure is repeated on the other side, and the gauze is then retrieved to inspect for foreign bodies, fungal plaques or tissue remnants. Nasal tumours, especially lymphomas, are friable and the nasal flush may yield tissue fragments for histopathology. Prior to extubating the cat, the pharynx should be suctioned carefully.

- Histology of nasal mucosal biopsies is essential to distinguish CRS from neoplasia and fungal infection. Inflammatory infiltrates in CRS can be neutrophilic, lymphoplasmacytic, eosinophilic or mixed and may be accompanied by epithelial erosions, turbinate lysis/remodelling, fibrosis, necrosis and glandular hyperplasia. Special stains to detect fungal hyphae such as PAS should be requested. If eosinophilic infiltrates are present and fungal stains are negative, immunohistochemistry for FHV may be performed.

Management of CRS

Treatment is palliative. Antibiotic therapy is guided by susceptibility results or given empirically for 6 – 8 weeks using clindamycin, doxycycline, amoxicillin-clavulanate or azithromycin. Longer-term or intermittent therapy may be required. Use of nasal decongestants should be limited to three days because rebound vasodilation can exacerbate signs. Famciclovir controls signs in some cats using 90 mg/kg q 12 h PO for one week beyond resolution of clinical signs. Management of nasal cryptococcosis Fluconazole is first-line therapy and is usually required for at least several months. Itraconazole is also effective but has more adverse effects (Table 1). In severe disease combination therapy with amphotericin B and flucytosine is recommended for four weeks followed by azole monotherapy. Treatment is continued until the cryptococcal antigen latex assay titre is zero, with follow-up antigen testing one month after therapy has been stopped. The prognosis for resolution of infection is good (>75% successful), although relapses occur in 17% of cases. Aspergillosis Endoscopic removal of all visible fungal plaques

combined with 1% topical clotrimazole infusion is recommended for SNA and prognosis is generally favourable. For SOA the prognosis is generally poor, although individual cases have been cured. Posaconazole monotherapy or combined with terbinafine is recommended for first-line therapy but treatment should be guided by the results of antifungal susceptibility testing (Table 2). Itraconazole should not be used empirically because resistance is common. Management of nasal neoplasia Both radiation and chemotherapy have benefits in treating nasal lymphoma. B-cell lymphomas, the most common type of nasal lymphoma, respond well to radiation therapy and have the best prognosis. In one study of 97 cats with nasal lymphoma treated with radiation therapy (RT) alone, chemotherapy alone or RT + chemotherapy. The median survival time for all therapies was 473 days and was not significantly different for the different treatment modalities.

TABLE 2 Dosages of Antifungals Used in the Treatment of Fungal Rhinitis

Drug	Dosage/Route of Administration	Diseases	Adverse Effects
Fluconazole	2.5-10 mg/kg q12h PO or 50 mg per cat q12-24h	C	Inappetence Hepatotoxicity (rare)
Itraconazole	Capsules: 5 mg/kg q12h or 10 mg/kg q24h PO. Administer with food Oral suspension: 3 mg/kg q24h PO	C, A	Anorexia, vomiting Hepatotoxicity: elevated liver enzymes, jaundice.
Posaconazole	15 mg/kg PO loading dose, then 7.5 mg/kg q 24h Administer with food	C, A	Hepatotoxicity: infrequent compared to itraconazole
Voriconazole	12.5 mg per cat q 72h PO	A	Hypersalivation Blindness, ataxia, stupor Consider use when other therapies have failed.
Terbinafine	30 mg/kg q24h PO	A	Anorexia, vomiting, diarrhea
Flucytosine	50 mg/kg q8h PO or 75 mg/kg q12h PO		Anorexia, vomiting, diarrhea
Amphotericin-B deoxycholate	0.5 mg/kg of 5 mg/ml stock solution in 300 ml per cat of 0.45% NaCl + 2.5% dextrose SC 2 -3 times weekly to a cumulative dose of 10-15 mg/kg	C, A	Nephrotoxicity: Monitor urea/creatinine. Discontinue for 2 to 3 wks if azotaemic.
Liposomal amphotericin	1-1.5 mg/kg IV q48h Give as a 1 mg/ml solution in 5% dextrose by IV infusion over 1-2 h	C, A	Nephrotoxicity: Monitor urea/creatinine every 2-3 days during administration.
Caspofungin	Loading dose 1 mg/kg IV, then 0.75 mg/kg q 24h IV	A	Uncommon

A, Aspergillosis; *ALP,* alkaline phosphatase; *ALT,* alanine aminotransferase; *C,* cryptococcosis; *PO,* orally.

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ANIMAL WELLNESS & WELFARE

THE ROLE OF VETERINARY NURSES IN OPTIMIZING ANIMAL WELFARE

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Many countries around the world currently have no veterinary nursing profession and challenges to animal welfare are common. This presentation will introduce the role of the professional veterinary nurse and discuss the role of veterinary nurses in supporting veterinary clinical excellence and improving animal welfare.

Professional veterinary nurses are instrumental in performing a multitude of tasks; freeing up time for the vets to focus on their areas of interest and improving patient welfare through supporting good standards of clinical practice and low-stress handling. The distinct role of veterinary nurses will be outlined and their role in supporting good animal welfare and veterinary clinical excellence will be discussed.

WSV18-0030

NUTRITION AND DIAGNOSTIC IMAGING (LECTURES GIVEN IN MANDARIN CHINESE)

TIPS FOR NUTRITION ASSESSMENT IN CATS

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TIPS FOR NUTRITION ASSESSMENT IN CATS

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Sacropenia in geriatric cats:

The age related loss of muscle mass and strength is a multi-factorial condition that occurs in old cats. In veterinary medicine, skeletal muscle atrophy is often observed in cats as they reach old age, but the process is not well understood. Weight loss and muscle wasting are very common and important for their health in senior cats. There are a lot of factors to be considered in assessing the nutritional needs of senior cats to ensure their health condition. Cats spend most of their time sleeping and grooming.

Therefore a 5-year-old cat may not move much more than a 5-year-old cat. Elderly cats also require more dietary energy because their fat and protein digestion is impaired. Approximately 30% of cats older than 12 years of ages have decreased fat absorption, and 20% have decreased protein digestibility. Protein should not be restricted in elderly cats that do not have underlying renal diseases. Therefore senior diets may not be appropriate for all geriatric patients, and the veterinarian should assess body condition and overall health status before making a dietary recommendation. A Basic Nutritional Assessment should be performed when a vet group is initially evaluating a cat. The first step when developing a nutritional plan for complete history, physical examination, and laboratory test should be performed to rule out diseases responsive to specific nutritional modifications. A key component of physical examination should be assessment of body condition.

The Global Nutrition Committee (GNC) developed global nutrition guidelines, which were first published in 2011. The goal of these guidelines is to help the veterinary healthcare team and pet owners ensure that dogs and cats are on an optimal nutrition plan tailored to the needs of the individual dog or cat.

In 2012, the GNC launched a Global Nutrition Toolkit, containing a suite of resources to assist practitioners and nutritionists to educate pet owners that each pet should receive an individual nutritional assessment and recommendation. They are also published and translated into many languages. In Asia region, Chinese and

Japanese versions are available.

Body Condition Score (BCS):

BCS scales are typically range from 1-9. This scale allows us to assess subtle changes in your pet's weight. Using this scale, cats are scored from 1 to 9 out of 9 with 5/9 being an ideal body weight.

Muscle Condition Score (MCS):

Veterinarians also use Muscle Condition Scoring to determine cat's health. The muscle condition score also helps estimate whether or not the pet is receiving enough protein. MCS is an useful toolkit to evaluate the muscle wasting in sick or old patients.

Resting Energy Requirements (RER):

Resting Energy Requirements or RER), which can be calculated by multiplying the cat's body weight

$$RER(kcal/day) = (Body\ weight\ _{kg})^{0.75} \times 70$$

Or

$$RER(kcal/day) = (Body\ weight\ _{kg} \times 30) + 70$$

Conclusion:

This presentation will address communication strategies for nutrition discussion, along with clinical case examples focusing on muscle wasting and weight management.

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WSV18-0056

NURSES (HILLS)

OXYGEN THERAPY

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OXYGEN THERAPY

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It is without question that adequate oxygen supply to all tissues and cells is essential for life. For oxygen to be delivered to the cells there must be adequate oxygen exchange in the lungs to the blood and therefore adequate pulmonary function is critical.

The Respiratory System

The respiratory system works in conjunction with the blood and cardiovascular system to provide the body with the required exchange of oxygen and carbon dioxide.

External respiration is the gaseous exchange of O_2 and CO_2 between the environment (air in the lungs) and the blood. Pulmonary ventilation and diffusion of gases through the alveolar membrane of the lungs as required in order for external respiration to take place.

Internal or tissue respiration is the exchange of O_2 and CO_2 between the blood in the capillaries and the tissue cells.

The respiratory system is referred to in two parts:
- **Upper** respiratory system; structures that are outside of the lungs – nose, pharynx, larynx, trachea
- **Lower** respiratory system; structures within the lungs – Bronchi, Bronchioles, Alveolar ducts and Alveoli

Bronchi and Bronchioles

The primary bronchi are the first branches of the trachea. They are quite large with C-shaped chondral rings like the trachea. They continue to branch, becoming smaller as they progress, resulting in secondary and tertiary bronchi. They are kept tubular by flattened, overlapping, curved cartilages. As branching continues into the smaller bronchioles, the cartilage component to their walls disappears when the diameter has reduced to 1mm or less. The respiratory bronchioles then give rise to the alveolar ducts and alveoli.

After the bronchi enters the lung, each main bronchus divides into even smaller bronchi and then finally into even smaller bronchioles. The bronchioles continue to subdivide down to the smallest air passages – the microscopic alveolar ducts. The alveolar ducts then end in groups of alveoli – a bit like a bunch of grapes – called alveolar sacs.

The bronchial tree is not a rigid tube; the diameter of each one can be adjusted by the smooth muscle fibres in its wall. The autonomic (unconscious) portion of the nervous system controls this smooth muscle.

Bronchodilation - This is when the smooth muscle relaxes (e.g. during times of intense physical activity) allowing the air passageways to dilate to their maximum capacity which helps the respiratory effort move the greatest amount of air back and forth into the alveoli with each breath.

Bronchoconstriction - During times of rest, the smooth muscle partially contracts reducing the size of the air passageways which in turn lessens the workload for the respiratory tract. However, some respiratory irritants and disease can cause severe bronchoconstriction making it difficult for the animal to breathe.

Alveolar ducts and Alveoli

External respiration takes place in the alveoli. This is when oxygen and carbon dioxide are exchanged between blood and the air in the alveoli. The rest of the respiratory structures just move air in and out of the alveoli.

The alveoli are tiny, thin walled sacs that are surrounded by a network of capillaries. The wall of each alveolus is made up from the thinnest epithelium in the body – simple squamous epithelium. The capillaries that surround the alveoli are also made up from simple squamous epithelium.

As these two layers are so thin, they freely allow oxygen and carbon dioxide to diffuse between the air and blood.

Each alveolus is lined with a thin layer of fluid that contains a substance called surfactant. This surfactant helps reduce the surface tension (the attraction of water molecules to each other) of the fluid. This prevents the alveoli from collapsing as air moves in and out during each breath.

Respiratory membrane

This is the alveolar and capillary wall through which gas exchange takes place. The alveoli make up the bulk of pulmonary tissue.

Dyspnoea can be defined as the sensation of difficulty in breathing, which is experienced by patients suffering compromised respiratory function.

The sensation is initiated by either hypoxaemia (low oxygen levels) or hypercapnoea (high carbon dioxide levels).

Signs of Dyspnoea / Respiratory Distress"
- · Open mouth breathing
- · Panting
- · Tachypnoea
- · Extension of head and neck

- · Abducted elbows
- · Exaggerated abdominal component

Airway Obstruction and Injuries to the Respiratory Tract

Causes of airway obstruction and respiratory dysfunction may include:

- · Trauma – Hit by car, dog attack
- · Foreign bodies
- · Degenerative physical conditions – such as: laryngeal paralysis, tumours, collapsing trachea
- · Haemothorax (blood in the chest cavity)
- · Pneumothorax (air in the chest cavity)
- · Chest wall trauma (flail chest)
- · Lung disorders (primary - e.g. chronic bronchitis, or secondary – e.g. due to cardiac failure)
- · Respiratory paralysis
- · Physical deformity e.g. brachycephalic – bulldogs / pugs etc

Pneumothorax

A pneumothorax is caused by leakage of air from the airways or parachyma into the pleural space. It is often caused by blunt trauma to the chest (e.g. hit by car).

Many patients that have sustained trauma to the chest may have a small leak that seals over quickly and may not produce clinical signs.

- **Open Pneumothorax** A pneumothorax is termed 'open' when there is a penetrating wound to the thoracic cavity and air is being 'sucked in' through the wound. First aid action is to cover the open wound immediately to reduce air being introduced.

- **Closed Pneumothorax** The most common type seen, especially following blunt trauma. There is no penetrating wound. The air is leaking within the pleural space.

- **Tension Pneumothorax** As the air leaks into the pleural space it is like a one-way valve, the air leaks in but is not removed. When the pressure from the leaking air becomes higher than atmospheric pressure, this is termed a tension pneumothorax – a life threatening condition and thoracocentesis must be performed immediately.

Haemothorax

A haemothorax is defined as blood within the pleural cavity. This can result from trauma, or accumulation of blood due to, for example, a clotting disorder and an active bleed has occurred. A haemothroax is initially stabilized (depending on cause) by performing thoracocentesis to remove blood.

Pyothorax

A pyothorax is caused by bacterial contamination of the pleural space, both cats and dogs can present with this condition. However, the causes can vary greatly. In cats, the most common cause of bacterial contamination is from the oral cavity of other cats – bite wounds to the chest. Other causes of pyothorax include:

- · Bacterial contamination from penetrating chest injuries
- · Migrating foreign bodies (grass seeds etc)
- · Inhalation and migration of foreign body
- · Rupture or perforation of the oesophagus
- · Rupture or perforation of the trachea
- · Bacterial pneumonia leading to lung abscessation
- · Bacterial spread from other site (systemic sepsis)

Flail Chest

When a rib segment is broken and becomes free-floating, it is termed a flail chest. Pain and dyspnoea are observed and often the flail segment can be located by visualization of the chest wall.

Pleural Effusion

The pleural lining is the lining of the thoracic cavity and outer surface of the lung, the lining covering the outer portion of the lung is the VISCERAL PLEURA, the lining covering the mediastinum and diaphragm is the PARIETAL lining, these two linings are separated by a thin layer of fluid to reduce friction between the two surfaces. A pleural effusion is defined as an excessive collection of fluid between the visceral and parietal pleura.

Causes of pleural effusion include:

- · Neoplasia
- · Heart worm
- · Congestive heart failure
- · Trauma
- · Hypoalbuminaemia

Delivering Oxygen

Provide supplemental oxygen. It is essential that the patient receives supplemental oxygen in the least stressful manner. The patient should be placed in sternal recumbency as this facilitates easier breathing. The method chosen is dependent upon the patient

presentation, disease and availability within the clinic.

Methods include:

- · 'Flow-by' / 'Fly-by'

- · Face mask

- · Head Tent

- · Oxygen Box

- · Nasal Oxygen – not recommended for immediate stabilisation as too stressful to patient and time consuming

Fly By / Flow By Oxygen

This is an excellent way to provide oxygen for a distressed patient on presentation.

Equipment Required:

- · Oxygen supply

- If this is not available the anaesthetic machine can be used, however ensure that the gaseous anaesthetic is turned off and the system has been flushed, so no anaesthetic residue is in the line.

- · A humidifier is preferred for any prolonged administration time

- Specific oxygen tubing is available that can be attached to the regulator / humidifier. This tubing can also be attached to the oxygen port on the anaesthetic machine with a small adaptor.

Procedure

- · Hold tubing as close to the patients airway as possible

- · If the patient has reduced consciousness you may be able to place the tubing just inside the mouth without distressing it

- · Have the oxygen on the highest flow rate

Face Mask

Although a face mask will provide a greater amount of inspired oxygen than 'fly by', it can be very stressful for the patient and often make the situation worse. Imagine if you could not breathe and someone tried to place something over your airway!

The mask, if used, does not have to be an airtight fit as expired carbon dioxide needs to be released.

High oxygen flow rates should be used and if full coverage of the mouth / nose is not possible, hold as close to airway as tolerated.

Equipment

- · Mask of appropriate size

- · Oxygen supply

- · Tubing

- · The anaesthetic machine is often used when providing oxygen via a mask, as the mask fits snugly on the end of the appropriate anaesthetic circuit.

- · Again, remember to ensure that the gaseous agent is turned off and the circuit has been thoroughly flushed prior to use.

- · Masks can also be used with a humidified oxygen source with the correct adaptor.

Head Tent / Box Oxygen

This is a simple and effective technique for providing supplemental oxygen, although may not be tolerated by some patients.

Equipment required

Elizabethan collar (or similar)

- · Glad wrap

- · Oxygen tubing

- · Oxygen supply

- · Tie for collar

Procedure

- · Collect appropriately sized Elizabethan collar and assemble

- · Cover the collar front with glad wrap / cling film, leaving a small gap at the top for expired carbon dioxide to be excreted

- · Secure glad wrap / cling film with tape

- · Position end of oxygen tubing on the bottom of the e-collar (away from gap) and secure in place with adhesive tape

- · Place e-collar on patient and secure with tie

- · Provide oxygen at a flow rate of 8-10L/min

Considerations to be aware of include:

- **Facial trauma** Although this is an excellent way to provide oxygen support when nasal oxygen cannot be placed due to facial trauma, the patient must be closely monitored for pooling of fluids (e.g. blood, saliva) within the head tent.

- **Heat** The head tent can become quite hot if a suitable area has not been left unwrapped. Careful monitoring should be provided to ensure panting from excessive heat or discomfort from heat within the box does not occur.

- **Patient observation** As the head of the patient

is enclosed within the oxygen tent, monitoring of mucous membranes, capillary refill time and pupil size and reaction are impeded. Therefore, other monitoring techniques should be implemented.

Oxygen Cage / Box

Oxygen cages are commercially available or can be homemade. Using a cage is a non-invasive and less stressful way to provide supplemental oxygen. It is necessary to have a 'clear' box so the patient can be visually assessed at all times.

Commercial oxygen cage

The commercial oxygen cage enables 'pure' oxygen to be delivered to the cage, eliminating the use of an anaesthetic circuit.

Home-made Oxygen Cage

This oxygen cage has been made from a plastic storage box.

Several sizes can be made

- · A hole was drilled at one end of the box to facilitate the placement of the oxygen source via the anaesthetic circuit.

- · Two holes are drilled at the other end of the box to facilitate removal of expired carbon dioxide.

- · Although, not as clear as the commercial anaesthetic cage, the patient can still be continuously observed.

Disadvantages

- · These boxes can become quite hot and therefore careful monitoring should be implemented to prevent this.

- · When the lid is removed from the box the oxygen content will drop dramatically and need to be replenished when re-sealed.

- · High flow rates of oxygen are required.

WSV18-0065

ANESTHESIA AND PAIN MANAGEMENT FOR VETERINARY DENTISTRY

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Learning objective: Oral and maxillofacial disorders require general anaesthesia for appropriate treatment. Therefore, anaesthesia and pain management are crucial areas of veterinary dentistry. An analgesic plan including a multimodal approach should be in place during the peri-operative period and for several days to a week after hospital discharge and it includes local anaesthetic techniques performed preoperatively. Patients must be comfortable and relaxed in order to provide comprehensive dental therapy. Although patients may be sedated deeply enough to tolerate local anesthetic blocks and dental procedures, sedation leaves the airway and lungs unguarded. An unguarded airway predisposes patients to aspiration pneumonia, both from regurgitation and from the water and debris associated with the procedure. Placement of an endotracheal tube is the safest way to protect the lungs. With an endotracheal tube in place, inhalant anesthesia is easily administered and becomes the best option for anesthetic maintenance in nearly every patient.

Anesthetic monitoring is significantly correlated with decreased morbidity and mortality. The following vital parameters should be monitored during general anesthesia: Pulse oximetry which can be a challenge to monitor during anesthesia for oral procedures since the probe can be easily displaced, however it can be placed over the ears and paws. Mean blood pressure which is particularly important in dogs and cats with chronic kidney disease. Respiration should be ideally monitored using a capnograph since monitoring of respiratory rate does not provide information of the "quality of the respiratory function" Body temperature should be maintained between 37 and 38°C and monitoring it is necessary to react on time and prevent hypothermia.

Most oral and maxillofacial disorders and therapies involve inflammation and tissue damage/trauma. Oral disease and associated pain is a welfare issue since it impacts quality of life and nutritional status. Dental disorders cause pain suffering, alter behaviour and cause physiological signs of stress Pain management is not only important from the ethical and welfare point of view but also as a therapeutic strategy. Pain management is performed preoperatively preoperatively and and postoperatively. It involves use of anesthetic drugs, techniques and their combinations aiming to minimise postoperative pain and discomfort.

Opioids are the first line of treatment in acute pain management. Unless contraindicated, NSAID therapy is commonly administered for approximately 3-7 days depending on type of oral disease/procedure. Local anesthetic drugs produce a reversible block of sodium and potassium channels and transmission of nociceptive input. Specific local nerve blocks techniques for oral and maxillofacial procedures include: inferior alveolar nerve block, palatine, infraorbital, and maxillary nerve blocks.

WSV18-0325

SVA PRACTICE MANAGEMENT

HOW TO EDUCATE, GAIN AND RETAIN IDEAL CLIENTS

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1. SS – How to educate, gain and retain ideal clients

Internet marketing can transform practice revenue, increasing the numbers of new clients and increasing the spend by existing ones. However, only a few practices have mobilised this powerful force. To do so, a 5-step marketing program is discussed for practices and individuals hoping to build strong brands. Using detailed case studies this session reviews SMART goals, creating a clear brand, and building a modern social media and digital marketing strategy.

1. Brand

Your brand should encapsulate the best features of your practice and those that make you stand out from other competing practices. Once established the brand should be consistent in all areas of marketing. As Simon Sinke tells us 'People don't buy what you do they buy why you do it' so think carefully about why you are doing what you do and experiment with methods of communicating this.

Evidence shows us that people will pay more for a brand they trust, and that if people trust your brand they are far more likely to use your services. Demonstrating that you are trusted by your clients is straightforward and requires only minimal investment and time input. Displaying third party reviews on your website and having an active social media profile are two easy ways to get going with this.

2. Platforms

Once your practice has an established brand careful planning is necessary to ensure that the brand comes across clearly on every platform. There are a plethora of available platforms and decisions need to be made, taking into account your core values, target demographic and return on marketing investment. It is essential that you play to the strengths of each platform.

3. Content

Content going out across the various digital platforms should work in sync with any offline marketing activities and reflect any seasonal aspects of the caseload. A good mixture of "pure marketing" and relevant educational content is valuable. Content should be tweaked for optimum performance on each platform - what works on Facebook may not work on a website or an email, for example. Where possible, content should be targeted to the most appropriate audience segment - too much irrelevant content (the "shotgun approach") can damage your brand image.

4. Exposure

Publishing relevant content and having a strong brand are only useful if they are seen by local pet owners. Facebook Ads and offline marketing will help to build up the 'Likes' or potential audience of the page. Creating a vibrant community and Facebook Ads will ensure people that like the page see important posts. Search Engine Optimisation will ensure that the practice is found when local owners are searching for a vet or for relevant content.

5. Measure

One of the joys of digital marketing is that its results are usually measurable. Defining the measurements that will be used as Key Performance Indicators should form part of the digital marketing strategy. These should be used to optimise and refine the approach on a regular basis, allowing your practice to be responsive to your client base and local communities.

WSV18-0149

NAVC SHORT TOPICS FROM EXPERTS

HOW I USE ULTRASOUND TO PERFORM PULMONARY TISSUE SAMPLING

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HOW I USE ULTRASOUND TO PERFORM PULMONARY TISSUE SAMPLING

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Percutaneous ultrasound-guided sample collection is used to obtain needle aspirates and tissue core biopsy specimens for a more definitive diagnosis due to the non-specific nature of most ultrasound abnormalities. Common examples are hepatomegaly, diffusely hyperechoic liver, multifocal liver and splenic nodules. Increased echogenicity and nodules have many differential diagnoses from benign to malignant. While ultrasound is screens and identifies diffuse and focal and multifocal lesions, it is non-specific. Ultrasound-guided tissue sampling offers real-time visualization of needle placement for precise sampling of a selected portion of an organ or a focal abnormality, such as a mass lesion. Common clinical indication in animals include evaluation of enlarged lymph nodes, masses nonspecific hepatopathies and splenic disease.

Patient selection and preparation

Animals should have a complete sonographic examination performed of the body region. Complete work up with blood work is also imperative and underlying coagulopathies should be screened for. Animals with coagulopathies should not have tissue sampling performed as a general rule.

Sedation is not always necessary and many animals can have the liver and spleen sampled with a fine needle aspirate without it. However, risk should not be taken and if the animal is not cooperative, painful or panting heavily, then sedation should be performed for FNA. On the other hand, sedation is always recommended for Tru-Cut biopsy sampling.

The approach to the organ should be investigated carefully. Check the probe position so that the needle has the shortest path between the skin and the lesion.

Never pass between two body cavities or another organ to sample the lesion or organ.

Typically, cavitary lesions can be sampled with FNA, but not a tru-cut biopsy. Solid parenchymal organs or lesions

can have FNA performed.

Vascularity is not typically a contraindication to sampling. For example, the kidney is highly vascular but can have a tru-cut biopsy performed very safely. Thyroid carcinoma is very vascular and bleeds readily when sampled, but the bleeding is self-limiting.For fine needle aspiration (FNA), an 18- to 20-gauge needle is inserted into the selected site with ultrasound-guidance, most commonly using a free hand technique. Tissue core biopsies for histopathology are obtained using a larger needle (Tru-Cut®) with an automatic biopsy device, which allows the operator to control the depth of needle placement and length of sample obtained. A 14- to 18-gauge needle is suggested depending on the location and size of the lesion being sampled. Generally, 2-3 specimens are obtained for histopathologic evaluation. As a general rule, 18G biopsy needles are preferred in small dogs and cats and small organs and 14-16G needles for larger dogs and large organs like the liver.**Technique for FNA**

Clip and clean the skin with a cleaning solution like Hibiscrub or other skin prep and wipe with alcohol. Use a 3 or 6 ml syringe with the plunger pulled back 1ml. Determine the probe position and place the needle in the same plane as the scan plane of the probe (middle of the probe) and use a 45 degree angle. Place the tip of the needle in the skin at the mid point and edge of the probe and be careful to not pierce the probe housing. Insert the needle and make short in and out (woodpecker like) excursions with the needle while watching the screen to make sure the needle is visible just under the skin so that when it is further inserted that it can be well visualized. Once the needle position is confirmed, advance the needle to the lesion and stab the needle in and out a few times, without aspirating, then withdraw the needle without suction applied. Remove the needle, draw the plunger on the syringe back, and re-attach and spray the contents of the needle onto a clean glass slide.

Use a different syringe and needle for each organ and do not place the glass slides of different lesions and organs near each other when spraying the samples onto the slides and this can cause cells from one region to be sprayed onto the slides of another organ, confusing the diagnosis.

If the stab technique does not result in enough material on the slide, then repeat the procedure and use aspiration with the plunger during the stabbing movement to attempt to get more cells in the needle. This can use to more blood dilution, however.

Technique for Tru-cut biopsy

Typically, the liver and kidney are the main organs of interest for Tru-cut biopsy procedures. They are larger and more amenable than smaller organs.

Lesions less than 4cm in size are typically not suited for Tru-cut biopsies. Small livers can be difficult to biopsy (cats, small dogs) and one should not hesitate to send those animals to laparoscopy for surgical biopsies.

The main difference from FNA is that the needle is larger and the animal should be sedated. A small nick with an 11-blade in the skin is necessary to advance the needle through the skin and some force is required to penetrate the body wall. Once the needle is through the body wall, place the tip of the needle on the surface of the organ to be sampled and line it up with the plane of the ultrasound image.

Measure the surface of the organ with calipers to see how deep 2cm is (typical penetration depth) to be sure that the needle will not strike a vessel or other vital structure deep to the lesion being sampled.

Fire the spring-loaded device and remove the needle from the organ. Open the needle sample volume notch and remove the delicate tissue core sample by running a stream of sterile saline over it and the sample will slide into a reciprocal or slide. Put the sample in formalin directly afterwards.Check the patient for focal fluid accumulation around the biopsy site. The PCV may also be checked periodically a few hours later to monitor for blood loss. It is suggested to perform biopsies in the morning and observe the animal in the hospital for the day to monitor for blood loss. This is typically not necessary with an FNA, however.

WSV18-0232

ORTHOPEDIC SURGERY

OPEN FRACTURES: IS THERE A BETTER OPTION THAN EXTERNAL FIXATORS?

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LEARNING OBJECTIVES

At the end of this session you will be able to:

- Use the grading system for open fractures to decide treatment options
- Recognise the significance of a full thickness skin wound over a fracture
- Decide on appropriate antibiotic use for open fractures based on an understanding of the difference between contamination and infection

Open fractures are a common presentation in small animal practice. The long- standing approach to managing open fractures has been open wound management and the use of external skeletal fixators (ESFs). This "traditional" approach has been associated with a relatively high occurrence of non-union resulting in the need for revision surgery and, in some cases, limb amputation. Prolonged open wound management leading to "treatment fatigue" of both owners and veterinary staff, significant on-going patient morbidity and often the development of multi-resistant bacteria are also associated with this older-style approach.

More recently, through a better understanding of managing traumatic wounds and a better understanding of fracture biology, newer approaches to managing open fractures have been developed that limit or avoid many of the negative issues associated with "traditional" treatment of open fractures. These newer approaches concentrate on early primary closure or early delayed primary closure of open wounds through a variety of strategies in combination with fracture stabilisation with bone plates.

This session will focus on the following key points:

- The treatment of an open fracture on initial presentation affects the outcome. Open fractures are contaminated initially rather than being infected. Infection will only develop if wound management is not appropriate. Infection is more likely with prolonged open wound management and in the presence of poor soft tissue blood supply.
- The majority of infections in open fractures are acquired in hospital. The long- term prophylactic use of antibiotics in open fractures is not justified and encourages the establishment of resistant bacteria within your practice.
- Longer courses of antibiotics if infection does develop is justified but should be based on cultures of deep tissue samples from the fracture site.

- Classifying the type of open fracture helps determine both the prognosis and the recommended treatment options.
- Primary or delayed-primary wound closure **following** appropriate debridement improves blood supply to the healing fracture, reduces morbidity, reduces the likelihood of producing resistant bacteria, saves time and in most cases is ultimately cheaper than on-going open wound management.
- ESFs in many severe open fractures (Type III) will **not** last the distance until the bone is healed and will fail or need revision before the fracture has healed.
- Bone plates and interlocking nails are **not** contraindicated in open fractures. Early re-establishment of healthy well-vascularised soft tissue over a healing fracture speeds fracture healing and reduces the occurrence of infection.
- Should implants be removed after fracture healing? Implants placed in open fractures may be contaminated and so there is some degree of risk of future cryptic infection developing. How likely this is to occur in canine and feline open fractures is not clear. Currently owners should be advised that bone plates probably should be removed on completion of fracture healing.

Open Fracture Classification

Identifying the type or severity of open fracture is useful in determining the prognosis and in deciding on the treatment options available in that case.

Open fractures may be divided into three types based on the severity of the injury.

Type I

Type I open fractures are **low energy** fractures where an open wound typically less than 1cm is created by the internal force of a bone fragment protruding through the skin. These have a small external skin wound that in many cases is not apparent until the hair is clipped for surgery.

Any full thickness skin wound near a fracture should be classed as open. Probing the wound to determine whether it connects with the fracture is contraindicated, as this will carry bacteria and debris into the soft tissues and fracture site.

Type II

Type II open fractures involve higher energy trauma in which the soft tissue wound is created by a combination of internal and external force. There is more damage to the soft tissues than with a type I. The skin laceration is larger than a type I but sufficient tissue exists to close the wound without needing to use a skin flap or other advancement technique.

Type III

Type III open fractures are the most severe type and result from **high-energy external trauma** such as

gunshot injuries and motor vehicle trauma producing shearing injuries. Extensive skin and soft tissue injuries exist. There is **insufficient soft tissue to cover the wound**.

Complications including infection are significantly higher in type III fractures reflecting the greater degree of soft tissue compromise. Type III open fractures can be repaired with very good outcomes however this requires considerable financial and time commitment on the owner's part to achieve a good outcome.

Open wound management of type III open fractures is prolonged and expensive and is nearly always complicated by the development of significant infection including the establishment of multi-resistant bacteria.

Early primary closure or delayed- primary closure of type III fractures usually "short- circuits" prolonged open wound management, reduces morbidity and the occurrence of infection, speeds fracture healing and is ultimately cheaper and less time- consuming than open wound management.

In most type III open fractures, treatment with an ESF will facilitate open wound management but the ESF will rarely last long enough without revision for the fracture to heal. In most cases where an ESF is used revision surgery or replacement with a bone plate is necessary.

It is **not** contraindicated to use bone plates or interlocking nails in severe open fractures.

Management of Open Fractures
* **protect the wound from nosocomial contamination**

Open fractures should be **covered with a quick sterile dressing** to prevent further contamination while patient assessment and stabilisation is underway. In a study of open fractures in people it was found that 80% of infections were hospital-derived with only 20% of subsequent infections developing from bacteria present on initial presentation.

It is important when applying temporary support to open fractures at this stage to <u>not</u> reduce protruding bone fragments, as this will further contaminate the soft tissues at the fracture site. Reduction of any protruding bones should not be performed until <u>after</u> debridement.

·administer intravenous antibiotics

Early administration of broad-spectrum intravenous antibiotics as soon as possible on presentation has been shown to reduce subsequent infection rates. The degree of soft tissue compromise in type III open fractures means that these are more susceptible to infection than both type I and type II fractures.

The most common bacteria present in open fractures are gram-positive bacteria such as **Staphylococcus** spp. and **Streptococcus** spp. and gram-negatives such as **E.coli,**

Pseudomonas Klebsiella. First or second-generation cephalosporins or **intravenous** forms of potentiated amoxicillin / clavulanic acid are appropriate.

Addition of a fluoroquinolone has been shown to be beneficial in reducing infection rates in type III open fractures in humans.

It is important to differentiate both the concepts of prophylactic and therapeutic antibiotic usage and contamination and infection.

Antibiotics should be administered prophylactically for **three-five days** initially then subsequent administration either prophylactically or therapeutically based on exit cultures or deep tissue cultures in response to clinical signs of infection if indicated. The long-term usage of antibiotics is **only** indicated in established osteomyelitis and is not routinely necessary in open fractures.

·wound debridement

Type II and Type III open fractures should be debrided prior to fracture stabilisation ideally as soon as the animal is physiologically stable for anesthesia for debridement. Type I fractures rarely need debridement preoperatively.

Debridement should be performed in a sterile surgical manner. The wound should be covered in clean **single use** K-Y jelly, which is water miscible and protects the wound from the hair and debris of clipping. After clipping the K-Y jelly is cleaned away as part of the surgical preparation. A full surgical preparation should be performed. Do **not** use open or multi-use K-Y jelly.

The wound should be explored in a sterile manner. It is important to remember that open fractures are biologically compromised areas and as such are **more susceptible to infection than normal sites**.

Copious lavage with warm sterile isotonic saline should be undertaken. Use of a sterile giving set, three way stopcock, 35ml syringe and 18-gauge needle will facilitate this. Given the large amount of fluid used the use of waterproof drapes and/or suction is advisable to minimise the risk of nosocomial contamination, as cloth drapes will be rendered non-sterile once wet.

Sharp debridement of necrotic tissue and any particulate matter should be performed. Bone fragments that have soft tissue attachments should be minimally disrupted. Small bone fragments with no soft tissue attachments should be discarded. In severe injuries where there is some doubt over the viability of soft tissue serial debridement over several days will enable subsequent identification of devitalised tissue.

Controversy exists over the value of entrance or exit cultures. An exit culture at the time of major debridement and, if definitive fracture repair surgery is delayed, then again at the time of fracture repair.

Type I open fractures rarely require any debridement prior to definitive fracture stabilisation however the same approach to lavage should be undertaken at the time of fracture repair as with the more severe types.

·**Definitive fracture repair**

Rigid stability is essential in the repair of open fractures. For this reason **intramedullary pins, orthopaedic wire and external coaptation are <u>contraindicated</u>** in open fractures.

External skeletal fixators (ESFs) have been reported to be the fixation method of choice in type II and III open fractures (provided a sufficiently rigid frame can be placed). This dogmatic advice should be carefully considered as in many type II and

nearly all type III open fractures **definitive** fracture repair with an ESF is usually not achieved. This is because the poor biology of type II and III open fractures means that healing will be prolonged and, in most cases, **the ESF will fail before the fracture has healed**.

ESFs have several **theoretical** benefits in open fractures:
- ESFs provide rigid stability remote from the fracture site. This minimises soft tissue damage and reduces the likelihood of infection localised around the implant.
- ESFs allow easy access for open wound management.
- ESFs are useful in radial and tibial open fractures (which are the most common sites of open fracture)

The reality however is that the majority of ESFs placed will fail to a greater or lesser degree **prior to completion** of fracture healing in more severe open fractures necessitating ESF revision or transfer to some form of internal fixation. In some cases revision surgery of the ESF with removal of loose pins and replacement with other pins may be effective. In most cases however removal of the ESF completely and replacement with a bone plate and bone graft to manage the delayed or non- union that has developed is necessary.

It is advisable to inform owners prior to surgical management of type II and type III fractures with an ESF that initial treatment with an ESF to facilitate open wound management followed by definitive treatment with internal fixation once the open wounds are resolved will be necessary.

Bone plates, interlocking nails or ESFs are suitable for type I open fractures as they all can provide sufficiently rigid stability. Bone plates are the implant most commonly used in managing type I open fractures.

Bone plates and interlocking nails **can be used** for type II and III open fractures. **In our practice bone plates are used in preference to ESFs** in type II and III fractures **provided that the open wound can be closed either primarily or through delayed-primary closure**. To

achieve this in severe fractures usually requires closure of the wounds with axial pattern flaps or other soft tissue transfer methods. This has the dual advantage of closing the open wound and replacing traumatised tissue with **robust well-vascularised tissue that is much more resistant to soft-tissue infection**.

The advantage of bone plates and interlocking nails over ESFs is that they have greater longevity, which is often necessary in more severe open fractures.

Owners should be advised prior to surgery that removal of the bone plate after fracture healing is advised to reduce the risk of future cryptic infection that can potentially occur when implants are placed in contaminated sites. ESFs also require subsequent surgery to remove although removal is relatively simpler than bone plate removal.

Cancellous bone grafting is recommended in open fractures managed with open reduction. Direct placement of a graft is usually possible in type I and II open

fractures and in all type III fractures where primary closure or delayed primary closure is performed. Where prolonged open wound management is elected in type III fractures bone grafting cannot be performed until healthy granulation tissue has covered the fracture site. The graft is usually placed at a second surgery through a separate incision to the open wound through an area of good soft tissue coverage.

Where ever possible closure of open wounds in type III open fractures using an axial pattern flap or pouching or other methods of closing the wound with well- vascularised robust soft tissue is preferable to on-going open wound management. This improves outcome, minimises morbidity, minimises treatment time and is ultimately cheaper than prolonged open wound management.

Dos of open fractures
- Do place a quick sterile dressing to cover the open wound while treating the animal for shock. This will reduce the likelihood of the traumatised soft tissue wound picking up a hospital-derivedinfection.
- Do provide temporary splint support with the initial sterile dressing in fractures that are distal to the elbow or stifle joint. This will greatly reduce the animal's pain and limit ongoing damage of the soft tissue envelope of the fracture from the fractured boneends.
- Do give prophylactic intravenous broad-spectrum antibiotics on initial presentation and for the first few days only until the results of the bacterial deep tissue culture obtained at surgery (the "exit" culture) are obtained. If the exit culture at the time of surgery grows bacteria then this suggests an established or an establishing

infection and requires a therapeutic course ofantibiotics.

- Do appropriately lavage and debride open wounds associated with fractures to reduce the amount of devitalized tissue andcontamination.

- Do use an appropriate method to close an open wound and minimize the need for ongoing open wound management. Closure should be with healthy well- vascularised soft tissue that is not undertension.

Don'ts of open fractures

- Do **not** use long-term antibiotics **prophylactically**. This will increase the likelihood of developing resistant bacteria. Only use long-term antibiotics **therapeutically** to treat a confirmed bacterial infection. The choice of antibiotic should be based on culture and sensitivity identification of a deep tissue sample or swab from the fracture site.

- Do **not** obtain superficial tissue or discharge samples for bacterial culture. They will not represent what, if any, bacteria are present at the fracturesite.

- Do **not** use ESFs for **definitive treatment** of open fractures unless the fracture will heal relatively quickly before the ESFs begin to fail. Most type III and many type II open fractures will **not heal** before the ESF fails, necessitating revision surgery. Owners should be advised prior to treatment that a 2-stage surgery with ESF and open wound management followed by further surgery with a bone plate etc may be necessary.

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VECCS

APPROACH TO TRAUMATIC SHOCK

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Initial traumatic injury can be a combination of blunt force tissue injury, blood loss and hypoxia due to impaired pulmonary function. This talk will review the pathophysiology of the initial hours of traumatic injury and outline an up-to-date approach to managing traumatic shock in the first 6 hours.

Pathophysiology of traumatic shock

Blunt force trauma, caused by incidences such as motor vehicle accidents, dog bite injuries or falling from a height, is associated with changes in tissue perfusion and a cascade of pro-inflammatory mediator release. Lack of oxygen to the tissues leads to release of Danger-Associated Molecular Pattern molecules (DAMPs) that act as chemo-attractants and activate immune cells, such as neutrophils and monocytes. Molecules such as high mobility group box-1 (HMGB1) and cell components, such as mitochondrial or cell-free DNA, are released early in trauma and can promote an acute inflammatory response.

Release of DAMPs is likely one of the key early steps to recruitment of neutrophils and monocytes to the site of injury but DAMPs are also probably one of the main instigators of distant organ injury as a part of a 'second hit'.

In concert with the release of DAMPs, the innate immune system is promptly activated, including neutrophil recruitment and complement activation. Inflammatory cytokines are also quick to rise within hours of traumatic injury, including interleukin (IL)-6, IL-1β, IL-8, IL-10 and tumour necrosis factor-α (TNFα). They are joined by a host of other pro-inflammatory and vasoactive mediators released from the endothelium, immune cells and damaged cells. These mediators are designed to mediate tissue recovery and repair, and mitigate organism invasion, however, the response can be excessive, leading to Systemic Inflammatory Response Syndrome (SIRS) and, potentially, Multiple Organ Dysfunction Syndrome (MODS).

In addition to the inflammatory cascade that occurs, ischaemia due to hypovolaemia adds additional endothelial and tissue cell injury. After reperfusion, complement activation, production of reactive oxygen species, platelet activation and neutrophil margination prompt a cascade of pro-inflammatory events that can lead to distant organ injury. All of these responses can contribute to increased vascular permeability, loss of

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vasomotor tone (vasodilation) and remote organ damage in the hours and days after an ischaemic event.

Acute traumatic coagulopathy (ATC) can develop rapidly in trauma patients, not only due to dilution from intravenous fluids but also as a part of the pathyphysiological response. The majority of ATC is most likely caused by activation of the protein C pathway. Activated protein C has a direct inhibitory effect on coagulation proteins (V and VIII) and plasminogen activator inhibitor-1, causing hypocoagulation and enhanced fibrinolysis. Tissue plasminogen activator is also released secondary to endothelial activation. One study that measured coagulation variables before intervention in dogs with severe trauma found hypercoagulability in a third of the dogs.(1) In contrast, a study in dogs with spontaneous haemoperitoneum found the presence of hypocoagulability, protein C deficiency and hyperfibrinolysis.(2) Another study that evaluated dogs with trauma within 12 hours of hospitalization found that hypocoagulation was associated with mortality.(3)

Both platelet hyperreactivity and hyporeactivity have been also observed in trauma, likely reflecting earlier and later effects of injury, respectively. One canine model of haemorrhagic shock has also identified platelet hyporeactivity,(4) whereas another did not. (5) Acidosis, associated with shock, interferes with enzymatic reactions in the body and also contributes to hypocoagulation. Hypothermia, often present in the early hours after traumatic injury, also contributes to hypocoagulation and poor peripheral blood flow.

The combination of acidosis, coagulopathy and hypothermia, known as the lethal triad, contributes to morbidity and mortality in human trauma patients. Several studies have associated mortality with acidosis in dogs with trauma.(3, 6) Hypoxia due to lung injury may also contribute to complications. The damage done by these conditions in combination with hypovolaemia can propagate an ongoing inflammatory response and coagulopathy, for the reasons outlined above. If patients survive the initial 24 hours, then a secondary 'immune paralysis' can ensue, making the patient susceptible to sepsis and MODS. These complications can lead to death of the patient despite best efforts to re-establish effective circulating blood volume, replace coagulation factors and achieve metabolic homeostasis.

The first phase of resuscitation

Most trauma patients will present with physical examination signs consistent with vasoconstrictive shock, such as tachycardia, pale mucous membranes and weak femoral pulses. Even if the changes appear mild (such as only the presence of tachycardia), the possibility of hypovolaemia should be addressed before any other actions taken. Even without blood loss, trauma patients may be relatively hypovolaemic due to, for example,

shedding of the endothelial glycocalyx or regional vasodilation (such as splanchnic) due to inflammatory mediators. This is especially true for dogs and is why their gut is called the 'shock' organ. Although rare, patients may also have an obstruction to blood flow, such as pericardial tamponade.

On initial assessment when abnormal perfusion parameters are first identified, the 'first phase' of resuscitation should be commenced immediately. This includes a fluid challenge to test if the patient's physical examination abnormalities respond to blood volume expansion. Blood volume expansion can be achieved by a number of different types of fluid therapy including isotonic crystalloids, hypertonic saline and synthetic colloid fluids. Transfusion products are only appropriate for the first phase if there is evidence of massive bleeding combined with severe shock.

Isotonic crystalloids provide rapid blood volume expansion to re-establish tissue perfusion and are easily excreted. They can be titrated according to the volume deficit. However, aggressive resuscitation with isotonic crystalloids in the first phase can contribute to bleeding, damage to the endothelium and peripheral oedema. All fluids that dilute plasma constituents cause a dilutional coagulopathy. This is often recognised by mildly prolonged coagulation tests after a bolus. This effect may not be clinically relevant unless combined with bleeding and traumatic coagulopathy. Also, the larger volume causes a spike in blood volume expansion before redistribution,(7) which may disrupt an established blood clot. Other effects of rapid fluid administration may contribute to inflammation (see notes for 'The Adverse Effects of Rapid Fluid Administration'). Doses of isotonic crystalloids should be judicious and titrated to effect for these reasons. However, the benefits of isotonic crystalloids usually outweigh the adverse effects and remain the **most appropriate fluid** for the first phase of resuscitation. Overdosing crystalloid likely has less adverse effects than overdosing other fluid types.

Hypertonic saline has limited effects in regards to blood volume expansion(7) though it can be useful for reducing cerebral oedema in traumatic brain injury.(8) The use of synthetic colloids has become controversial. (9) There is experimental evidence that some synthetic colloids cause mild platelet dysfunction, and deficits on coagulation tests, whereas other studies have shown no effect beyond hemodilution. There is currently no strong evidence that synthetic colloids cause clinically relevant harm in veterinary patients. However, in the actively bleeding patient, these fluids must be used judiciously and in small doses, if at all. If blood products are an immediate option, then they should be considered instead. Based on our group's research that is yet to be published, gelatine products are best avoided due to multiple adverse effects in the experimental setting.

Second phase of resuscitation

During initial blood volume expansion, the patient is usually surveyed again by physical examination as well as point-of-care ultrasonography, if available. This gives the clinician an idea of where the major injuries are located, if there may be significant blood loss (such as intra-abdominal) and if other immediate interventions are required, such as thoracocentesis. If the patient continues to show signs of shock, despite initial blood volume expansion and analgesia, then there are several options to consider, including further fluid therapy, transfusion, antifibrinolytic therapy and, in rare cases, immediate surgical intervention.

Transfusion products, especially transfusion of stored red cells, are associated with increased free iron concentrations and a pro-inflammatory response. Red cell products that have been stored for a longer period have been associated with coagulation disturbances and thromboembolic disease in canine recipients.(10) There is also risk of acute reaction with transfusion of red cells and plasma.(11) Due to these known adverse effects of transfusion products, they should only be administered for the clinical indications of anaemia and coagulopathy.

If the patient requires further blood volume expansion, has no evidence of coagulopathy or active bleeding, has no prior history of renal disease and there is physiologic justification for restricting interstitial fluid accumulation, then synthetic colloid solutions may be considered in this phase of resuscitation (preferably low molecular weight hydroxyethyl starch (HES)). If a patient requires blood volume expansion and there is a good reason to avoid isotonic crystalloids, then synthetic colloids currently have the least amount of clinical evidence associated with harm, compared to plasma and human albumin products. However, due to the possible adverse effects of coagulopathy in a trauma patient, doses should be limited to less than 20mL/kg in a 24-hour period. Prospective clinical trials are still needed to determine if synthetic colloids, particularly HES, are harmful to veterinary patients.

Administration of an antifibrinolytic may be beneficial in the initial phases of resuscitation, especially for patients with active bleeding or evidence of hyperfibrinolysis on viscoelastic coagulation tests. Although the benefits of antifibrinolytic therapy, such as tranexamic acid, have been well identified in human trauma patients, the evidence in veterinary medicine is limited. However, tranexamic acid is unlikely to cause harm when administered as a slow IV bolus followed by a constant rate infusion for several hours,(12) and may be beneficial in a bleeding patient.

Decision-making trees will be presented in this talk, reflecting the author's approach to traumatic shock. Attendees are welcome to request a copy of the flow diagrams by email.

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WSV18-0214

SVA FELINE

CLINICAL APPROACH TO CHRONIC UPPER RESPIRATORY TRACT DISEASE IN CATS - HOW RHINOSCOPY CAN HELP

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Indications, History and Physical Examination

Before conducting any endoscopic examination, it's essential to first acquire a detailed clinical history and thorough patient examination to accurately localize the disease process. Additional general screening tests (e.g. haematology, biochemistry, electrolytes and urinalysis) can also be performed to establish the general health and potential anaesthetic risk of the patient. The depth of evaluation will vary depending on the case; however, every case should receive a comprehensive history and full clinical examination.

The indications for rhinoscopy include: chronic and/or recurring sneezing and reverse sneezing, nasal discharge, epistaxis, abnormal respiratory sounds such as stertor (nasopharyngeal) and/or stridor (laryngeal). Physical examination should therefore include an assessment of nasal air ow (decreased or normal, unilateral or bilateral change) and palpation of the palate and facial bones for pain, swelling, ipsilateral epiphora, ipsilateral exophthalmos or evidence of bony lysis.[1,2] A full oral examination should ideally include a dental assessment and oropharyngeal examination. If dental disease is suspected, dental radiography may be indicated, paying special attention teeth 104, 204, 108 and 208. Neurological examination should focus on cranial nerve evaluation and also detecting signs of cerebral dysfunction such as weakness, decreased conscious proprioception, and visual deficits indicative of invasive disease. If clinically suspicious of Cryptococcosis, cytology slides of nasal secretions and Latex Cryptococcal Antigen Testing (LCAT) should be submitted, especially in those patients travelling from endemic areas (e.g. Canada, Australia, USA). A thorough otoscopic examination should be performed of the external ear canals.

Cats with epistaxis should have a coagulation profile (e.g., platelet count, PT/PTT and/or a mucosal bleeding time-**MBT**) performed and their blood pressure checked prior to starting as these patients may have an increased risk of bleeding.

Bacterial culture and antimicrobial susceptibility testing of superficial nasal swabs are often unrewarding and not generally recommended.[3] Results typically yield normal intranasal bacterial flora and are difficult to interpret. Others suggest that results of culture and sensitivity testing may be useful in guiding antibacterial therapy.[3] Cultures of nasal biopsy samples may be more representative for deep mucosal infections, but this has not been definitively proven.

FHV-1 or FCV virus isolation and nucleic acid amplification techniques are often used to implicate infection by these organisms. FHV-1 PCR assays are widely available and feline calicivirus reverse transcriptase PCR assays are also available. However, none of the PCR assays for FHV-1 have been shown to distinguish between wild-type virus and vaccine virus. Additionally, test sensitivity (detection limits and rates) varies greatly between the tests and laboratories. These infectious agents can be detected in healthy cats as well as in clinically ill cats. Thus, the positive predictive value for these assays is low and thus diagnostic: cost value is questionable in those cats with chronic nasal disease.

For a complete evaluation of the nasal cavity, sinuses and nasopharynx the assessment should include imaging such as (radiographs), CT/MRI, dental radiography, and rhinoscopy.

Introduction to Rigid Endoscopy

The novice endoscopist should strongly consider participating in hands-on wet lab courses, provided by experienced endoscopists, before attempting rigid rhinoscopy in the cat. This will ensure a level of competence that justifies the potentially high learning curve and initial investment in providing this type of service.

What is a Rigid Endoscope?

In simple terms, a rigid endoscope is a long slender stainless steel tube with a series of solid glass rod lenses which allow for the transmission of light and image.[1,2] Light transmission is achieved from the use of an extracorporeal light source attached to the optical end of the endoscope. The image is then viewed via an oculus, or eye-piece, directly to the operator's eye or a video camera which can be transmitted to a video monitor and stored in an archiving system.

Laryngoscopy

Upper airway examination begins with laryngoscopy on induction. Laryngeal structure (i.e. anatomy) and function should be assessed in relation to phase of respiration. This examination should be assessed under a light plane of anaesthesia. It is vital for an assistant to 'announce' the phase of respiration and to not confuse normal movement to **paradoxical** movement found in complete laryngeal paralysis.

Caudal (Flexible) Nasopharyngoscopy:

At the beginning of the procedure, a retroflexed examination behind the soft palate should be performed in attempt to exclude mass lesions, nasopharyngeal stenosis or foreign bodies. A dental mirror and bright light can sometimes provide an image of this region, or a specialised instrument with light source and a flexible mirror can be obtained from commercial vendors.

A 4.0mm-5.0 mm diameter 2-way deflection endoscope with biopsy channel can access the nasopharynx in all but the smallest dogs or cats.

Nasal flushing, culture and cytology

Vigorous nasal flushing can be useful to dislodge mass lesions or foreign bodies. Cytology of nasal flush fluid is likely to be very superficial and of limited value in most cases. In particular, neoplastic conditions may be misdiagnosed as rhinitis following the finding of inflammatory cells only on cytology.[4,5] Cytology can, however, be highly diagnostic for diseases such as nasal cryptococcosis and friable tumours (e.g. lymphoma).

Rostral (Rigid) Rhinoscopy

This procedure can be performed quite easily on most feline patients using a 1.9mm x 30 degree telescope with sheath.

This instrument has two-way stopcocks for continuous fluid ingress and egress which removes blood, mucus or other tissue debris from the field of view. Another advantage of continuous fluid irrigation is that it can act as a superior medium and enhance tissue magnification compared to that of air.[1] The entirety of both the dorsal and ventral nasal meati can be examined adequately to the level of the ethmoid turbinates.

Patient Preparation

Sternal recumbency places the patient is in a more 'natural' position in relation to the viewing monitor. The head is propped up with either a sandbag, wedge foam protected with an incontinence pad, or rolled towel. A cut down needle cap[6,7] is used as a speculum to allow for evacuation of infused fluids from the nose, to be directed out of the mouth.

A 1L bag of sterile saline is hung and connected to one of the stopcocks of the endoscope sheath. Another giving set can be attached to the egress stopcock and allowed to drain into the wet table or into a bucket. The endoscope **unit** is held in a 'pistol' position with the light guide cable pointing towards the ground. Continuous irrigation as will help magnify structures as well as remove discharge and haemorrhage that can obscure the view. Control of saline flow is best managed using the control on the ingress port on the sheath.

Biopsy

The operator can collect biopsy using a pair of 3 mm rigid cupped biopsy forceps. These can be placed in a premeasured depth (not to exceed the length measured from the tip of nose to medial canthus of eye). Nasal samples with bone fragments/ spicules represent deep sampling technique which is necessary in many cases for an accurate diagnostic interpretation. Samples should be submitted for histopathology (+/- PARR), bacterial culture & sensitivity and aspergillosis culture if indicated.

Complications

Haemorrhage is the commonest complication of anterior rhinoscopy but is rarely long lasting or significant. Aspiration of fluid can be prevented by fitting an appropriately sized endotracheal tube and leaving adequate space for the free flow of irrigant fluid over the free edge of the soft palate and out through the mouth.

Conclusion

In conclusion, the author's experience using rigid endoscopy has provided an easy and rewarding minimally invasive alternative to traditional diagnostic and surgical interventions for upper respiratory conditions. Endoscopy can be an extremely valuable and versatile part of clinician's diagnostic and therapeutic armamentarium.

Suggested Reading:

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ANIMAL WELLNESS & WELFARE

JUST BECAUSE WE CAN, SHOULD WE?

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Pets may exhibit behaviours that owners find unacceptable or distressing. Therefore many ways of managing pets and their behaviour have been recommended. From this many training techniques and devices have been developed but not all of them are humane. They often promise "quick fix" solutions and thus appear attractive to owners. Unfortunately the "normal" pet may respond differently from the pet with mental health issues. The focus should always be on the effect it has on the pet and its short and long term welfare.

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NUTRITION AND DIAGNOSTIC IMAGING (LECTURES GIVEN IN MANDARIN CHINESE)

THE ROLE OF NUTRITION IN THE MANAGEMENT OF FELINE DISEASES

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THE ROLE OF NUTRITION IN THE MANAGEMENT OF FELINE DISEASES

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Nutritional treatment in critical care patients is an important factor of the complete treatment plan. Assessment of nutritional status and careful consideration of the disease course will help the selection of the most appropriate feeding method. Enteral nutrition is better than parenteral nutrition when the gastrointestinal tract is functional. Common routes of enteral feeding include nasoesophageal, esophagostomy, gastrostomy, and jejunostomy feeding tubes. Parenteral nutrition is indicated for patients who cannot tolerate enteral feedings, or are high risky anesthetic candidates for feeding tube placement.

An anorexia 4 kg pacreatitis patient is admitted for assisted nutrition support by feeding tube. This cat's Resting Energy Requirement is: RER=30×4+70=190 calories per day. Using a standard illness factor of 1.2, this cat requires 228 calories per day. The maximum meals size is 45 ml/kg (stomach capacity of the cat). The cat is fed one third of the RER (76 calories) on day 1, two thirds of the RER (152 calories) on day 2, and all of the RER after day 3. The risk of re-feeding syndrome supports the practice of slow initiation of full nutritional support, monitoring of electrolytes and glucose, phosphorous, anemic and addition of vitamins (especially thiamine) the nutritional formula.
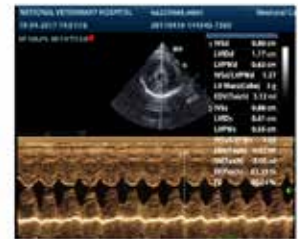
Appetite stimulating medications will need to be considered in each case to ensure rational use of these agents. Appetite stimulation drug should never replace monitoring and ensuring adequate caloric intake, and may not be appropriate in some cases, such as critically

ill or severely malnourished patients. Enteral feeding is a more appropriate choice to ensure adequate nutrition in these patients. If caloric intake remains insufficient after 2–3 days of using an appetite stimulant (or maximum 3–5 days since cessation of food intake), further measures, including enteral feeding, must be considered.

Appetite stimulants used in cats:

1. Cyproheptadine

2. Diazepam

3. Megestrol acetate

4. Mirtazapine

5. Nandrolone

6. Oxazepam

7. Prednisolone

Antiemetics and Prokinetics:Vomiting in cats is a complex coordinated reflex, resulting in expulsion of gastric contents, coordinated by neurons distributed in the brainstem. Antiemetics are used to control or prevent vomiting through specific receptor interactions mediated either centrally or peripherally, making some more effective in cats than others.

1. Mirtazepam

2. Metoclopramide

3. Dolasetron and Ondansetron

4. Maropitant

5. Phenothiazines

6. Cisapride

Temping cats to eat:Where possible and appropriate, persuading a cat to eat is preferred over other feeding methods.Warming the food to just below body temperature may help encourage intake. If this is not successful, colder foods may be tried, as cats show variation in their preferences. Some cats will eat while being offered food by hand. Placing a small amount of wet food gently in the cat's mouth or on their paws may initiate feeding. A variety of foods may be offered and some cats only eat at night when the wards are quiet.

Conclusion:

This presentation will address enternal nutrition for critical care patients, along with clinical case examples focusing on pancreatitis and hypertrophic cardiomyopathy due to hyperthyroidism.

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NURSES (HILLS)

FLUID THERAPY - THE ESSENTIALS

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FLUID THERAPY THE ESSENTIALS

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Body water

The water content of the adult body is, on average, 60% by weight. However, the water content can vary with age and nutritional status. For example, the water content of an older animal may be as little as 50-55% whilst the water body content of young animals may be as high as 70-80%.

The body water content is also affected by the proportion of fat to lean tissue within the body. Fatty tissue contains much smaller amounts of water than other organs and tissue. This is an important consideration (to avoid overhydration) when devising a fluid therapy plan for the obese patient; the plan should be based on the patient's ideal weight not current weight.

Distribution of Body Water

- **Intracellular fluid ICF** Two thirds of the total body water is located inside the cells of tissues, this is called intracellular fluid

- **Extracellular fluid ECF** The remaining one third of the total body water is located outside the cells. This is further divided into:

- **Intravascular fluid** The water contained within the blood vessels

- **Interstitial fluid** The water present in the spaces between the cells (also bone, cartilage and dense tissue)

- **Transcellular fluids** Specialised fluids, e.g. cerebrospinal fluid, gastrointestinal secretions. However, this is only a very small amount of the extracellular fluid.

Water Balance

We need to be able to match the amount of water being lost by the body to the amount of water being taken in to the body to enable water balance. The normal water balance in a healthy dog or cat is generally calculated at 50ml per kg bodyweight per day. However, this ranges from 40-60ml/kg/day (24hrs).

This calculation is devised from the amount (on average) that a dog or cat will lose due to sensible and insensible losses. A sensible loss refers to fluid loss that an observer can readily detect with their senses (e.g. can see it coming out)

Source of fluid loss	Sensible / Insensible	Amount
Respiration / sweating	Insensible loss	20mls/kg/24hrs
Urinary loss (normal range)	Sensible loss	20mls/kg/24hrs
Faecal loss (normal range)	Sensible loss	10mls/kg/24hrs
Total fluid loss		50mls/kg/24hrs

Abnormal fluid loss

Causes of abnormal fluid loss include:

- · Vomiting

- · Diarrhoea

- · Blood loss

- · Anorexia

- · Evaporation (increased panting etc)

- · Disease

Goals of fluid therapy

The goals are to replace lost fluid and electrolytes and to maintain a state of normal fluid and electrolyte balance in the presence of inadequate intake and ongoing losses.

When formulating a fluid therapy plan, the following five questions should be considered.

- **indicated**? Consider the clinical indications, laboratory data and patient presentation that signify that fluid therapy is indicated

- **much** fluid should be given? Consider rehydration, maintenance and ongoing losses.

- **rate** should fluid be given? The rate is very much dependent upon patient presentation and amount of fluid that can be administered to rehydrate without fluid overload occurring. Sometimes a deficit will be replaced over 24 hours while in other cases a portion of the deficit will be replaced rapidly then the remainder of the deficit at a slower rate. The daily rate is calculated and then the hourly rate to ensure correct administration

- **route** should fluid be given? This has been previously discussed. In this section we are concentrating on intravenous fluid administration

- **type** of fluid should be given? Several types of intravenous fluids are available. Choice of fluid depends on the nature of the deficit of electrolytes and disease factors.

Assessing Dehydration

Each patient should be individually assessed for the level of dehydration they are suffering from and an appropriate fluid therapy plan devised. There are several clinical and laboratory methods of assessing dehydration. These include:

Patient history

A thorough history assists in correct assessment of the

type and amount of fluid deficit present. Areas to be considered include:

- **Patient eating and drinking status** It is important to establish if the patient has been eating and drinking normally. If the patient is anorexic or has had reduced food intake this should be noted. The drinking status should also be recorded; this may include lack or fluid intake or excessive fluid intake (polydipsia)

- **Gastrointestinal Losses** If the patient has had any vomiting or diarrhoea, the frequency, amount and consistency should be noted. When devising a fluid therapy plan any 'on-going' losses should be taken into account; vomiting and diarrhoea are an on-going loss until the condition is resolved.

- **Trauma** Haemorrhage should be duly noted and attended to, along with a history of when the trauma occurred, circumstances, patient status.

- **Urinary status** Notation of the patient's urinary status should be made. This includes reduced or increased urine output, colour and consistency of the urine.

- **Abnormal discharges** Any abnormal discharge should be recorded (for example, an open pyometra or weeping large sore). A history should be taken of how long the discharge has been present, amount and consistency of discharge.

Physical Examination

Although they are not always accurate, clinical signs are a useful means of assessing dehydration. It should be noted however that clinical signs will not become apparent until the animal is at least 5% dehydrated.

To assess hydration, feel the mucous membranes and perform the 'skin tenting' test. Mucous membranes should normally be moist. Dry, tacky mucosa is an indication that hydration is not adequate.

To perform 'skin tenting', gently lift the skin of the animal, twist and observe how long it takes before skin returns to its normal position.

Clinical sings of dehydration per percentage:

5%	Slight decrease in skin turgor, slightly tacky MM
5 – 8%	Delay in skin 'tent' to normal, tacky and dry MM, increased CRT, eyes may appear slightly sunken
10 – 12%	Tenting of skin, dry and tacky MM, increased CRT, tachycardia, sunken eyes, cold extremities
12 – 15%	Severe dehydration, clinical signs of shock are apparent, life threatening, hypothermic, weakness

Blood work may indicate increase in PCV, TPP and BUN

Routes of Administration

Oral

The oral route is the most physiological. This route should not be used in the presence of vomiting or if contraindicated due to illness or surgical procedure. This route is also inadequate for animals that have had acute or extensive fluid losses. Fluid absorption is not sufficiently rapid via the oral route for those cases where the fluid loss has been extensive and the blood flow is inadequate.

Subcutaneous

Fluids are usually administered in the subcutaneous tissues over the dorsal neck and cranial trunk. In the absence of vasoconstriction and/or hypovolemia the rate of absorption is approximately six to eight hours. Fluids should be administered at body temperature to decrease the discomfort to the patient and improve absorption. Only isotonic fluids should be administered by this route. Potassium supplementation up to 40 mmol/L may be added to the fluids. The rate and volume of administration will vary from patient to patient. Skin necrosis and infection are complications associated with this route of fluid administration.

When administering subcutaneous fluids try to massage the area being injected. This will help prevent formation of a lump. If large amounts of fluid are being administered subcutaneously, divide the total amount into smaller injections and change sites. E.g. if a 100mls is being given, give four separate injections of 25mls.

Intravenous

This is the route of choice when vascular volume restoration is desired. Fluid absorption is rapid. In addition to isotonic solutions, hyper- and hypotonic solutions may be administered via this route. The rate and volume administered will vary from patient to patient and be based upon the patient condition and desired end-point.

Intravenous catheter placement and fluids are discussed in more detail further in this text.

Intraosseous

Fluids are administered via the bone marrow. Like intravenous administration, fluid absorption is rapid. This route is indicated when it is difficult to gain venous access using standard techniques, for example neonate patients or patients with collapsed circulation.

The veins in the bone marrow drain into the systemic venous system and enable quick effective absorption of fluids

Sites for placement of an intraosseous catheter in the cat or dog include:

- Tibial crest

- Inter-trochanteric fossa of the femur

- Wing of ilium

- Tibial tuberosity

- Greater tubercule of the humerus

The sites for intraosseous placement in birds are:

- The distal ulna
- Proximal tibiotarsal bone

How Much Fluid?

Once we have assessed how dehydrated our patient is, we can calculate how much fluid is required to **replace (rehydrate)** the loss. We then look at how much is required to **maintain** normal hydration and finally if there are any **ongoing losses** (e.g. vomiting, diarrhoea, draining wounds etc) to take into account.

Types of fluids available will be discussed later in this section.

Rehydration

Replacement fluids are typically used for rehydration.

To calculate the amount of missing fluid (the amount that needs replacing) multiply the body weight in kg by the percentage dehydration. Remember that mathematically 5% is expressed as 5/100. This will give you the missing volume in litres. So for a 10kg animal which is 5% dehydrated: 10 X 5 / 100 = 0.5 litres (i.e. 500mls)

A quick rule-of-thumb formula to give you the result in millilitres is: percentage dehydration (just the number) X Body weight (kg) X 10 So for a 10kg animal which is 5% dehydrated: 5 X 10 X 10 = 500mls

Maintenance

The goal of maintenance fluid therapy is to replace fluid lost in normal body functions.

Maintenance fluid requirements = Insensible fluid losses + Sensible fluid losses

Insensible losses (GIT, Respiratory tract, Skin) = 20ml/kg/day

Sensible losses (urine) = 1-2ml/kg/hour (i.e. 24-48ml/kg/day)

Therefore, combined maintenance volume is generally estimated at 40-60ml/kg/day. Larger dogs typically have lower requirements than small dogs (due to surface area: body mass ratio).

Ongoing fluid losses

Ongoing losses refer to fluid losses over and above the maintenance requirements of a normal patient. Examples include vomiting, diarrhoea, bleeding and wound exudate.

These can be difficult to predict and so a best estimate is used.

Calculating Fluid Requirements

When calculating fluid requirements all three losses/deficits are considered

Requirement = fluid deficit **+** maintenance requirements **+** ongoing losses

If a fluid pump is used to deliver the fluids then it has the capacity to have an hourly fluid rate set. If a gravity fed administration system is used then the fluid rate should be calculated to drops per second. Conventional administration sets generally administer 20 drops/ml. It will be printed on the packaging.

Example Calculation:

10kg dog is 8% dehydrated

Has diarrhoea – estimate it will lose 140mls of fluid via D+ in next 24hrs

Maintenance choice of rate = 50ml/kg/day

Deficit + maintenance + ongoing losses

- 10 (kg) x 8 (% dehydration) ÷ 100 = 800mls (replacement amount)

- 10 x 50 (mls/kg/day) = 500mls (maintenance amount)

- 140mls over 24hrs = 140mls (ongoing losses)

- 800 + 500 + 140 = 1440mls

- 1440mls ÷ 24 hrs = 60mls per hour

Calculate drops per minute

- Hourly rate (mls/hr) ÷ 60 (mins/hr) x 20 (drops/ml – giving set factor)

- 60 ÷ 60 x 20 = 20 drops per minute

- Calculate drops per second

- Minute rate ÷ 60 (secs/min) = 20 ÷ 60 = 0.33

- Convert decimal value to fraction – 0.33 = 1/3rd

- **1 drop every 3 seconds**

Type of Fluid Required

The type of fluid given will depend on many factors including:
- Composition of lost fluid
- Abnormalities requiring correction
- Severity and type of fluid depletion

Considerations of choice of fluid should also include:
- Tonicity
- Need for glucose
- Electrolyte balance
- Acidity
- Osmotic pressure
- Oxygen carrying capacity

Fluids can be divided into three main categories
- Crystalloids

- Colloids
- Blood and blood products

Crystalloids

Crystalloids are water-based solutions that contain small particles (ions and molecules). Common constituents include electrolyes (e.g. sodium, potassium, calcium, magnesium and chloride to replace or maintain plasma levels), glucose or dextrose (to provide energy) and sometimes a buffer (to correct or maintain the normal acid-base balance of the plasma).

These small particles readily pass through membranes and into all body compartments. The fluid is rapidly redistributed through the body. Generally, 75% of crystalloid fluids given will leave the intravascular space within 30 minutes of administration.

Crystalloid solutions can be:
- Isotonic
- Hypotonic
- Hypertonic

Colloids

Colloids contain large molecules that do not readily pass through membranes. Fluid is drawn into blood vessels.

Colloid solutions refer to solutions that have an osmolality greater than the extracellular space; they contain large molecules which are designed to stay in the intravenous space longer than crystalloids. They are sometimes referred to as 'plasma expanders' due to their ability to draw fluid out of the interstitial and intracellular spaces into plasma and thus maintain vascular volume more effectively.

Much smaller volumes of colloids are just as effective as large amounts of crystalloids.

Their main indications for use are:
- Hypovolaemic shock
- Hypoproteinaemia

They are often administered as a bolus (supported with crystalloid fluids) or over several hours.

Examples of Synthetic colloids include:

Voluven 6%

Voluven 6% contains 6% hydroxyethyl starch in 0.9% NaCl. Its main use is as a plasma volume replacement. It does NOT replace red blood cells or clotting factors. It is a clear colourless solution. It should be stored below 25°C and should not be frozen.

Contraindications
- Do not use in patients with pre-existing disorders of haemostasis and coagulation
- Do not use in patients with established renal failure

Side-effects
- Minor allergic reactions due to histamine release
- Transient increase in bleeding time may occur
- Hypersensitivity reactions may occur including, rarely, severe anaphylactic reactions

Voluven dose rates (as per manufacturers recommendations):
- Up to 50ml/kg/day – maximum due to coagulation problems if larger amounts given
- Continuous infusion can be calculated at 1-2 ml/kg/hr although not recommended to use as a CRI now due to adverse renal effects

Or
- Larger bolus over shorter period can be given at 20-40ml/kg per day (so larger amount given over 2 hrs instead of smaller amount of 8 hrs but must not exceed 50ml/kg/day)

Monitoring of and Care of Intravenous Catheters

It must be remembered that intravenous catheters can be a port of entry for bacteria into the vein. The catheter and the dressing must be checked regularly.

Intravenous catheter checks should be performed frequently (at least four times a day) and includes:
- Remove and replace immediately if contaminated in any way (dirty, wet, chewed etc).
- Check for swelling of the distal paw
- Check for swelling or heat proximal to the bandage
- Ensure catheter is not leaking

At least once a day the following check should be employed:
- Remove the catheter bandage
- Visualise the insertion site of the catheter and check for:
- Any redness
- Any heat or swelling
- Any discharge (note colour and amount)
- Catheter dislodgement
- Adhesive tape or padding dislodgement
- Pain on palpation of area

Re-bandage and note catheter check performed and findings

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DENTAL EXTRACTIONS MADE EASIER

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DENTAL EXTRACTIONS MADE EASIER

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Extractions are surgery, and therefore need to be treated with appropriate respect. Patience and gentle technique is the best way to achieve a successful outcome. All extractions can be broken down into simple, single rooted extractions. Therefore proper elevation and extraction techniques learned and performed on incisor teeth will make all extractions easier.

Proper and well maintained equipment is critical for successful extractions. This author prefers luxating elevators to standard or winged elevators. Small extraction forceps and needle holders will also benefit the surgeon. Step 1: **OBTAIN CONSENT**

NEVER extract teeth without owner consent (preferably written), no matter how bad the problem, or how obvious the decision is. Make sure that you have a valid daytime number (or numbers) for the client and inform them they must be available during surgery hours. Consider loaning pagers to clients for the day, as this author has found this to be a very effective means to contact clients. If the client cannot be reached and prior consent was not obtained, DO NOT PULL THE TOOTH. Document the problem, recover the patient, and reschedule the work. Remember, the tooth can always be extracted later, but it cannot be put back in!

Step 2: **DENTAL RADIOGRAPHS**

Dental radiographs should be exposed on all teeth prior to extraction. Dental radiographs are invaluable resources for the practicioner. Radiographs allow the practitioner to determine the amount of disease present, any root abnormalities or ankylosis. Help with radiographic interpretation is available while the patient is under anesthesia at www.vetdentalrad.com. In addition, the radiographs will serve as evidence for the extraction in the medical record. Radiographs should also be exposed post-extraction to document complete removal of the tooth.

Step 3: **OBTAIN PROPER VISABILITY AND ACCESSABILITY**

The patient should be positioned in such a way as to allow maximum visibility of the area as well as make the surgeon most comfortable. Note that during the extraction procedure the ideal position may change and the patient should be adjusted appropriately. The lighting should be bright and focusable on the surgical field. Suction, air/water syringes, and gauze should be utilized continually to keep the surgical field clear, and mouth gags can be used to hold the mouth in proper position for surgery. Finally, magnification may help the surgeon locate furcations or retained root tips.

Step 4: **PAIN MANAGEMENT**

Extractions are surgical procedures and are moderately to severely painful for the patient. Depending on patient health, a multimodal approach (combination of opioids, NSAIDs, local anesthetics, and dissociative) should be employed, as this provides superior analgesia. Preemptive analgesia is proven to be more effective than post-operative, and it is therefore important to administer the drugs BEFORE the painful procedure. **(see notes in previous lecture for pain mamagement)**

SINGLE ROOT EXTRACTIONS

Step 5: **INCISE THE GINGIVAL ATTACHMENT**

This is accomplished with a scalpel blade (number 11 or 15), elevator, or luxator. The selected instrument is placed into the gingival sulcus with the tip of the blade angled toward the tooth (this will help avoid going outside the bone and creating a defect or cutting through the gingiva). The blade is then advanced apically to the level of the alveolar bone, and the instrument is carefully worked around the entire tooth circumference.

This step is very helpful as the gingival attachment contributes approximately15% of the retentive strength of the periodontal apparatus. More importantly, however, this procedure will keep the gingiva from tearing during the extraction procedure. This is most important with mobile teeth where little elevation is needed, but one edge is still attached. Gingival tearing can cause defects that require closure or can make a planned closure more difficult.

Step 6: **ELEVATE THE TOOTH**

Elevation is the most dangerous step in the extraction procedure. Remember that you are holding a sharp surgical instrument and working in an area of numerous critical and delicate structures. There have been many reports of eyes that have been gouged and lost by extraction instruments as well as at least one confirmed fatality due to an elevator puncturing a patient's brain. The index finger is placed near the tip of the instrument to avoid causing iatrogenic trauma in the event of instrument slippage or encountering diseased bone. In addition, the jaw should be gently held with the opposite hand to provide stability and avoid mandibular fracture.

First, select an instrument which matches the curvature and size of the root. There are numerous instruments available including the classic elevator, the luxating elevator, and the winged elevators. Classic elevators and winged elevators are used in an "insert and twist" motion to tear the periodontal ligament, whereas luxators are used in a rocking motion during insertion to fatigue as well as cut the periodontal ligament. Luxators can be GENTLY twisted for elevation, but they are not designed for this and can be easily damaged when used in this manner.

Elevation is initiated by inserting the elevator or luxator firmly yet gently into the periodontal space. The insertion should be performed while keeping the instrument at about a 10 to 20 degree angle toward the tooth, to avoid slippage. Once in the space between the bone and the tooth, the instrument is **gently** twisted with two-finger pressure. This is not to say that the instrument should be held with two fingers, rather the entire hand should be used to hold the instrument. Twist only with the force that you could generate when holding with two fingers. Hold the position for 10-30 seconds to fatigue and tear the periodontal ligament.

It is important to note that the periodontal ligament is very effective in resisting intense, short forces. It is only by the exertion of prolonged force (i.e. 10-30 seconds) that the ligament will become weakened. Heavy stresses only serve to put pressure on the alveolar bone and tooth which can result in the fracture of one of these structures, so it is important not to use too much force.

After holding for 10 to 30 seconds, reposition the instrument about 1/8 of the way around the tooth and repeat the above step. Continue this procedure 360 degrees around the tooth, each time moving the elevator apically as much as possible. Depending on the level of disease and the size of the tooth, a few to several rotations of the tooth may be necessary. The key point to successful elevation is PATIENCE. Only by slow, consistent elevation will the root loosen without breaking. It is always easier to extract an intact root than to remove fractured root tips.

Step 7: **EXTRACT THE TOOTH**:

Removing the tooth should only be attempted after the tooth is very mobile and loose. This is accomplished by grasping the tooth with the extraction forceps and gently pulling the tooth from the socket. Do NOT apply undue pressure as this may result in root fracture. In many cases, especially with premolars, the roots are round in shape and will respond favorably to **gentle** twisting and holding of the tooth while applying traction. This should not be performed if there are root abnormalities (significant curves, weakening) seen on the pre-operative radiograph. It is helpful to think of the extraction forceps as an extension of your fingers. Undue pressure should

not be applied. If the tooth does not come out easily, more elevation is necessary. Start elevation again until the tooth is loose enough to be easily removed from the alveolus.

Step 8: **AVELOPLASTY**

This step is performed to remove diseased tissue or bone, as well as rough boney edges that could irritate the gingiva and delay healing. Diseased tissue can be removed by hand with a curette. Bone removal and smoothing is best performed with a carbide, or preferably a coarse diamond bur on a water-cooled high-speed air driven hand-piece. Alternatively, ronguers or bone files may be used if a high-speed dental unit is unavailable. Next, the alveolus should be gently flushed with a 0.12% chlorhexidine solution to decrease bacterial contamination. After the alveolus is cleaned, it may be packed with an osseopromotive substance.

Step 9: **CLOSURE OF THE EXTRACTION SITE**:

This is a controversial subject among veterinary dentists, and thus some texts recommend suturing only in large extractions, other authors (including this one) recommend suturing almost all extraction sites. Closure of the extraction site promotes hemostasis and improve post-operative discomfort and aesthetics. It is always indicated in cases of larger teeth (e.g. canines, carnassials), or any time that a gingival flap is created to allow for easier extraction. This is best accomplished with size 3/0 to 5/0 absorbable sutures on a reverse cutting needle. Closure is performed with a simple interrupted pattern with sutures placed 2 to 3 mm apart. It is further recommended to utilize one additional throw over manufacturer's recommendations to counteract tongue action.

In regards to flap closure, there are several key points associated with successful healing. The first and most important is that there must be no tension on the incision line. If there is any tension on the suture line, it will not heal. Tension can be removed by extending the gingival incision along the arcade (called an envelope flap) or by creating vertical releasing incisions and fenestrating the periosteum. The periosteum is a very thin fibrous tissue which attaches the buccal mucosa to the underlying bone. Since it is fibrotic, it is inflexible and will interfere with the ability to close the defect without tension. The buccal mucosa is very flexible and therefore will stretch to cover large defects. If there is no tension, the flap should stay in position without sutures.

If at all possible, the suture line should not be made over a void. If sufficient tissue is present, consider removing some on the attached side to make the suture line over bone. Always suture from the unattached (flap side) to the attached tissue, because this avoids tearing the flap as the needle dulls. Finally, ensure that all tissue edges have been thoroughly debrided as intact epithelial

tissues will not heal.

EXTRACTION OF MULTI ROOTED TEETH:

Section all multi-rooted teeth into single rooted pieces. The roots of almost all multi-rooted teeth are divergent and this will cause the root tips to break off if extractions are attempted in one piece. Root fracture can occur even if a tooth is relatively mobile to start with. With mobile teeth, the sectioning step alone often allows for simple extraction.

The best tool for sectioning teeth is a bur on a high-speed air driven hand piece. Besides being the quickest and most efficient tool for the job, it also has air and water coolant that will avoid overheating the tooth. Many different styles of burs are available, however this author prefers a cross-cut taper fissure bur (699 for cats and small dogs, 701 for medium dogs and 702 for large breeds).

The best way to section the teeth is to start at the furcation and work towards the crown of the tooth. This method is used for two major reasons. First, it avoids the possibility of missing the furcation and cutting down into a root, which subsequently weakens the root and increases the risk of root fracture. In addition, this method avoids the possibility of cutting through the tooth and inadvertently damaging the gingiva or alveolar bone.

After the tooth has been properly sectioned, follow the above steps for each single rooted piece. In some cases, the individual tooth pieces can be carefully elevated against each other to gain purchase.

Key Points:

- Proper preparation and mindset are crucial

- Small/sharp instruments are the most effective choice

- Gentle elevation and **patience** are necessary for successful outcomes

- All teeth can be broke down into simple single root extractions

- Dental radiographs are an invaluable resource

WSV18-0106

SVA PRACTICE MANAGEMENT

LEARNING FROM THE BEST - LESSONS FROM OTHER BUSINESSES TO TRANSFORM YOUR CLINIC

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LEARNING FROM THE BEST – LESSONS FROM OTHER BUSINESSES TO TRANSFORM YOUR CLINIC

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Market forces combine to create innovation, which in turn can occasionally lead to a business making such a profound transformation that their business sector is irreversibly changed. Many such examples exist, and in this session I look at some of those that have a particular relevance to the veterinary profession and identify the practical lessons that we can draw from the success of these 21st Century market leaders to help us to transform our own veterinary clinics

Virgin Airlines

What Virgin did well was to make the customer experience whilst using their primary service as comfortable and pleasurable as possible. They identified by speaking to long haul flyers what is was that frustrated them most about flying, and set about designing a service that addressed their needs. They introduced a whole range of service innovations that we take for granted when travelling today, such as Limousine service from home to airport, kerbside check in, airport lounges, and on board entertainment. They have also continued to innovate.

So, it we ask our own clients today, what issues do they have in our clinics today? Clients will tell us that they are often kept waiting on uncomfortable chairs, or that their pet was stressed by the close presence of other species, that they were anxious that their children didn't touch things, that their phone calls were unanswered, that lab results were not phoned through, etc. etc. What would your clients say, and what could you change in your clinic that would address their concerns.

Easyjet

What Easyjet did well was to improve the utilisation of their key equipment and resources to reduce costs. Veterinary clinics increasingly have to invest in ever

more expensive technology in order to provide high quality clinical care, yet much of it sits unused for long periods. When deciding how much to charge for a service we look at the cost of providing that service and this includes amortisation of the capital or leasing costs across each service, such that the more we use an asset the less it costs each time we use it. Underused assets increase the charge we have to make to clients, or if we cannot pass on that cost, they reduce future profits.

So, can we either reorganise the way we use our key assets to get more out of them, such as grouping services into a time slot; or can we market our services in a way that will increase the use of key assets, reducing the cost, such as offering discounts.

McDonalds

What McDonalds did well was to systemise their process to achieve consistency of service at the lowest cost. They did this by breaking the service delivery down into steps and introducing controls such that these steps could be performed by less highly trained staff (chefs) reducing labour costs and training times. In our clinics our most valuable staff members are our veterinary surgeons, yet much of their time is spent on tasks that don't require their specialist skills. How can we organise ourselves so that vets on do the things that only vets can do! Developing appropriate best practice protocols and procedures will let us identify tasks that can be performed by less qualified staff, and provide the process control to ensure that they are delivered correctly.

Disney

What Disney did well was to motivate their "Cast members" to deliver exceptional service to their "Guests" at all times. In fact they went beyond that, they taught their staff how to anticipate their guest's needs and to provide this, often even before the guest realises their need for themselves. The result is an organisation that delivers service that often delights and surprises guests, and which creates the "Disney Experience"

How often do our staff fail to anticipate our client's needs, or worse, are so focused on the needs of the patient that they forget the client altogether? Too often I find reception teams who see their principal role as protecting the vets from the clients, or who provide highly inconsistent service depending on whether or not the client is known to them. All too often this comes about because staff have bee poorly trained, and failures are quickly excused and forgotten. Your values should drive your client's experience and performance should be regularly reviewed and changes made as appropriate.

GE

What GE did well was to recognise the value of people to a knowledge based business. As a result they developed their people management processes to be at the heart of what they do. The first item on any review of the business is the staff. How well do they meet the needs of the business today, how equipped are they to meet the challenges of tomorrow?

They recognise the value of their top people, and also the contribution of the majority, who just do a good job. They also have processes to deal with those who do not come up to standard, or make the changes necessary to adapt as the business develops.

Without even basis staff development tools many practices find that as they evolve they can outgrow long serving staff members, who have not adapted to change. In the majority of cases this is often because the development and support that they needed was not available, but they are now seen as intransigent. Our people development systems don't need to be sophisticated, but they do need to exist.

WSV18-0321

COMMINUTED FRACTURES: NEW STRATEGIES THAT SIMPLIFY TREATMENT OF THESE CHALLENGING FRACTURES

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Comminuted Fractures New Strategies that Simplify Treatment of this Challenging Fractures

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Comminuted fractures can be especially challenging due to the complexity of the fracture fragments and concomitant soft tissue injury. Careful consideration should be given to decision-making prior to onset of fracture repair. Factors that should be considered include mechanical, biological and postoperative compliance. Complex fractures that are treated with a mechanically sound repair often leave the surgeon pondering what could have possibly gone wrong when a "perfect" repair fails. Often times, the answer lies in the neglect of the biological or postoperative compliance factors. Neurologic function should always be assessed because complex fractures are often associated with high-energy trauma that also can injure the brachial plexus or peripheral nerves of the forelimb. This lecture will focus on presentation of clinical cases involving complex fractures of the forelimb and hindlimb, with an emphasis on the decision-making process. A variety of fracture repair techniques will be discussed including interlocking nails, plate-rod construct and linear external fixators.

Minimally-invasive surgical approaches reduce pain and minimize trauma to the soft tissues. Biological factors important for fracture healing are preserved, enhancing the body's ability for indirect bone healing. The technique can be used with all fracture types, but is particularly useful for stabilization of comminuted fractures. This type of bone healing is also referred to as secondary bone healing, spontaneous bone healing and callus healing. Stabilization of fractures using the principles of biologic fracture management is performed with the same type of implant systems used with traditional fracture repair, including externally and internally applied devices.

Fracture Management

Comminuted fractures of the extremities can be challenging. It is always a race between a fracture healing and an implant failing. Steps can be taken to tip the scale in the direction of early fracture healing. These steps include:

1. Minimally invasive surgical approach
2. Preservation of soft tissue attachments to bone fragments
3. Use of cancellous bone grafts
4. Rigid method of fracture stabilization
5. Early return to function

It is always important to obtain an accurate history prior to stabilizing fractures. A complete physical exam and appropriate diagnostic tests should be performed. Pathologic fractures are more likely to be seen in the geriatric dog and cat and should be identified preoperatively to ensure proper client education and communication

Indirect Bone Healing

Biological fracture management utilizes indirect fracture reduction to preserve the soft tissue envelope at the expense of anatomic reduction. Indirect bone healing occurs as a result. Indirect bone healing consists of three elements: first the formation of granulation tissue at the fracture site, second is fracture gap widening due to resorption of the bone ends and third is the new bone formation involving generation of a bone callus. Less disruption of the vascular supply to bone fragments is achieved through minimal handling of the fragments, promoting early callus formation.[2,3,6,7] Indirect bone healing is first associated with the formation of fibrous connective tissue and cartilage callus between the fragments.[4] Indirect bone healing occurs due to instability at the fracture site and is partially regulated by fragment gap strain.[4] Interfragmentary strain is a ratio of change in the gap width to the total width prior to physiological loading.[1,5] A study of the "interfragmentary strain hypothesis" using ovine osteotomy models demonstrated that the initial stages of indirect bone healing occur earlier and more extensively between gaps with lower shear strain.[1] Management of a non-reducible diaphyseal fracture with an implant system that does not utilize anatomical reconstruction and creation of subsequent small fracture gaps avoids high interfragmentary strain, favouring bone healing.

Implant Systems

External and internal implant systems can be used to achieve bone healing using biological fracture management. Examples of external devices when used in an appropriate manner include casts, splints, linear external fixators and circular fixators. Internal devices commonly used for this application include the plate-rod system, interlocking nail and bone plates. Other implant systems can also be used for biologic fracture management as long as the soft tissue envelope is preserved at the fracture site. Whatever implant system is used, its application must be possible with minimal or no handling of the comminuted fracture fragments.

External Fixator

External fixators provide rigid stabilization and can be

used with minimally-invasive technique. Many fractures of the radius and tibia can be reduced closed and stabilized with an external fixator. The main disadvantage is the potential for complications with premature pin loosening and the added care needed in the postoperative period. The use of external fixators for fracture repair is not optimal if the patient or owner is likely to have poor compliance in the postoperative period. External fixators frames can be applied in one of 3 configurations- linear, circular or as a hybrid of linear and circular.

Plate-rod construct

The plate rod system has been found to be an ideal implant system for biological fracture management. Management of a non-reducible diaphyseal fracture with a combination of an IM Steinmann pin and bone plate can be applied without anatomical reconstruction and thus, avoids the development of small fracture gaps with high interfragmentary strain. The addition of the IM pin to the plate also significantly increases the construct stiffness and estimated number of cycles to fatigue failure when compared to a plate only construct. An IM pin serves to replace any trans-cortical defect in the bone column and acts in concert with the eccentrically positioned plate to resist bending.[2] Mathematical analysis of the plate-rod construct in the canine femur demonstrated that the pin and plate act most like a dual-beam structure, assuming slight motion of the pin in the canal.[2] Addition of an IM pin to a bone plate has been shown by Hulse et al. to decrease strain on the plate two-fold and subsequently increase the fatigue life of the plate-rod construct ten-fold compared to that of the plate alone.[1] In the canine femur, plate strain is reduced by approximately 19%, 44%, and 61% with the addition of an IM pin occupying 30%, 40% and 50% of the marrow cavity, respectively.[3] Stiffness of plate-rod repairs may be as much as 40% and 78% greater when the pin occupies 40% and 50% of the marrow cavity, respectively.[2] Ideal diameter of the IM pin should be between 30 and 40% of the medullary canal diameter measured at the istmus. Increasing diameter up to 50 dramatically challenge the ability to insert screws trough the plate holes.

Locking Plates

Locking plates have become very popular for minimally-invasive fracture repair. Many locking plate systems are available including the Synthes, FIXIN, SOP and ALPS. Locking plates have the ability to lock the screw into the hole of the plate. The mechanism for locking varies amongst manufactures. The Italian design FIXIN locking plate system has a conical locking mechanism while the Synthes system has a threaded locking mechanism. The FIXIN plate hole is tapered to match the conical nature of the head of the screw. This type of fitting is similar to the Morse taper of the head and neck fitting of the Total Hip Replacement implant. The stability of this design is extremely secure. The Synthes locking plate has threaded holes in the hole of the plate. Corresponding threads in the head of the screw engage the threads of

the hole, locking the screw to the plate. The ability to lock the screw to the plate increases pull-out strength of the screw and construct stability. Traditional plates do not have threaded holes. Screws placed in ordinary plates apply pressure to the plate, pressing it onto the bone surface. The friction between the plate and the bone provides the stability to the bone-implant construct. In contrast, the locking plate achieves stability through the concept of a fixed-angle construct. The locking plate is not pressed firmly against the bone as the screws are tightened. The locking screws and plate function more like an external fixator. Locking plates are essential "internal fixators." The plate functions as a connecting bar and the screw functions as a threaded fixator pin. The tapered or threaded head of the locking screw engages the hole of the plate, similar to the clamp of an external fixator. The Synthes locking plate also has combi-holes which allow use of traditional or locking screws when desired. Traditional screws should be place prior to locking screw when using locking plates.

Locking plates are ideal for minimally-invasive fracture repair for several reasons. Blood supply to the bone is preserved because the plate is not pressed tightly against the bone. The plate does not require perfect anatomic contouring because the displacement of the plate will not occur as the screw is tightened into the hole of the plate. Accurate contouring is difficult with a minimally-invasive approach due to the minimal exposure to the shaft of the bone. Lastly, locking screws give fixed angle support to the non-reduced fracture, increasing stability and less chance of collapse and instability at the fracture gap.

Interlocking nail

The Deuland interlocking nail system presently available in the U.S. (Innovative Animal Products, Inc., Rochester, MN) is a modified Steinmann pin modified by drilling one or two holes proximally and distally in the pin, which allows the placement of transverse bolts or screws through the bone and nail. The nail, bolts and screws can be applied in closed or open fashion due to the incorporation of a specific guide system that attaches to the nail. The equipment needed to place the nail includes a hand chuck, extension device, aiming device, drill sleeve, drill guide, tap guide, drill bit, tap, depth gauge, and screwdriver. Cost of the system is reasonable and each nail is approximately half the cost of a comparative bone plate. The nails are available in diameters of 4.0, 4.7, 6, 8 and 10 mm and varying lengths and hole configurations. The 4.0 and 4.7 mm nails use 2.0 mm screws or bolts. The 6 mm nail is available in two models and will accommodate either 2.7 or 3.5 mm screws or bolts. The 8 mm nail is also available in two models and will accommodate either 3.5 or 4.5 mm screws or bolts. The 10 mm nail uses 4.5mm screws or bolts. The solid cross locking bolts have a larger

diameter compared to a similar diameter screw, thus are less likely to break. Bolts also provide superior mechanical behavior compared to screws.

The interlocking nail is placed along the mechanical axis of the bone. The interlocking nail neutralizes bending, rotational and axial compressive forces due to incorporation of transfixation bolts or screws which pass through the pin and lock into the bone. This is in contrast to a single intramedullary Steinmann pin which is only effective in neutralization of bending forces. The interlocking nail has a similar bending strength compared to bone plates, but is slightly weaker in neutralization of torsional forces. The screws also prevent pin migration, a common complication seen with Steinmann pins.

When using an interlocking nail, the largest diameter nail should be selected that can be accommodated by the medullary cavity at the fracture site. In most large dogs, an 8 mm nail and either 3.5 or 4.5mm screws or bolts can be used in the femur and humerus. In medium-sized dogs, the 6 mm nail and either 2.7 or 3.5 mm screws or bolts are typically used. In small dogs and cats, the 4.7 mm nail and 2.0 mm screws are typically used. The tibia of medium and large - sized dogs will usually accommodate a 6 mm nail, but some large dogs will accept an 8 mm nail. Small dogs and some cats will accept a 4.0 mm nail for repair of tibial fractures. Dejardin et. al. have developed a novel interlocking nail that provides an angle stable locking mechanism. The advantage of angle stable locking is the elimination of torsional and bending slack, resulting in reduced interfragmentary motion. This interlocking nail system provided comparable mechanical performance to a plate system. Dejardin's nail is currently available.

Surgical Approach

Closed reduction and stabilization is the optimal method of treatment when possible. Unfortunately, this method is rarely possible in the senior patient due to the severity of fractures seen, long time until bony union, and the tendency for patients to develop bandage sores. Open surgical approaches can be either traditional or minimally invasive. The minimally invasive approach has also been described as an "open but don't touch" approach. The acronym, OBDT, is used to describe this technique. The advantages to using an OBDT technique is preservation of vascular supply to the fracture site and thus quicker healing, shorter intraoperative time, less postoperative pain and early return to function. Methods of stabilization that work well with an OBDT approach include the interlocking nail, plate-rod hybrid and external fixation. The key feature of a minimally-invasive approach is the preservation of the soft tissue envelope at the fracture site. Small comminuted fragments will become quickly incorporated into the bony callus if left with a vascular pedicle. Anatomic reduction of small fragments is difficult if vascular supply to the fragment is to remain uncompromised.

Bone Grafts

Numerous sites for harvest of cancellous bone graft have been described in the dog, but the most practical are the greater tubercle of the humerus, wing of the ilium and the medial, proximal tibia. The humerus provides the greatest amount of cancellous bone, but the ilium and tibia provide sufficient amounts for most applications. All of these sites are readily accessible, have easily recognizable landmarks, have little soft tissue covering, and provide relatively large amounts of cancellous bone. The greater trochanter can also be used if other sites are not available; however, the yield of cancellous bone is markedly less. Occasionally multiple sites are required to harvest sufficient quantities of bone to fill large bone defects or during arthrodesis.

Minimal instrumentation is required for harvest of cancellous bone graft. Basic surgical instruments are used to approach the site selected for harvest. A hole is drilled through the near cortex using either a drill bit, trephine or trocar-pointed pin. A curette is used to scoop the graft out of the metaphyseal cancellous bone. The cancellous bone should be scooped out in large clumps if possible. Use a curette that can be comfortably manipulated in the medullary cavity; I prefer to use a relatively large curette as this speeds harvest and reduces trauma to the graft. Closure is performed routinely in 2-3 layers. Recently, a technique was described using an acetabular reamer to harvest large amounts of cortico-cancellous bone graft from the lateral surface of the wing of the ilium.

The graft collected should be handled gently. It is desirable to collect the graft immediately prior to usage. This increases the osteogenic properties of the graft. As graft is harvested, it should be placed on a blood-soaked gauze until transfer to the recipient site. Extreme care should be taken to store the graft properly; do not accidentally discard the graft due to misidentification of the gauze as being used. The graft should be atraumatically packed into the recipient site. Lavage of the site should be avoided after the graft is placed.

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WSV18-0125

VECCS

THE ADVERSE EFFECTS OF RAPID FLUID ADMINISTRATION.

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In the setting of shock, rapid fluid administration is often life-saving. Isotonic crystalloids remain the first choice in the majority of patients, with the least amount of evidence for causing harm. However, there is growing discussion that the administration of large volumes of crystalloid fluid may cause harm, especially for patients with systemic inflammation and increased vascular permeability. This talk will outline the potential adverse effects of large volumes of crystalloid, including those related to the constituents of the fluid (chloride, acetate), wash-out of the endothelial glycocalyx, and pro-inflammatory responses.

Constituents of isotonic crystalloids

The adverse effects of using 0.9% saline are probably the most well-recognised detriment of using high volumes of crystalloid fluid. The two main concerns with using 0.9% saline for blood volume expansion are dilutional acidosis and acute kidney injury. Dilutional acidosis is created by delivery of excessive chloride and subsequent dilution of bicarbonate, an effect that has been documented in dogs with haemorrhagic shock.(1) However, in this study, the change in blood pH was small and self-limiting. Still, if a patient is already severely acidaemic, reducing the blood pH further may have adverse consequences. The second concern is increasing the risk of acute kidney injury; which has been raised as a potential in human medicine.(2) The theory is that delivery of large amounts of chloride to the macula densa may reduce much-needed renin-angiotensin-aldosterone system activation in times of need (i.e. reduced renal perfusion). It is unknown if this effect is clinically relevant for veterinary medicine but suffice to say, 0.9% saline can be easily avoided and should only be used in alkalaemic patients.

The remaining choices for isotonic crystalloid administration belong in the group of 'balanced' fluids, which avoid excessive chloride by providing other anions such as lactate, gluconate and acetate. The most commonly used fluid is lactated Ringer's solution or compound sodium lactate. The provision of lactate does not contribute to lactic acidosis but provides a substrate for bicarbonate production. However, in a patient that can't metabolise lactate due to liver dysfunction, this fluid may cause mild hyperlactataemia. This is only inconvenient in the sense that it is difficult to then use lactate as a marker of perfusion. The remaining

choices are fluids containing combinations of acetate and gluconate, such as Plasmalyte-148. These anions are metabolised by the kidneys and skeletal muscle, providing a source of buffer. One possible adverse effect of injecting acetate rapidly is vasodilation,(3) which may momentarily worsen perfusion. Also, acetate has been shown in rats to be somewhat pro-inflammatory when used for fluid resuscitation.(4, 5) Therefore, if a choice can be made between a balanced crystalloid with either lactate or acetate, then the fluid with lactate is probably the better choice.

Shedding of the endothelial glycocalyx

The endothelial glycocalyx (EG) is a carbohydrate-rich scaffold of proteoglycans and glycosaminoglycans on the luminal surface of endothelial cells (Figure 1). The meshwork of carbohydrates and proteins creates an immobile plasma layer and barrier, preventing interaction between cells in circulation and endothelial cells. This prevents adhesion of platelets and white blood cells, and discourages thrombosis.

Figure 1 – The main components of the endothelial glycocalyx
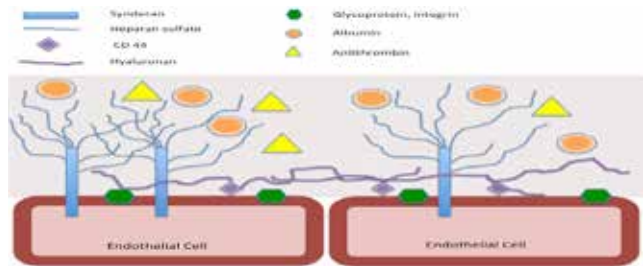
The EG also plays an important role in fluid flux across the endothelium, which has led to a revision of the traditional Starling's forces.(6) Albumin molecules within the EG layer provides some oncotic pressure, and it is the oncotic balance between the intravascular (flowing) compartment and the EG compartment that affects fluid flux according to protein levels. This explains why raising oncotic pressure in the intravascular compartment does not actually draw fluid from the interstitial space, much to the disappointment of clinicians attempting to reduce peripheral oedema.

It is likely that shedding of the EG is one of the first steps in inflammation and coagulation, which then allows interactions between endothelial cells and circulating leucocytes and platelets.(7-11) There is some evidence that rapid intravenous crystalloid fluid therapy, without coinciding injury or inflammation, can increase EG shedding.(12) It is unknown if this response to blood volume expansion is related shear stress, release of atrial natriuretic peptide, dilution of EG constituents, or a combination of these factors. In studies using euvolaemic models, it is difficult to separate the effects

of hypervolaemia from the effects of the fluid infusion itself and the scenario is less applicable to emergency medicine. Several rodent haemorrhagic shock models comparing large volumes of isotonic crystalloids to plasma or albumin infusions have demonstrated that large-volume crystalloids are associated with greater glycocalyx shedding, vascular permeability and reduced glycocalyx thickness. Preliminary work conducted by our group found that dogs given Plasmalyte-148 for haemorrhagic shock showed evidence for more glycocalyx shedding and inflammation, compared to those given synthetic colloid fluids or whole blood. (13) These types of studies raise cause for concern that large volumes of crystalloid, rapidly administered, may not be innocuous as previously thought, and may be contributing to a pro-inflammatory states that our patients may already be experiencing. Ongoing work is being conducted in this area, especially in the area of sepsis,(14) where systemic inflammation and vascular dysfunction may make patients particularly sensitive to further EG shedding.

Are synthetic colloid fluids safer?

Some may be tempted to use synthetic colloid fluid to avoid the administration of large volumes, which may reduce degree of shear stress and atrial distension (therefore avoiding release of natriuretic peptides). However, in order to achieve the same degree of blood volume expansion, the blood will still be diluted. Therefore, there may still be some adverse effect on the endothelial glycocalyx. Also the choice of diluent for synthetic colloids may be limited; it is most commonly suspended in 0.9% NaCl, which is not ideal for the reasons discussed above.

Additional adverse effects of synthetic colloids include interference with haemostasis, either due to platelet dysfunction or interference with secondary coagulation. Therefore, in a patient with active bleeding, this may not be the best choice. Also, an association between synthetic colloid fluids and acute kidney injury is currently being investigated.

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WSV18-0122

HOW I TREAT FRACTURES USING MINIMALLY INVASIVE TECHNIQUES

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WHY MINIMALLY INVASIVE FRACTURE TREATMENT?

The primary goal of all fracture treatment is restoration of normal limb function and we must never lose this focus. Surgical repair of fractures can be performed with open reduction & fixation (ORF) or minimally invasive osteosynthesis (MIO). When properly performed on carefully selected patients, MIO can restore normal limb function, but often with faster bone healing times and with less perioperative patient morbidity than associated with ORF. Morbidity concerns such as postoperative swelling, pain and incisional complications can often be reduced by virtue of the less aggressive surgical approaches.

WHY NOT MINIMALLY INVASIVE STABILIZATION OF ALL FRACTURES?

Just as MIO has inherent advantages, it also has some disadvantages that must be respected in case selection. First, there is a significant learning curve associated with the development of these skills. Most veterinarians are accustomed to visualization of the fractured bone for implant insertion, implant contouring, and restoration of bony alignment, etc; MIO often requires more training and advance planning. The inability to directly visualize the entire bone may result in fracture mal-alignment in some instances, especially for those inexperienced in MIO. Similarly, the inability to fully visualize the affected bone may result insufficient fixation. Intraoperative imaging using a fluoroscopic imaging (C-arm) unit is tremendously helpful for most (but not all) MIO fracture treatments. Such imaging represents a significant additional hospital expense and a source of radiation exposure to the patient and hospital staff. Such imaging may be less essential for some external skeletal fixation applications.

SACROILIAC LUXATION REPAIR VIA MIO

Surgical fixation is indicated for SI luxation when one or more of the following clinical or radiographic signs are present: 1) significant instability and displacement of the hemi-pelvis, 2) neurologic deficits or pain attributable to the luxation or 3) obstruction/collapse of the pelvic canal. Surgical fixation can be achieved by open reduction and internal fixation (ORIF) or minimally invasive osteosynthesis (MIO). ORF induces some morbidity associated with the surgical approach and traction for visualization of the articular surface of the sacral body.

MIO for SI luxations utilizes percutaneous reduction and fixation that drastically reduces the patient morbidity associated with open surgical exposure and reduction. Studies by Bowlt, Shales and Langley-Hobbs have also made it clear that the "safe corridor" for lag screw fixation is relatively small and that there is considerable anatomic variation between patients that can challenge or preclude accurate and secure fixation when ORF is employed. While intraoperative imagingisrequired for MIO of SI luxation, this imaging simplifies the surgical procedure and improves fixation accuracy. Accurate application of fixation implants is important due to the proximity of important neural and vascular structures and it ensures optimal fixation strength.

Tomlinson, et al nicely described the technique for MIO of SI luxations under fluoroscopic guidance in the dog. Patients are positioned in lateral recumbency upon the surgical table and this position is adjusted until thefluoroscopic appearance of superimposition of the transverse processes of the lumbar vertebrae and a distinct triangular shape of the sacral body confirms **perfect**laterality. The luxation is reduced via percutaneous application of bone forceps on the ilial wing and/or ischium. Provisional fixation is achieved with a k-wire placed from the ilium into the sacral body using fluoroscopic guidance. Next, 1 or 2 screws are placed in lag fashion using the same intraoperative imaging technique. Because of the need for such imaging, the surgery table should have a radiolucent surface and operating room personnel should adhere to appropriate radiation safety guidelines.

MINIMALLY INVASIVE NAIL FIXATION (MINO)

Interlocking nails (ILN) are specifically designed intramedullary devices with transverse cannulations that accept fixation bolts. The intramedullary nail imparts resistance to disruptive bending forces while the rigid interlock of the fixation bolts within the nail cannulations on each side of the fracture or osteotomy imparts stability against axial compression and rotational forces. Since these nail cannulations cannot be visualized after the nail is placed within the intramedullary canal, a specific aiming guide system is part of the instrumentation set to ensure accurate placement of the fixation bolts. With technical training and acquired expertise, the nail and fixation bolts can often be placed percutaneously without disruptive of the fracture zone. Recent advances in veterinary ILN devices offer enhanced fixation strength imparted by angle-stable fixation bolts (i-LocÒ, BioMedtrix, Whippany, NJ). Normograde insertion of ILN is the only option for MINO. MINO is an option for many highly comminuted (non-reducible) femoral, humeral and tibial fractures. Preoperative planning typically includes orthogonal radiographs of the intact contralateral bone using magnification calibration and templating methods to determine the optimal nail diameter and length.

MINIMALLY INVASIVE PLATE OSTEOSYNTHESIS (MIPO)

MIPO is the application of bone plates, often in bridging fashion, without performing an open surgical approach to the fracture. MIPO is often used for highly comminuted ('non-reducible') diaphyseal fractures as anatomic reduction is not the goal for such fractures. Instead, "functional reduction" is the goal; also called "spatial alignment", it restores bone length and proper alignment in the frontal, sagittal and transverse planes. Indirect reduction techniques are used to obtain this spatial alignment without opening fracture zone.

Preoperative planning for MIPO, as with MINO, typically requires orthogonal radiographs of the intact contralateral bone using magnification calibration and templating methods to determine the optimal plate width and length. Plates that span the full bone length are often used for MIPO to reduce fixation stress; longer plates with a limited number of screws at the ends of the plate are able to sustain greater loads to failure than shorter plates in which all holes are filled with screws. Plates are usually pre-contoured to the approximate bony contours by fitting to the bone surface on size-matched radiographs. As it is difficult to estimate the twisting contours of the bony surface from a 2-dimensional radiograph, fitting to size-matched skeleton can be very helpful. The precontoured plate is then sterilized and is ready for use at surgery.

Standard bone plates utilize screw fixation to pull the bone firmly against the underneath surface of the bone plate. Fixation, then, relies upon the strength of screw purchase and its ability to generate friction between the bone and the bone plate. Thus, conventional bone plating requires excellent screw purchase and precise anatomic contouring of the bone plate. Many locking plate/screw systems have been introduced to the veterinary market in recent years. In brief, these systems utilize a rigid interlock between the screw and bone plate. This interaction between the fixation element (screws) and bridging element (plate) resembles the mechanical principles of an external skeletal fixator in which fixation pins are rigidly linked to the connecting rod. Thus, many have referred to locking bone plates/screw systems as "internal fixators", especially when used for bridging fixation. One advantage to locking plate/screw systems (compared to conventional plating) is that these plates do not require precise anatomic contouring (much like the connecting rod of external fixators is not contoured to the bone). Many of the MIPO implant systems and techniques were, consciously or not, developed to ascribe to internal fixation the many inherent advantages of external fixators.

MIO VIA EXTERNAL SKELETAL FIXATION

External skeletal fixation (ESF), though historically associated with high patient morbidity, was revolutionized by the development of advanced devices and surgical techniques in veterinary medicine. This resurgence of interest and innovation began in the 1990's and continues today. Current generation techniques and instrumentation permit user-friendly application of simpler, yet mechanically robust fixation frames. This concurrent optimization of methodology and instrumentation, which fostered a profound reduction in patient morbidity, revealed the inherent versatility and biological advantages of the system. Modern ESF includes linear, circular and hybrid external fixator devices. Linear ESF utilizes fixation pins and connecting rods, whereas circular ESF typically uses fine wires tensioned between clamps on rings placed around the limb. Hybrid ESF has become very popular for stabilization of juxta-articular fractures and osteotomies; the linear portion of the frame is secured to the longer bone segment while the fine-tensioned wires placed on a single ring are used for fixation of the short, juxta-articular bone segment.

Modern ESF is a very versatile system that is well suited to the ideals of MIO. It provides variable angle, locked fixation that can be applied with minimal/no disruption of the fracture zone. Rigid bilateral or multi-planar frames are relatively simple to apply in instances of nonload-sharing fixation of non-reducible fractures and simple, timely, progressive frame disassembly allows for gradual shift of loading from the fixation device to the healing bone. Understanding and strict adherence to the principles of ESF are essential in order to reduce complications and fully realize the advantages inherent to the system. Annual courses that provide comprehensive training opportunities are available in the US and elsewhere (www.imexvet.com).

Helpful Resources:

Techniques in External Skeletal Fixation – I, VideoVet 2017, www.videovet.org

Surgical Approaches of the Canine Pelvic Limb – I; VideoVet 2017, www.videovet.org

Surgical Approaches of the Canine Thoracic Limb – I; VideoVet 2017, www.videovet.org

43RD WORLD SMALL ANIMAL VETERINARY ASSOCIATION CONGRESS AND 9TH FASAVA CONGRESS

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SVA FELINE

GI ENDOSCOPY: A MINIMALLY INVASIVE OPTION FOR THE MINIMALLY INVASIVE CAT OWNER

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Feline gastrointestinal (GI) endoscopy is in high demand, particularly by cat owners already aware of the clinical benefits and availability of this procedure within the human heathcare system. This lecture will provide a basic introduction to GI flexible endoscopy including important aspects of endoscope selection, clinical indications, and basic techniques required to perform a thorough and diagnostically meaningful examination in the cat.

Introduction:

Gastrointestinal endoscopy offers a minimally invasive method for obtaining a relatively thorough examination of the GI. Procurement of biopsy samples is often necessary in achieving a diagnosis in patients presenting with chronic or recurrent gastrointestinal tract signs (>2 weeks). In addition, endoscopy also offers a minimally invasive option for foreign body retrieval, oesophageal stricture dilation therapy and percutaneous gastrostomy tube placement.

Endoscopy should be considered the next logical step when investigating those patients where haematology, biochemistry, electrolytes, anthelmintic therapy, B12 assessment/supplementation, fPLI/TLI, and abdominal imaging have failed to provide an explanation for presenting clinical signs. In cats, oesophagoscopy, gastroduodenoscopy, proximal jejunoscopy, distal jejunoscopy, illeoscopy and colonoscopy should always be considered in those cases presenting chronic weight loss, polyphagia, hyporexia/anorexia or chronic hypocobalaminemia. This is especially true for cases with confirmed hypocobalaminemia where significant distal small intestinal evaluation/biopsies are required.

The World Small Animal Veterinary Association (WSAVA) International GI Standardisation Group and the American College of Veterinary Internal Medicine (ACVIM) have published multiple statements to provide guidance and standards for performance and interpretation of various diagnostic tests in dogs and cats presenting with gastrointestinal signs, including treatment trials, patient response, and outcome.[1,2] Interestingly, inflammatory bowel disease (IBD) and lymphosarcoma (LSA) are often the remaining differential diagnoses in these patients. The clinical history and findings up to this critical point seems to present a clinical conundrum for the practitioner resulting in either polypharmacy or offering invasive interventions (e.g. exploratory laparotomy). It's not unsurprisingly that both of these options are often met with resistance by both patient and cat owners.

For most practitioners, the most challenging question associated with obtaining biopsies is how to obtain tissue samples of adequate depth and at the correct anatomic location. Whether to obtain full thickness biopsies or endoscopic biopsy samples has been a subject of discussion over the past 10-15 years. Benefits of endoscopic biopsies include the ability to directly visualise and document mucosal changes and lesions, enabling direct access and biosies of these changes, to collect multiple tissue samples from each anatomic site, and to begin antiinflammatory

or chemotherapy without delay after the procedure.[3]

Unfortunately, endoscopy has some limitations in that it rarely diagnoses functional diseases (e.g. dysmotility, dietary hypersensitivity, antibiotic-responsive disease, etc.), lesions outside the GI tract (liver, pancreas, etc.) nor allows for full evaluation of the entire jejunum.[3] Other clinical challenges associated with implementing endoscopy into a GIT investigation include lack of appropriate/suitably-sized equipment to perform a thorough examination, insufficient operator training/understanding how to 'drive the 'scope' through the gastrointestinal tract, and/or confidence with identifying normal from abnormal. The endoscopist performing procedure should also have a high level of proficiency, as GI endoscopy in cats is more demanding in skill than in dogs, due to the small size of the animal and the decreased tolerance to anaesthesia.

Theoretical risks which should be communicated with the owner include: 1) general anaesthesia 2) bowel perforation 3) bleeding 4) non-diagnostic samples (depth of sample and length of endoscope in relation to lesion). Saying that, these risks are considered rare.

Equipment:

A flexible endoscope suitable for performing an upper and lower GI examination in cats should be no greater than 8.0mm in diameter, at least 100cm in length and must have four-way tip deflection. In Oriental lines, where the antral canal, pylorus and ileocolic sphincter are seemingly more narrow compared to other breeds will benefit from the use of a smaller diameter endoscope (<6.0mm).[4]

To obtain adequately sized biopsies, the instrumentation channel should be at least 2.2mm, preferably ≥ 2.5mm. The latest model of endoscopes specifically designed for veterinary use, and suitable for most cats, is a 7.9mm outer diameter x 1.40m videoendoscope with an instrumentation channel of 2.8mm in diameter.[4]

Anaesthesia:

General anaesthesia is required for GI endoscopy and intubation with a cuffed endotracheal tube is mandatory because of the risk of gastro-oesophageal reflux. Some anaesthetic agents affect intestinal motility and sphincter function making the passage of the cardia and pylorus potentially more difficult.[5] Atropine and other anticholinergic drugs should not be used unless necessary to increase heart rate, as these drugs may alter gastric motility patterns and increase pyloric tone. Pure opioid agonists may also increase pyloric tone and should ideally be avoided. Fluid support should be given during anaesthesia; dehydrated animals need to be rehydrated before anaesthesia unless endoscopy needs to be performed as an emergency procedure. In hypoproteinaemic patients colloid administration should be considered to ensure reasonable oncotic pressure.

Anaesthesia monitoring during upper GI endoscopy includes at minimum assessment of heart rate, respiration, blood pressure and pulse oximetry. Overdistension of the stomach during insufflation can cause cardiorespiratory.

Biopsies

The quality of endoscopically obtained biopsies greatly influences the likelihood for an accurate histopathological diagnosis of a specific mucosal lesion.[1,2] In general at least six adequate samples need to be collected from the feline gastric or duodenal mucosa to reliably detect abnormalities.[1,2] An adequate biopsy should have a full thickness mucosa and at least three or four intact, if possible contiguous villi.[2] Samples containing submucosa are preferred. These requirements can be challenging to meet in the feline intestine due to the small intestinal diameter and therefore limited feasibility to direct the forceps tip in to the mucosa at a suitable angle and depth.

After obtaining the specimen, the biopsy forceps are removed from the endoscope and the tissue sample is carefully removed from the jaws. Placing the jaws containing the sample into a container filled with isotonic saline and then "washing-off" the sample by shaking the tip of the forceps helps to avoid damaging the edges of the cups Specimens should be submitted in fixative, to the pathology laboratory, as quickly as possible for histopathological examination. Submission of a complete history and macroscopic evaluation report to the pathologist is mandatory to obtain best results.

Unfortunately, there's often a big discrepancy between the macroscopic appearance and final histological diagnosis thus emphasising the absolute importance of collecting endoscopic biopsies during all endoscopic examinations. Differentiation between lymphoplasmacytic IBD and small-cell lymphoma is notoriously difficult and should be confirmed with immunohistochemistry and PARR testing in those clinically suspicious cases.[7,8]

Colonoscopy

Large intestinal disease in cats commonly results in large bowel diarrhoea or constipation. It must be remembered, that vomiting is another frequent complaint in cats with large bowel disease. Furthermore, haematochezia can be due to local problems in the large intestine or anorectal disease and must be differentiated from generalised coagulopathies. As mentioned previously, a thorough investigation for systemic diseases is warranted for all these problems including haematology, biochemistry, coagulation testing, faecal analysis and diagnostic imaging.

The commonest abnormal large intestinal disease in cats is some form of colitis. This can be limited to the colon or be part of IBD or even small cell lymphoma of the entire intestinal tract. Colonic tumours, especially lymphoma, can look identical to inflammatory colitis and must be differentiated by histopathology and PARR (+/-immunohistochemistry)[7,8], for definitive diagnosis. Other large intestinal tumours in cats include adenocarcinoma and leiomyosarcoma, both being more focal and often invading the lumen with an irregular proliferative appearance. Other rare findings in cats include ileocolic or ileocaecal intussusception (often diagnosed via ultrasound prior to endoscopy) or rectal stricture.

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43RD WORLD SMALL ANIMAL VETERINARY ASSOCIATION CONGRESS
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ANIMAL WELLNESS & WELFARE

ONLINE SALE OF DOGS AND CATS: THE GOOD, THE BAD AND THE UGLY

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ONLINE SALE OF DOGS AND CATS: THE GOOD, THE BAD AND THE UGLY

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The popularity and market for pets

A large proportion of the population live with a dog or cat in many parts of the world. These pets can become part of the family, and people spend enormous amounts of money on care of them. In many parts of Asia, such as Singapore and China, there is an exponential increase in the keeping of dogs and cats as pets.

As most dogs and cats live considerably less time than their owners, there is a large market for breeding and selling pets. Prior to the introduction of the internet, people would find their dogs and cats mostly through friends or family who bred the puppies or kittens, or local breeders or shelters. If people wanted a rarer breed then they might use newspapers or magazines to find an animal in another area, and then drive to pick the puppy or kitten up.

Today in our online economy, people have moved to buy most goods and services online, and the online sale of dogs and cats has also increased dramatically. To find out more about pets traded online we collected Gumtree ads for dogs and cats at three time points during February 2016 [1]. To date we have analysed only dogs and cats identified as being relinquished by their owners, not the younger puppies and kittens being sold. Extrapolation gives an estimate of over 31,000 dogs and 24,000 cats relinquished on this single website per year around Australia. The numbers are likely to be much lower than the total numbers traded online as there are many more online websites on which pets are traded, including Facebook®.

While there is limited research into the numbers of dogs and cats sold online, research in the European union suggests there are up to 269,620 dogs and 67,847 cats advertised online on any given day [2]. In fact it is estimated that online sales are now the most common method people use to find a pet.

Why do online sales matter?

The main implications for the exponential rise in online sales of dogs and cats can be grouped into five main areas as described below. None of the reasons listed are specific to the internet. However, the inherent risks related to the large numbers of animals that can be easily advertised to a wide market, and the potential anonymity of sales online, are substantially higher than through other methods of trade.

1. Risk to the welfare of the dogs and cats: Selling online has opened up the market for breeders only interested in making a profit. These breeders can now advertise to a much larger audience, and the anonymity of the internet makes it easier to hide the breeding premises if they are not complying with local standards of animal welfare (also see point #5 below). Puppies and kittens may be weaned too early, and breeders may lie about the true age of the animal.

2. Biosecurity and movement of diseases between regions: Diseases such as heartworm and leptospirosis only occur in some regions of Australia. There are other examples around the world where animals are taken from an area where a disease is endemic to another area in which the disease is less common or does not occur. Further research is needed to determine the implications to disease spread of this movement of dogs and cats.

3. Unknown or incorrect previous history of the animal: In well managed animal shelters the dogs and cats have their behavior and health assessed prior to being made available for adoption. In contrast, animals traded online may have a blank history, even if there are problems with their behaviour and/or health. Responsible breeders and owners will inform new owners of a health or behavioural problem, but not all owners are responsible. They may also be conflicted, as telling people that the dog they want to rehome has a behavioural problem may mean that they cannot find a new home for it.

4. Long distance transport: In many areas the air transport of young puppies or kittens is not regulated, for example there are no regulations in Australia. For a young animal, being taken abruptly from its mother and littermates and put into air freight with loud noises and unfamiliar people, must be a frightening experience. The timing is also likely to coincide with the socialisation period in which positive or negative experiences are magnified, which may set the animal up for anxiety-related problems throughout its life.

5. Scams and misleading ads: There are reports of scams on the internet, including people receiving sick or diseased animals, or a breed unlike the description on the online ad. Veterinarians are left supporting their clients through expensive veterinary treatments and sometimes even legal battles.

What can veterinarians do?

In the UK the Pet Advertising Advisory Group (http://paag.org.uk/) promotes responsible advertising and minimum standards for the online sale of animals. Although voluntary, this at least provides some improvement in advertising and also education via pop-ups, for people looking for a pet online about what to look for to identify a 'good' ad. Similar groups have now arisen in other countries. As a profession we should be advocating for a group to be developed in Australia.

Individual veterinarians in practice can have a significant impact by educating the pet owners they see. Good advice includes always seeing both parents when buying a puppy or kitten, and meeting the animal and finding out its history before taking home a relinquished pet. This needs to be case specific. For example, mature people with dog training experience can take home a dog with some behavioural problems, but a young busy family with children under 5 years of age should be counselled not to. Veterinarians can also inform people of the risks of online trade, including scams where there is no pet, receiving a sick or diseased animal, or even receiving an animal that does not fit the breed advertised.

Conclusions

The internet has changed forever the way people find their new pets. There are reputable breeders and owners using the internet to advertise their pets. The main problem is for members of the public to be able to easily determine the good from the bad ads. Veterinarians in clinical practice are at the frontline in seeing newly acquired pets, and should include in their history both the location the pet came from and how it was found. Veterinarians also have an important role in education. Protecting the welfare of pets will help to nurture the human-animal bond which is a great outcome for everyone.

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THE THORAX EXAMINED BY RADIOLOGISTS - HOW TO AVOID MAKING MISTAKES

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NEW SONOGRAPHY TECHNOLOGY IN VASCULAR IMAGING

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Doppler Effect is a change in frequency (and wavelength) caused by the motion of a sound source, reflector and receiver. If a reflector is moving toward the source and the receiver, the received echo of the reflector has a higher frequency than would occur without the motion. On the other hand, if the motion is away from the source and the receiver, the received echo of the reflector has a lower frequency. The amount of the frequency change depends on the speed motion of the reflector, the angle between the wave propagation, direction and the motion direction, and the frequency of the wave emitted by the source. The change cause by the motion is called the Doppler-shift frequency or **Doppler shift**. The **Doppler shift** depends on the speed of the reflector and the frequency of the source. It is the difference between the frequency of the source and the returning frequency recorded by the receiver.

In medical ultrasonography, the reflector is the cells in the blood stream, and the source and the receiver is the transducer used in the examination. The **Doppler Effect** is a result of the motion of blood. A maximum positive **Doppler shift** occurs if the direction of the sound propagation is exactly opposite the flow direction. A maximum negative Doppler shift occurs if the direction of the sound propagation and the flow direction is similar. If the flow is perpendicular to direction of the sound propagation, the Doppler Shift, no frequency change and thus no **Doppler Shift** is detected.

There are three forms of displaying Doppler: Colour Doppler, Power Doppler and Spectra Doppler (tissue Doppler). The Continuous Wave (CW) and Pulsed Wave (PW) PW are display in a form of Spectra Doppler.

Note that the blue colour coded blood vessels represent blood vessels that are flowing away from the transducer (at the top of the image). In the top half of the kidney, they represent renal vein, where the blood flow from

the peripheral to the center of the kidney. The red colour coded blood vessels at the top half of the image represent renal arteries that are flowing from the hilus to the peripheral margin of the kidney.

In colour Doppler, we sometimes encounter colour flow aliasing, which will lead to reversal of the colour map showing mixing of blues and reds, which is termed Mosaic pattern. Aliasing happens due to high velocity flow with and velocity scale setting error. To optimize the colour Doppler, a lower transduce frequency, and a smaller color sector width should be utilize. Most of the currently ultrasound machine will be able to display a normal black and white image with colour Doppler at the same time.

Power Doppler:

Power Doppler displays the integrated power of the Doppler signal by a single color map instead of the mean frequency shift. There is no aliasing with power Doppler. Flow direction and velocity information normally is not known. This display is more sensitive then colour Doppler to small vessels and those with slow flow. **In some advance ultrasound machines, directional power Doppler, which is sensitive to slow flow but at the same time showing the direction of the flow is available.**

In recent ultrasonographic technology advances, multiple companies has produced Doppler that is very sensitive to slow flow. Most of them are available in the premium line of products. All of them has specific name by the company: for example Superb Micro-Vascular Imaging (SMI) of Toshiba, B-Flow of GE, S-Flow™ of Samsung, MicroCPA of Philips and Fine Flow of Hitachi Aloka. One of the advantages of this technology is be able to detect the actual margins of the blood vessels.

The potential application of the advance technology in veterinary medicine is still unknown. The extreme sensitivity of this for detection of blood flow may be useful in the determination of change of blood flow in the treatment of neoplasia, thus evaluating the effective of treatment.

NURSES (HILLS)

UPDATE ON CARDIOPULMONARY RESUSCITATION

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THE INS AND OUTS OF ANAESTHETIC MONITORING FOR OPTIMAL PATIENT OUTCOMES

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Stages of Anaesthesia

Four stages and planes of anaesthesia have been classified. The use of intravenous anaesthetic agents plunge the animal through the initial stages so rapidly that they may not be recognised.

Stage 1	Still conscious but disorientated, amnesia, unaltered threshold to pain, but some pain is tolerated
Stage 2	Unconsciousness, excitement, delirium & uninhibited action. Suppression of higher control centres; struggling, crying, exaggerated response to pain / stimuli Irregular respiration

Stage 3	Progressive depression of respiration, circulation, protective reflexes, muscle tone. Divided into 4 planes
	Plane 1
	• Respiration: regular, full use of intercostals muscles & diaphragm
	• Pupils: constricted, slow nystagmus may be present
	• rd eyelid partially protruding
	• Salivation and lacrimation and pharyngeal and laryngeal reflexes persist. Can intubate a horse or dog but not a cat.
	• Muscle tone still present.
	• Painful stimuli cause limb retraction and elevation in HR, RR and blood pressure.
	Plane 2
	• Respiration: slight decrease in tidal volume (intercostals not working as well) and increase in respiratory rate.
	• Eye: eccentrically fixed with halothane, centrally fixed with ether and methoxyflurane, centrally fixed with halothane in the horse.
	• Lateral nystagmus still present in the horse.
	• Surgical stimulation produces HR, BP and RR.
	• Oropharynx reflexes abolished in dog but not completely in the cat.
	• Deep tracheal reflexes persist.
	• Muscle tone lessens.
	Plane 3
	• Deep Surgical Anaesthesia
	• Respiration: The intercostal muscles weaken and lag behind the diaphragm contraction. This unevenness creates a rocking boat type respiratory movement.
	• Abdominal muscles are relaxed.
	• Eye: eyeball is slightly downwardly rotated in a ventro-medial direction
	• Palpebral reflex: absent in dog and cat but only slowed in the horse.
	• Corneal reflex: absent in dog and cat but present in the horse.
	• Lacrimation, salivation, oropharynx and laryngeal relaxes all abolished but deep visceral pain and vagal responses remain.
	• Muscle relaxation is good.
	Plane 4
	• Too Deep
	• Respiration: only diaphragmatic breathing. Tidal volume is reduced.
	THE INSPIRATORY DIAPHRAGMATIC CONTRACTION MAY PRODUCE A TRACHEAL TUG WHICH IS AN EXAGGERATED MOVEMENT OF THE TRACHEA AND LARYNX AND EVEN THE MANDIBLE, ALL OF WHICH CAN BE INCORRECTLY (AND DANGEROUSLY) INTERPRETED AS LIGHTENING OF ANAESTHESIA.
	• This plane starts with the paralysis of the intercostal muscles and ends in apnoea.
	• Eye: cornea is dry and dull. Pupils are dilated.
	• Corneal reflex vanishes in the horse.
	• Heart rate and blood pressure fall.
	PLANE 4 IS A DANGEROUS AND UNNECESSARY DEPTH OF ANAESTHESIA.
Stage 4	The period between respiratory arrest and cardiovascular collapse and arrest. The length of this period depends on the anaesthetic used, degree of oxygenation at the beginning of respiratory arrest and the species, i.e. this time is zero for birds!

To achieve surgical anaesthesia we must maintain the animal at Stage 3 between Planes 2 and 3. The deeper Plane 3 will be needed for painful procedures that require good muscle relaxation – eg, orthopaedic and ophthalmic operations. If the patient is being maintained at Stage 3, Plane 3 careful monitoring and I/V fluid support, if the procedure is prolonged will be required.

Monitoring Of Anaesthesia

Early detection of adverse events and evaluation of corrective intervention's the aim of the game. Various pieces of equipment are of use but reliance should not be placed on just one item. For example, the apnoea alert. By the time this alarm sounds to signal that the animal is no longer breathing, irreversible damage has occurred to the heart and brain. YOU ARE TOO LATE!

Remember these important rules:

• Monitor the patient not the equipment;

• Check the patient before checking the equipment;

• A machine should not replace a skilled anaesthetist.

A machine is primarily directed at the body systems that are essential for the maintenance of life - the central nervous system and the cardiovascular system. Thus we need to monitor the following:

• Central nervous system – reflexes, depth of anaesthesia.

• Cardiovascular system – heart rate, perfusion.

• Pulmonary system & airway

• Oxygenation

• Temperature – this is difficult to maintain and subnormal temperatures will slow metabolic rate and lengthen recovery and post anaesthetic complications.

Note: Never over interpret one piece of information. The aim of anaesthetic monitoring is to obtain data from a number of different body systems that will allow an overall picture of the patient to emerge. The most important monitor is the operator's senses – sight, hearing, touch etc.

Monitoring of Central Nervous System Reflexes

Basic reflexes and muscle tone are the method of monitoring whether the brain still functions. Under anaesthesia most of the central nervous system undergoes a selective, dose dependent reversible depression caused by the anaesthetic drugs at their site of action. Basic reflexes are:

• Spontaneous motor activity

• Jaw tone

• Palpebral reflex

• Corneal reflex

• Lacrimation

- Pupil position
- Respiratory effort (intercostal/diaphragmatic)

Summary of Central Nervous Monitoring

Jaw Tone	Absent following induction - by Stage 3
Palpebral reflex	Absent by Stage 3 plane 2 - 3 in dog and cat Slowed in horse at Stage 3 plane 3
Corneal reflex	Very dulled, almost absent by Stage 3 plane 3
Pedal reflex	Absent by Stage 3 plane 2
Lacrimation	Absent by Stage 3 plane 3
Eye position	Stage 3 plane 1 – located in medial canthus with 3rd eyelid partially protruding Stage 3 plane 3 – ventral medial direction Stage 3 plane 4 – central and fixed, eye dry and dull
Pupil Position	Stage 3 plane 1 – pupils constricted Stage 3 plane 3 – pupil mid-point Stage 3 plane 4 – pupils dilated *remember certain drugs affect pupil size*
Laryngeal reflex	Absent in the dog by stage 3 plane 2 (can intubate dog at stage 3 plane 1) Reduced (but not absent) in cat at stage 3 plane 2 Absent in cat at stage 3 plane 3
Respiration	Stage 3 plane 1 – regular with full use of muscles & diaphragm Stage 3 plane 2 – slight decrease in tidal volume & slight increase in respiratory rate Stage 3 plane 3 – intercostals muscles weaken and lag behind diaphragm contraction; creates unevenness & a 'rocking' boat type respiratory movement Stage 3 plane 4 – only diaphragmatic breathing Stage 4 - apnoea

Monitoring the Cardiovascular system

The aim of the cardiovascular system is the maintenance of adequate tissue perfusion with oxygenated blood. The aim of intraoperative monitoring of the cardiovascular system is to ensure that it is achieving this and intervene if it is not.

Mucous Membrane Colour

The colour of the mucous membranes (gums, conjunctiva, vagina etc) provides information on the adequacy of oxygenation of blood in the lungs, adequacy of blood volume, cardiac output and the delivery of oxygenated blood to the tissues.

- Pink – good perfusion and oxygenation.
- Pale – poor perfusion. This may be the result of (a) poor perfusion from low cardiac output – cardiac problem, low venous return; (b) poor perfusion from vasoconstriction – PAIN; (c) low blood volume – haemorrhage.
- White – severe blood loss, hypovolaemia etc – immediate action required
- Cyanotic – slight blue / grey tinge to mucosa – immediate action required
- Muddy – this indicates both poor oxygenation and poor perfusion. Beware, this animal is in trouble and the oxygen concentration in the blood is below normal!

Capillary Refill Time

Place firm digital pressure on a mucous membrane. This will push all blood from the capillary bed. The speed at which it returns is relative to blood pressure and blood volume. A normal rate is less than 2 seconds.

If it is longer than 2 seconds AND the colour is muddy, then the animal is shunting blood away from the periphery of the body because of a drop in blood pressure – alert the veterinarian immediately

Heart Rate

Although mechanical monitoring is often implemented by the use of a pulse oximeter or similar, it is always best to listen to the heart rate. This can be achieved by using a stethoscope (as long as it does not interfere with the surgical site) or by use of an oesophageal stethoscope.

External auscultation

Place the stethoscope on the left hand side where the elbow lies against the chest. Reposition slightly to give best hearing of heart sounds. The apex of the heart points downwards and lies near the sternum at the level of rib 7.

Listen to the heart sounds. The normal sound is described as being a "lub, dup" sound. The sound that we hear is caused by the snapping closed of certain heart valves as the chambers contract and start to eject blood.

The heart should have a regular beat, if you are concerned that the beat is irregular ask the veterinarian to listen.

Listen to the rhythm. Normally the heat beats will have the same interval between them or has a sinus arrhythmia. This means there is a regular irregularity. The heart speeds up slightly as the animal inhales and slows down slightly as it exhales.

If there is an abnormal arrhythmia there will be irregular gaps between heat beats. This irregularity can be heard

To calculate the heart rate, count the beats for 15 seconds then multiply by four to gain heart per minute.

Oesophageal Stethoscope

The oesophageal stethoscope is passed down the oesophagus to the level of the heart. It enables constant monitoring of the heart beat either via the ear pieces or can be amended to attach to small speakers.

Heart rate & anaesthetic Depth

General indicators:

- Increased heart rate – tachycardiac – decreasing depth of anaesthesia – becoming lighter
- Decreased heart rate – bradycardia – increasing depth of anaesthesia – becoming too deep

Arterial Blood Pressure

Arterial blood pressure is useful in providing continuous information on the performance of the cardiovascular system and indirectly on the depth of the anaesthetic. However, it is important to remember that good blood

pressure during anaesthesia does not always correlate with good perfusion. That is, increased blood pressure > vasoconstriction > decreased blood flow.

Direct measurement of blood pressure involves catheterisation of a peripheral artery – dorsal metatarsal, femoral, lingual and carotid. Indirect measurement is commonly done using a **Doppler ultrasound unit** to detect arterial blood flow and a pressure cuff.

Using the Doppler

- Cuff should be placed above hock for dorsal metatarsal or above carpus for dorsal pedal artery
- Follow the cuff manufacturer's instruction on choosing a size that is appropriate for the limb. In general, the width of the cuff should measure 30 – 50% the circumference of the limb
- Ensure snug fit
- Measurement should be performed several times to ensure correct.
- Clip area over artery to be used (dorsal metatarsal, dorsal pedal)
- Apply liberal amount of gel to area probe is to be placed
- Position probe perpendicular to the artery
- Tape in place
- When pulse audible – increase pressure until no longer audible
- Slowly decrease pressure until pulse just becomes audible once more. This is the systolic blood pressure reading
- This should be repeated at least 3 times to ensure correct reading

Pressure readings are expressed in mmHg (millimetres of mercury) e.g. 120mmHg.

The pressure (or tension) of the blood within the arteries is maintained by the contraction of the left ventricle, the resistance of arterioles & capillaries, the elasticity of arterial walls and viscosity and volume of blood. Changes to any one of these factors will affect the blood pressure.

Systolic blood pressure results from systolic contraction of the cardiac chamber. The highest arterial blood pressure reached during any given ventricular cycle

Diastolic blood pressure results from diastolic relaxation of the cardiac chamber. The lowest arterial blood pressure reached during any given ventricular cycle.

Normal Systolic Blood Pressure in animals	100 – 160 mmHg
Normal Diastolic Blood Pressure in animals	60 – 100 mmHg
Normal Mean blood pressure in animals	80 – 120 mmHg

*Blood pressure ranges can vary depending on referring text

A mean arterial blood pressure of greater than 60mm of mercury (mmHg) is required to ensure adequate perfusion of vital organs such as the brain and kidney. To be cautious, it is best that the anaesthetised patient maintain a mean arterial blood pressure of greater than 70mmHg. Because the Doppler ultrasound measures only systolic pressure and because the systolic pressure is about 20 to 30mmHg higher than mean pressure, Doppler readings of greater than 90mmHg are desirable.

In the dog it is time for action if the Doppler readings fall below 80mmHg. In the cat this time for action occurs at 65mmHg (there is an overestimation of mean arterial pressure in cats with this device).

Monitoring of the Respiratory System

It is not just good enough to have blood being delivered at an adequate rate and pressure to the body, there must be oxygen in that blood for any good to be done.

Pulse Oximetry

Use a pulse oximeter to monitor that the patient is oxygenating sufficiently. The pulse oximeter measures the pulse rate and the degree of oxygen saturation of arterial blood, by measuring the absorbency of particular wavelengths of red and infrared light.

Most instruments require a probe placed on non-pigmented skin or mucous membrane (most frequently the tongue). They are susceptible to movement, and interference to blood flow by the clamp resulting in erroneous measurements. Also, saturation measurements may be inaccurate in cases of hypovolaemia.

Pulse oximeter readings of SpO_2:

- 98 - 99%   Normal
- <95%   Hypoxemia
- <90%   Severe Hypoxemia
- <75%   Lethal Hypoxemia

Areas where a probe may be placed include:

- Tongue
- Lip
- Ear
- Between toes
- Prepuce / vulva

If placing the probe on the tongue, place a damp/wet swab over the tongue to keep it moist, then place probe on over this.

Respiration

The rate, rhythm and nature of breathing effort should be consciously observed. See previous notes in 'Summary of Central Nervous Monitoring' table. Changes in the respiratory rate and effort may indicate a change in areas such as

Anaesthetic depth

- Increased respiratory rate - becoming lighter
- Decreased respiratory rate – becoming deeper
- Increased or 'jerky' respiration may indicate that the patient is feeling pain stimuli

It is also important to monitor both the rebreathing bag and the patient's thorax to detect:

- Anaesthetic circuit disconnection;
- Inconsistency between breathing excursions of the chest and the amount of air moving in and out of the bag;
- Airway obstruction problems (kinked endotracheal tube).

End tidal CO_2 monitor

Capnometry is the measurement of carbon dioxide in exhaled gas of a patient. The basic physiology behind capnometry is that tissues generate carbon dioxide that is delivered to the lungs, via the blood and then exhaled. The exhaled carbon dioxide is sampled from the distal end of the endotracheal tube, analysed by a capnometer and displayed on a capnograph.

Capnographs generally work on the principle that CO_2 absorbs infra-red radiation; a beam of infra-red light is passed across the gas sample to fall onto the sensor, the presence of CO_2 leads to a reduction in the amount of light falling onto the sensor which in turn changes the voltage in the circuit and the reading is portrayed.

Phases of a Capnogram

There are 4 distinct phases of the capnogram:

Phase I

The initial flat portion. The animal is inspiring and because inspired gas is 100% O_2 the CO_2 reading is 0.

Phase II

Ascending portion of the graph that represents the first appearance of carbon dioxide during exhalation. As the animal exhales, the first air out represents what was in the trachea. This is relatively low in CO_2. Next the bronchial and bronchiolar air comes out with increasing levels of CO_2.

Phase III

A plateau representing the exhalation of alveolar gas which contains the highest level of CO_2. The very last part of phase III is the air that was deep in the alveoli and is referred to end tidal CO_2.

Phase IV

The downward slope that corresponds to the initial inspiration. Because there is some CO_2 present in the mechanical dead space of the anaesthetic machine the phase initially shows some CO_2. This rapidly falls to 0 once dead space gas is inhaled only pure O_2 is passing the sampling port.

Normal End tidal C02 = 35 – 45mmHg

Analysing alteration of the normal Graph

An elevated baseline **Phase I** (inhalation) is caused by the patient rebreathing CO_2.

Causes include:

- Exhausted Soda lime
- Incompetent one-way valves in circle system
- Inadequate fresh gas flow rates in a non-rebreathing system

A slanted or prolonged expiratory upstroke **Phase II** indicates an obstruction of airflow. The exhalation of the anatomical dead space air is prolonged giving a gentler slope to that part of the graph.

Causes include:

- Kinked endotracheal tube
- Secretions that may be partially blocking endotracheal tube or patient airways
- Patient bronchospasm

Lack of expiratory plateau **Phase III** indicates lack of good alveolar sample. The animal is not completely exhaling the alveolar air.

Causes include:

- Patients taking small shallow breaths
- High fresh gas flow rates in non-rebreathing systems

Terminology

Hypercapnia

From the Greek hyper = 'above' and kapnos = 'smoke', a condition where there is too much carbon dioxide in the blood. Hypercapnia will generally trigger reflexes that increase breathing and access to oxygen.

Hypocapnia

A state of reduced carbon dioxide in the blood and often results from hyperventilation – deep or rapid breathing.

The Electrocardiogram

The ECG, or electrocardiogram, measures the electrical activity of the heart. In a normal animal, electrical impulses pass through the cardiac tissue in an orderly and regular manner and cause the heart muscle (myocardium) to contract. If this electrical activity is abnormal, then this will result in abnormal muscular contractions.

There are two main types of ECG machines. More modern machines will display the image obtained on a video screen, while the type of ECG machines found more commonly in veterinary practice, record the electrical activity on heat sensitive paper. In both cases, the resulting image is referred to as a trace.

The use of ECG can be a useful aid in the diagnosis of heart conditions when used in conjunction with other diagnostic tools, such as ultrasound (echocardiography) or radiography.

In addition, ECG is also commonly used to monitor the heart rate and rhythm of patients undergoing general anaesthesia and also to monitor 'at risk' patients (i.e. those patients who may have an increased risk of developing cardiac arrhythmias such as post GDV surgery patients).

The **P wave** corresponds to **atrial depolarisation** (i.e. electrical activity), which results in the atrial myocardium contracting (i.e. muscular activity).

The **QRS** waves are usually considered together and are referred to as the **QRS complex**. This complex relates to **ventricular depolarisation** (i.e. electrical activity), which results in ventricular myocardium contraction (i.e. muscular activity).

The final component of the trace is the **T wave**, which relates to **repolarisation** of the ventricles (electrical activity) and resultant relaxation of the myocardium (muscular activity).

WSAVA DENTAL GUIDELINES

ADVANCED EXTRACTION TECHNIQUES AND DEALING WITH COMPLICATIONS

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SURGICAL EXTRACTIONS and COMPLICATIONS

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Challenging extractions are best performed via a surgical approach. Canine and carnassial (maxillary fourth premolar and mandibular first molar) teeth are typically considered "difficult". However, it is also beneficial for teeth with root malformations or pathology (ie.e ankyloses) and retained roots. A surgical approach allows the practitioner to remove buccal cortical bone, promoting an easier extraction process.

A surgical extraction is initiated by creating a gingival flap. This can be a horizontal flap along the arcade (an envelope flap) or a flap with vertical releasing incisions.

Envelope flaps are created by incising the interdental gingiva and then releasing the gingival attachment with a periosteal elevator along the arcade including one to several teeth on either side of the tooth or teeth to be extracted. The flap is created by incising the gingiva in the interdental spaces gingiva along the arcade and then releasing the tissue to or below the level of the mucogingival junction (MGJ). The advantages to this flap are

- Decreased surgical time
- Blood supply is not interrupted
- Less suturing.
- Less chance of dehiscence

The more commonly used flap includes one or two vertical releasing incisions. This method allows for a much larger flap to be created, which (if handled properly) will increase the defects which can be covered. Classically, the vertical incisions are created at the line angle of the target tooth, or one tooth mesial and distal to the target tooth. Line angles are theoretic edges of teeth. However, if there is space between the teeth, either a naturally occurring diastema or from previous extraction, the incision can be made in the space rather than carrying to a healthy tooth.

The incisions should be made slightly apically divergent. It is important that the incisions be created full thickness and in one motion. A full thickness incision is created by incising all the way to the bone, and the periosteum is

thus kept with the flap. Once created, the entire flap is **gently** reflected with a periosteal elevator. Care must be taken not to tear the flap, especially at the muco-gingival junction.

Following flap elevation, buccal bone can be removed. Again, this author favors a cross cut taper fissure bur. The amount is controversial, with some dentists removing the entire buccal covering. However, this author prefers to maintain as much as possible and starts by removing 1/3 of the root length of bone on the mandible and 1/2 for maxillary teeth. This should only be performed on the buccal side. If this does not allow for extraction after a decent amount of time, more can be removed. If ankylosis is present, a significant amount of bone removal may be required.

Following bone removal, multirooted teeth should be sectioned. Then follow the steps outlined for single root extractions for each piece. After the roots are removed (and radiographic proof obtained) the alveolar bone should be smoothed before closure.

Closure is initiated with a procedure called fenestrating the periosteum. The periosteum is a very thin fibrous tissue which attaches the buccal mucosa to the underlying bone. Since the periosteum is fibrotic, it is inflexible and will interfere with the ability to close the defect without tension. The buccal mucosa however, is very flexible and will stretch to cover large defects. Consequently, incising the periosteum takes advantage of this attribute. The fenestration should be performed at the base of the flap, and must be very shallow as the periosteum is very thin. This step requires careful attention, as to not cut through or cut off the entire flap. This can be performed with a scalpel blade, however a LaGrange scissor allows superior control.

After fenestration, the flap should stay in desired position without sutures. If this is not the case, then tension is still present and further release is necessary prior to closure. Once the release is accomplished, the flap is sutured.

Maxillary fourth premolar

The first step when extracting this tooth is to create a gingival flap. Classically this is a full flap with one or two vertical releasing incisors. This will allow good exposure, as well as providing sufficient tissue for closure. However, an envelope flap is sufficient for small and toy breed dogs, as well as cats.

Full flaps are created by making full thickness, slightly divergent incisions at the mesial and distal aspect of the tooth. These incisions should be carried to a point a little apical to the mucogingival junction. Be careful to avoid cutting the infraorbital bundle as it exits the foramen above the third premolar. The flap is then gently elevated with a periosteal elevator.

Following flap creation, buccal bone is removed to

a point approximately ½ the length of the root. Next, the tooth is sectioned. The mesial roots are separated from the distal by starting at the furcation and cutting coronally. Next, the mesial roots are separated by sectioning in the depression between the palatal and buccal roots. Another way to visualize this is to follow the ridge on the mesial aspect of the tooth. When performing this step, a common mistake is not fully sectioning the tooth. The furcation is fairly deep, so make sure that you have it fully sectioned by placing an elevator between the teeth and twisting gently. If fully sectioned, the pieces will move opposite each other easily.

Following these steps, extraction proceeds as described in the last lecture for single rooted teeth

Mandibular first molar

In canine patients, these extractions are further complicated by a groove on the distal aspect of the mesial root. In addition, the mesial root is often curved. Finally, in small breed dogs, there is commonly a significant hook at the apex. Moreover, this tooth is the most common place for an iatrogenic mandibular fracture and it is possible to damage the mandibular nerve and vessels. This is much more likely in small and toy breed dogs, because the roots of these teeth are much larger in proportion to the mandible than large breeds. Bony resorption can significantly weaken the bone and predispose to a mandibular fracture. It is advised to warn clients of these potential complications. Dental radiographs are required to demonstrate the level of remaining bone. Finally, consider referral for these extractions (or possible root canal therapy).

The first step when extracting this tooth is to create a gingival flap. Classically this is was full flap with one or two vertical releasing incisors. However, this author finds that an envelope flap is sufficient in virtually all cases. Following flap creation, buccal bone is removed. Next, the tooth is sectioned and the extraction proceeds as for single rooted teeth

Maxillary Canine

Maxillary canines are a very challenging extraction due to the significant length of the root. In addition, the very thin (less than 1-mm) plate of bone between the root and the nasal cavity often results in the creation of an oronasal fistula.

Vertical incisions are usually necessary for exposure and closure. At least a distal incision should be performed, and performing a mesial and distal incision will allow for increased tissue for closure.

The distal releasing incision is typically created at the mesial line angle of the first premolar. An exception exists if the first premolar is very close to the canine. In this case, carrying the horizontal component to the mesial line angle of the second premolar is recommended. This

is to allow sufficient exposure for bone removal, as the root curves back to over the second premolar.

If a mesial incision is performed, it should be in the diastema between the canine and third incisor. Classically it was made at the line angle of the canine or third incisor. However, in this author's opinion, the mesial line angle of the canine does not allow sufficient exposure and there is no reason to risk damaging the third incisor and increase surgical trauma. It is critical to fully incise the interdental gingiva to avoid tearing the flap. This is particularly challenging in the area mesial to the canine. Make sure to cut all the way to the bone. Following the creation of the vertical incisions, the flap is carefully elevated. If it is not elevating fairly easily, ensure that the interdental tissue is fully incised.

Once the flap is raised, approximately ½ of the buccal bone is removed. Make sure to remove some of the mesial and distal bone as the tooth widens just under the alveolar margin.

After the bone removal, elevate the tooth carefully. Do not torque the crown too much bucally as this will lever the apex into the nasal cavity. Once the tooth is elevated to a point of being very loose, it can be carefully extracted with forceps. The bone is then smoothed with a coarse diamond bur.

Closure is initiated with fenestration of the periosteum. When this is performed the tissue should stay in position over the defect. If it does not, tension is present and the flap will dehisce. It is critically important to relieve all tension if an oronasal fistula is present. Close the flap starting at the corners to avoid having to start over if it does not close correctly. Mandibular canine

These are quite simply the most difficult extraction in veterinary dentistry. This is due to the length and curve of the root, the hardness of the mandible, and the minimal bone near the apex. Furthermore, extraction of this tooth will greatly weaken the jaw and further predispose the patient to an iatrogenic fracture either during or after surgery. This tooth often holds the tongue in, and therefore it is not uncommon for the tongue to hang out following the extraction. Finally, the patient loses the function of the tooth. Therefore, it is strongly recommended to avoid extraction of this tooth. Referral for root canal therapy is a much better solution, if possible.

Some authors recommend a lingual approach to this extraction since less bone needs to be removed as to tooth curves lingual apically. However, this author prefers the standard buccal approach. This is because superior exposure is afforded and the flexible buccal mucosa allows for easier closure.

The flap for this extraction is generally triangular with just one distal vertical flap. A horizontal incision is created along the arcade to the mesial line angle of the first premolar. Then a distally divergent vertical incision is created. Next, the flap is carefully elevated and the buccal bone is removed to a point about 1/3 of the way down the root. More bone can be removed if necessary, but be careful with creating a larger flap or taking more bone as the mental nerve and artery exit approximately 3/4 of the way down the root. The tooth is then carefully elevated and extracted. Debridement and closure is as above.

Extraction of retained roots

Root fracture is a very common problem in veterinary dentistry. While it seems that removal of retained root tips is a daunting task, with proper technique and training it can be fairly straightforward. The first step is to create a gingival flap. Depending on the anticipated amount of exposure necessary to retrieve the fragments, this can either be an envelope flap or a full flap with one or two vertical releasing incisions.

Following flap creation, buccal cortical bone is removed with a carbide bur to a point somewhat below the most coronal aspect of the remaining root. If necessary, the bone can be removed 360 degrees around the tooth, but this author tries to avoid this aggressive approach.

Once the root(s) can be visualized, careful elevation with small, sharp elevators is initiated. Once the tooth is mobile, it can be extracted normally. After radiographic confirmation that the tooth is fully extracted, the bone is smoothed and the defect closed.

Oronasal fistula repair

In most cases, the single layer mucogingival flap technique is sufficient to repair ONFs, especially when done correctly the first time. This is the most common surgical treatment used to repair ONFs and therefore will be presented here.

The single layer mucogingival flap is created with either one or two vertical incisions. Depending on the size and location of the fistula as well as presence of the offending tooth, a horizontal interdental incision may also be necessary for successful repair. Proper design of the mucogingival flap will allow maximum exposure of the area for extraction of the tooth (if necessary), debridement of the fistula, and critically important tension-free closure.

Incisions are created with a number 15 or 11 scalpel blade. As described previously, the vertical incision(s) were classically started at the line angle of the teeth. A line angle is a theoretic corner of a tooth. When repairing an ONF associated with a maxillary canine tooth, the distal incision is made at the mesial line angle of the first premolar, and the mesial incision is started at the mesial line angle of the canine (if present). However, it is not necessary to cut over to a line angle if there is a

diastema. If the tooth is already absent, the incisions are made at the mesial and distal edges of the fistula.

When making flap incisions, adequate pressure should be placed to ensure full thickness of the soft tissue is incised down to the bone. Any vertical incisions should be created slightly divergent as they proceed apically. Divergent incisions allow for adequate blood supply for the newly created pedicle flap. It is important to choose the location of the incisions to ensure that sutured margins will have adequate bony support and will not lie over a defect.

The mucogingival flap is gently elevated off the bone using a periosteal elevator.

Approximately 2-3mm of palatal mucosa is also gently elevated/lifted off the palatal bone so that fresh epithelial edges are created. Any margins of the flap associated with the oronasal fistula should be debrided using a LaGrange scissors or coarse diamond bur to remove 1-2mm of tissue, leaving fresh epithelial edges.

A coarse diamond bur on a high-speed handpiece is used to smooth the edges of the remaining maxillary bone (if necessary) and to remove any epithelial remnants between the fistula and the nasal cavity.

As with any closure in the oral cavity, the key to success is to ensure there is no tension on the incision line. Fenestration of the inelastic periosteum (see previous section on surgical extractions) is performed to increase the mobility of the flap and allow for a tension free closure. This is accomplished by a combination of sharp and blunt dissection with a LaGrange scissors to ensure the overlying mucosa is not damaged.

The gingival flap is then placed over the defect so that it remains in position without being held. Once this is accomplished (i.e. no tension is present), the flap is ready to be sutured into place.

Placing a subcuticular layer can improve the chances of healing. A few buried horizontal mattress sutures will help maintain the flap as well as smooth out the incision line. Finally, this layer cannot be licked out by the patient.

Closure is performed as described in previous sections, with the initial sutures placed at the corners of the flap. This will avoid having to resuture the flap if it does not align correctly. This is not necessary if a subcuticular layer has been placed.

The remainder of the flap is then sutured over the defect in a simple interrupted pattern every 2-3 mm using an absorbable suture material.

USING THE WHOLE CLINIC TEAM TO DELIVER COST-EFFECTIVE CARE

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Identifying the opportunities

The key to providing cost effective healthcare is to ensure that a clinic's principle asset, our veterinary surgeons, are being used productively and supported appropriately. In many clinics experience has shown that veterinary surgeons can spend over half of their time carrying out tasks and duties that could be performed by less qualified, and hence expensive, members of the team.

There are many reasons why this situation occurs, however the most common reason I am given is that it is just the way we do things here! So how can you tell if your clinic is using the whole clinic team effectively? The simplest way I have found is to ask them. This can be achieved by using a simple survey, and/or using individual confidential interviews. Often using an independent person for this task encourages staff to be more open and honest about their feelings, so long as they are made to feel secure that their comments will be treated in confidence.

Key facts to establish include;

How well do the staff understand the aims, objectives and values of their clinic?

Who do staff go to when they have an issue to resolve?

Do staff feel that all of their skills are fully utilised?

Are staff being developed in way that is consistent with the clinic's goals?

What do staff most enjoy about working at the clinic?

What do they least like, and would change tomorrow if they could?

The barriers to good team-working

The biggest barriers to effective team-working are firstly that staff are unsure as to exactly what their role allows them to do, and secondly, that they do not know either what to say, or have the confidence to say it even when they do.

Often both of these situations have arisen because of the action of either the owners or veterinary surgeons when there have been issues arising out of staff doing or saying "the wrong thing" in the past. Without support and encouragement staff will not take "risks" and so absolve themselves from the situation.

The key steps to breaking down these barriers are to ensure all staff roles have been clarified, to provide support and encouragement to staff to develop and use new skills, and to develop best practice protocols and procedures that can be used to train all staff, provide a reference point when required, and to ensure consistency of service.

Protocols and procedures

Protocols and procedures are important because they ensure we provide a consistent message, and provide the means to delivering a consistent service regardless of which members of staff are involved.

The process of creating or updating a protocol should involve as large a group as practicable so as to draw upon the collective knowledge and experience of the team. We also know that veterinary surgeons can hold strong views, and it is important to reach a consensus if we are to develop protocols that have the support of the whole clinic team. Involving staff in the process is an effective way of locking them into the outcome.

Of course, even for the most common preventative treatments, there are many options, and chosing the right one will require us to understand the patient's circumstances. As such, protocols need to identify the key questions that staff has to ask to establish the lifestyle and risk factors that will be used to determine the most appropriate solution for that pet. Used correctly, good protocols should ensure that whichever member of staff asks the questions, based on the same answers the resulting recommendation will be the same.

Once developed, effective protocols allow us to share best practice across the whole clinic team, and provide the training documentation for new team members.

Key preventative healthcare protocols include Vaccination policy (based on local risks), Parasite control policy (based on local risk), and Neutering policy (by species and breed)

Engaging staff

The most effective way to engage staff is to define areas of responsibility, either collectively or individually, and to involve them in improving performance in these areas.

This provides lots of opportunities to draw on their experiences, to allow them to "own" solutions, to support each other, and for us to praise.

Two activities that lend themselves to this approach are running Clubs or Clinics, and managing and following up on reminders or recalls.

Clubs and clinics are an excellent tool to encourage staff to engage in a team approach because just the act of creating them reinforces the importance attached to working together. They also allow the more experienced members of staff to make better use of existing skills and to pass on their experience to more junior team members. Popular Clubs and clinics include Puppy and kitten socialisation and advice, Weight control, Senior pet clubs and for Behaviour issues.

Developing and maintaining the clinic's systems for Reminders and Recalls is an excellent way to engage less clinical staff such as Receptionists and Administrators. These staff have often joined a clinic specifically because of their affinity to animals, and so following up on reminders or recalls is an effective way for them to make a positive contribution to the health of our patients

Your action plan

Developing a team approach is about much more than just saying it is something we intend to do. If your clinic has not historically used staff in this way, then the level of cultural change required can be considerable. You will need to demonstrate your good intentions in practice before long serving staff begin to feel comfortable with the new process and fully engage. You will need to plan carefully, and take small steps. What the first step will look like is very much dependant on where you are now, which is why you should always start with a review.

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WSV18-0300

HOW I TREAT GIARDIASIS

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HOW I TREAT GIARDIASIS

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Giardia duodenalis is one of the most commonly identified gastrointestinal protozoal parasites in dogs, cats, and people worldwide. Despite the common nature of this infection, much confusion remains about the clinical significance of **Giardia** infections, the zoonotic nature of this parasite, and the diagnostic, treatment, and management strategies that are best suited to control the infection. One reason for the confusion is the fact that **Giardia** populations exist in different assemblages which vary in their infectivity for animals and humans. The total number of **Giardia** strains and the full range of host infectivity is unknown, but some patterns have been recognized[1]. For example, dog strains are not known to infect cats, and cat strains are not known to infect dogs. Similarly, human infections appear to be primarily acquired from other humans, and transmission from dogs or cats to humans, while still of great research interest, appears to be relatively uncommon.

For all **Giardia** isolates, cysts which are immediately infective when shed are passed into the environment in feces. Transmission to the next host occurs upon ingestion of cysts from fecal-contaminated water, food, or fomites or through self-grooming. Trophozoites may also be passed in feces but do not establish an infection in the next host. In dogs and cats, the greatest risk for infection leading to clinical disease appears to be in young animals — puppies and kittens — that have not yet acquired immunity. Dogs and cats frequently have subclinical **Giardia** infections but show no signs of disease; many parasitologists and gastroenterologists do not recommend treatment of these infected yet clinically normal patients unless there is specific concern about environmental contamination with cysts creating a risk to other animals in the facility.

When disease does develop, diarrhea is the most common clinical sign and is thought to occur due to trophozoites attaching to enterocytes in the small intestine leading to malabsorption and maldigestion. Diagnosis of **Giardia** may be achieved by identifying trophozoites on a direct smear or wet mount of diarrheic feces, or by identifying cysts in a fecal smear using a fluorescent antibody assay or on fecal flotation with centrifugation. Zinc sulfate (S.G. 1.18) is preferred for fecal flotation as it is less likely to collapse the cysts and prevent their recovery. Fecal antigen tests are also widely available and detect cyst antigen in fecal samples. Cysts can be shed intermittently, and repeat testing performed over several days may be necessary to identify infection even when all available methods are used. Advisory groups like the Companion Animal Parasite Council (CAPC) recommend only testing symptomatic dogs and cats with the fecal ELISA[2]. Many clinically normal animals will test positive for cyst antigen but are unlikely to need or benefit from treatment.

In many areas of the world, label-approved drugs to treat **Giardia** in dogs and cats may not be available. However, several effective treatments have been reported. Effective treatments for canine and feline giardiasis include fenbendazole (50 mg/kg PO q 24 hrs x 5-7 days), febantel (30 mg/kg x 3 days) (usually combined with praziquantel and pyrantel pamoate), and ronidazole (30-50 mg/kg PO q12 hrs x 7 days)[3-6]. Metronidazole was used historically to treat giardiasis but has poor efficacy; metronidazole (25 mg/kg PO q 12 hrs x 5-7 days) in combination with fenbendazole is considered more likely to be effective[2]. Safety concerns exist with both ronidazole and metronidazole. Regardless of the treatment selected, intensive hygiene management including disinfection of kennels and frequent bathing is often necessary to prevent reinfection and continued cyst shedding[3].

When an initial course of fenbendazole (5 days) fails to eliminate clinical disease, a second, longer course (10-14 days) should be implemented and combined with strict attention to hygiene to prevent continued re-infection. Bathing, with particular attention paid to the hind quarters to remove any fecal debris and associated cysts, is important. If acceptable to the owner, clipping the hair in the perianal area and around the tail and legs may facilitate keeping the area clean and thus prevent re-infections from self-grooming. Discussion with the owner about pet lifestyle habits can also be helpful; dogs that often visit dog parks or swim in areas frequented by other dogs are at particular risk for re-infection.

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WSV18-0326

SVA PRACTICE MANAGEMENT

ONLINE REPUTATION, WHY IT MATTERS SO MUCH AND HOW TO TAKE CONTROL

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SS – Online Reputation, why it matters so much and how to take control

Social media and online reviews bring huge opportunity to vet practices but also some risk. A disgruntled client can ensure their story is heard by thousands of people and an inadvertent post from a member of staff can get the practice into legal or reputational trouble. Take control of your online reputation and make sure you have a plan in place for what to do if the worst happens.

Reputation management is a crucial part of online marketing. It is useful to think of four main steps:

1. Monitoring
2. Positive Content
3. Responding
4. Social Media Policy

Monitoring

To be able to react to mentions of your name on the internet it is crucial to know what is being said. Setting up monitoring can be done very quickly and easily using free tools such as Google Alerts.

Positive Content

This strategy for reputation management is very similar to the old veterinary adage 'The solution to pollution is dilution'. Building up positive content about your practice allows viewers to put any negative content into context. For example, one negative review in isolation can be very damaging for a veterinary practice, but one negative review alongside several positive ones poses no problem and can actually give weight to the positive reviews showing that they are authentic.

Responding

It is very important to respond to all feedback good or bad. It is useful to have a process in place for dealing with negative content so that should the worst happen everybody knows what to do and how to respond.

Social Media Policy

Risk from online reputational damage comes not only from clients but also from the veterinary practice's own staff if they do not understand their online obligations, both on their practice's social platforms and their own, private social media. A practice social media policy is not only essential for good HR management but also it is a great way to get the whole team involved and familiar with the challenges and issues involved.

WSV18-0120

ORTHOPEDIC SURGERY

PATELLAR LUXATION – WHEN TO & HOW TO BALANCE SOFT TISSUE TENSION

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The key to successful management of patellar luxations is to identify and correct each of the factors contributing to mal-tracking of the patella in the specific patient being treated. No single treatment nor collection of treatments will be consistently effective in all patients. Bony contributions to patellar luxation may include a shallow and/or shortened trochlear sulcus, a displaced tibial tubercle or other skeletal malformations. Similarly, chronic and persistent patellar luxation (grade 3 and grade 4 luxation) tend to distort the proper balance of soft tissue tension surrounding the patella. Often, especially with grade 3 and grade 4 luxations, soft tissue reconstructions are required to balance the tension in the soft tissues that surround the patella.

Soft Tissue Reconstructionsinclude capsular/retinacular/ muscular release, imbrications, and anti-rotation sutures. Soft tissue reconstructions, by themselves, will not correct bony conformational abnormalities. Soft tissue reconstructions are most commonly performed in conjunction with skeletal reconstructions. Release of thickened and contracted medial joint capsule/ retinaculum is achieved by their incision from the tibial plateau to the suprapatellar recess. In most grade 4 and some grade 3 MPL cases, the quadriceps muscle group is medially displaced (Fig 1) and must be elevated from the suprapatellar region to the proximal femur, being careful to protect the descending genicular vessels (this, alone, is a good reason to refer these cases to a trained orthopedic surgeon). The pes anserinus muscle group (sartorius, gracilis, and semi-tendinosus muscles) can be released by elevation of their insertions on the medial aspect of the proximal tibia if their tension is causing internal rotation of the stifle. Stretched lateral joint capsule/retinaculum often need to be tightened to achieve balanced soft tension upon the patella. In grade 4 MPL cases, redundant joint capsule lateral to the patella must usually be excised. Reconstruction of the lateral joint capsule helps to properly balance the tension in the peri-patellar tissues. Extracapsular lateral fabello-tibial anti-rotation sutures can be placed to limit excessive stifle rotation and are particularly beneficial in dogs with combined MPL and cranial cruciate ligament rupture. The tibial anchor point of the extracapsular suture can either be to paired bone tunnels at the level of the extensor sulcus of the tibia (adjacent to the long digital extensor) or to a pre-formed eyelet in a tibial tension band wire.

WSV18-0237

VECCS

USE OF TFAST AND AFAST IN EMERGENCY PATIENTS

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The use of ultrasound has developed over the past 50 years as an indispensable first-line diagnostic tool for traumatic patients and cardiac/respiratory evaluation of symptomatic patients. AFAST focused examination of four sites in the abdomen designed to evaluate presence of free abdominal fluid, while TFAST is used to detect the presence of air or fluid in the pleural and pericardial space. The Blue VET (beside lung evaluation) was developed in as emergency lung ultrasound to detect interstitial- alveolar lung injury.

AFAST- Abdominal Focused Assessment with Sonography for Trauma

There are 2 applications of AFAST in dogs experiencing abdominal trauma:

1. AFAST- focused examination of four sites in the abdomen designed to quickly (5 minutes) to rule in or rule out the presence of free abdominal fluid (typically indicative of hemorrhage) in 4 quadrants of the abdomen. (figure 1) FAST examinations are very sensitive and specific for detection of free abdominal fluid, even when performed by non-radiologists, but are less sensitive at detecting intra-abdominal injury following penetrating trauma than following blunt trauma.

(Boyen & Rozanski JAVMA 2004).

2. Serial AFAST (Lisciandro et al, JVECC 2009) - Serial AFAST examinations was designed to detect changes in the quantity of fluid present in the abdomen over time, especially when combined with determination of abdominal fluid score (AFS). Serial AFAST examinations should be performed every 2 - 4 hours, or more frequently as dictated by clinical findings. Some dogs with negative results on the initial AFAST examination had positive findings on serial AFAST examinations. Abdominal fluid score- semiquantitative evaluation of the degree of free fluid (typically hemorrhage) performed by recording the number of sites among the four standard views. Animals with an AFS of 0, 1, 2, 3, and 4 would show negative findings at all sites, positive results at one site, positive findings at any two sites, positive results at any three sites, and positive findings at all four sites, respectively. The study indicate that AFS of 3&4 is associated with higher risk of anemia, blood transfusion and increased alanine aminotransferase (PCV<25%) (Lisciandro et al, JVECC 2009).

How to perform AFAST technique?

Free fluid tends to accumulate in the most dependent areas of the abdomen. The examination can be completed without clipping and application of alcohol. Can be performed in right or left lateral recumbency. To detect fluid in the abdomen, ultrasonographic views (transverse and longitudinal) are obtained at each of 4 sites; just caudal to the xiphoid process, on the midline over the urinary bladder, and at the left and right flank regions (Figure 1). The examiner may choose right lateral if volume status of the patient is to be evaluated ultrasonography or if the left retroperitoneal space is to be evaluated. Left lateral recumbency may be preferred if right retroperitoneal space is to be evaluated. The transducer is centered at one of four locations and then moved at least 4 cm and fanned through at least 45 degrees in a cranial, caudal, left, and right direction. The examination can be accomplished using only the longitudinal view at each site, although adding the transverse view is helpful if results of the longitudinal view are equivocal.

The four sites are:

1. **D**iaphragmatico- **H**epatic: just caudal to the xiphoid. Can also evaluate the thoracic cavity (pleaural and pericardial cavities)

2. **C**ysto- **C**olic: midline site over the bladder

3. **H**epato- **R**enal: right flank site

4. **S**pleno- **R**enal: left flank site

Figure 1
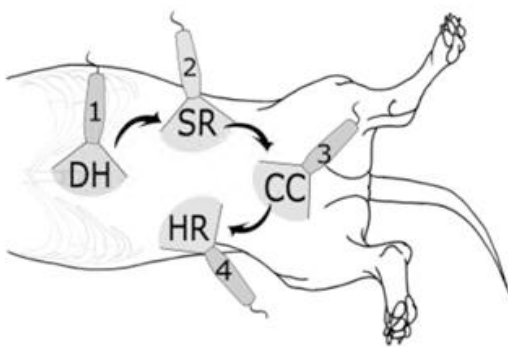
TFAST-Thoracic Focused Assessment with Sonography for Trauma

TFAST are designed to RI/RO the presence of air or fluid in the pleural space, and RI/RO the presence of fluid in the pericardial space. In the normal lung movement of the pleural line on each other create the normal Glide sign indicating a normal apposition of the lung against the thoracic wall: a dynamic finding. It may be intermittent with low respiratory rates or apnea. When the gliding is lacking and and there is step sign it may be indicative to

the presence of air in the pleural space.
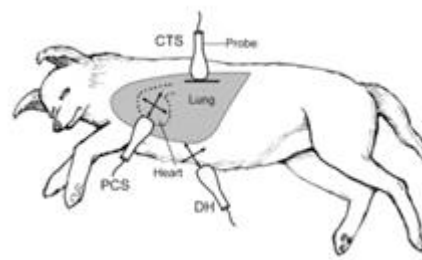
Lisciandro et al, JVECC 2008 showed that TFAST has 78% sensitivity and 93% specificity for detecting pneumothorax. In contrast to AFAST this technique need more experienced (non radiologist) emergency doctor. In that study there were considerable differences in sensitivity when performed by an experienced sonographer (95% sensitivity with >70 scans) compared with a unexpirienced sonographer (45% sensitivity with <15 scans). "Gliding sign" should be detected in order to diagnose pneumothorax. Despite variation, the negative predicative value of the TFAST examination is high, which indicates that the presence of the glide sign essentially rules out pneumothorax. However, failure to detect a glide sign may result from conditions other than pneumothorax.

TFAST technique:

TFAST can be performed in left, right, or sternal recumbency, and involves five views:

1. Chest tube site [CTS] view- one on each side of the thorax in longitudinal plane perpendicular (horizontally) to the ribs in the dorsal third, between the 7-9 intercostal spaces. Used to confirm or rule out pneumothorax. The sonographer must keep the transducer immobile on the chest wall to maximize the chance of detecting the glide sign.

2. Pericardial chest site [PCS] view- one on each side of the chest in the longitudinal and transverse planes, with movement and fanning of the transducer, between the 5-6 intercostal spaces.

3. Diaphragmtaico- hepatic view of AFAST.

In the normal lung there will so called the alligator sign or Bat sign: The pleural- pulmonary interface (PP line) is the roughly horizontal white line running between the two ribs and is usually visible just distal to the ribs (with exception of subcutaneous emphysema).

Lung point: The severity of pneumothorax can be evaluated by moving the probe in a dorsal to ventral direction. The point at which the glide sign returns is known as the lung point in which the step sign will be presented. This is a very specific finding and confirms the presence of pneumothorax. In the case of massive pneumothorax a lung point will not be detected.

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Step sign: glide sign deviates from its normal continuity. Suggests thoracic trauma, or in non- trauma patients, lung consolidation/ masses.

So in general we can say that:

Pneumothorax is ruled out if a glide sign and/or B-lines are detected;

Pneumothorax is strongly suspected when glide sign and B-lines are absent.

Pneumothorax is definitively confirmed if lung point is identified.

Emergency lung ultrasound to detect interstitial-alveolar lung injury:

BLUE (bedside lung evaluation): the key concept in this setting is the detection of B-lines that originate from the pleural line. The proximity and number or density of B-lines correlate with extravascular lung water, and the anatomic location of B-lines with respect to lung anatomy correlates with different pathologic lung conditions. Emergency lung ultrasound may add a particular niche in differentiating underlying causes of respiratory distress in the severely dyspneic patient that is in too unstable a condition to allow thoracic radiography.

Vet Blue is performed from both sides of the chest wall with 4 points in each side.

- Caudo dorsal lung view (Cd) similar to the CTS view in TFAST
- Perihilar lung region (Ph)- caudorsal 6-7 intercostal space
- Middle lung lobe (Md)- at 4-5 intercostal space
- Cranial lung region (Cr)- 3-4 intercostal space.

What signs and lines we can see in VET BLUE;

A-lines: a result of reverberation artifact causes the pleural line to be replicated in the sonographic far field. The distance between subsequent A-lines corresponds to the same distance between the skin surface and the parietal pleura. A-lines should be seen in the normal lungs, but also may be seen in patients with pneumothorax. A-lines should not be confused with the pleural line.

B-lines: (ring-down/ comet tail/ lung rockets) are reverberation artifacts originating from the visceral pleura lines appear as hyperechoic vertical lines extending from the pleural line to the edge of the far field image, passing through A-lines without fading. B-lines move in a to-and-fro fashion (pendulum) with inspiration and expiration, and are synchronous with the glide sign. An occasional B-line is considered normal; excessive B-lines are indicative of interstitial-alveolar lung abnormality. When B-lines are numerous they called a B-pattern.

Lung curtain- the lung artifact created by movement of the lungs at the costo-phrenic angles creates a vertical sliding artifact similar to the opening and closing of a theater curtain, which should not be confused with the glide sign. The ultrasound probe should be moved cranially if the curtain sign is noted.

Summary

A glide sign with A lines (horizontal lines) is considered a normal lung or "Dry Lung" only to be confounded with the pneumothorax (dry lung with absent glide sign). When B lines or so called lung rockets are presented the lung will be defined as Wet Lung meaning parenchyma lung abnormality (e.g lung edema, contusion and bleeding, pulmonary masses, lung infiltaration).

Here are some examples from Liscando et al.

WSV18-0215

SVA FELINE

FELINE TRACHEOBRONCHOSCOPY: WHY, WHEN, WHAT AND HOW?

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Laryngoscopy, tracheoscopy and bronchoscopy (the last two collectively called 'tracheobronchoscopy') are considered valuable procedures for investigating causes of feline lower airway disease. Airway endoscopy is indicated in those patients presenting with stridor, dysphonia, acute or chronic cough, inspiratory or expiratory dyspnea, haemoptysis, and/or unexplained radiographic infilltrates (focal, diffuse, lobar, bronchial, alveolar or consolidation) which have not been diagnosed by other means.[1-4]

Despite the potential risks associated with lower airway endoscopy, it can be performed safely in the vast majority of cases. When performed in favourable and controlled conditions, tracheobronchoscopy can still be considered a relatively safe and reliable procedure for the diagnosis, the treatment of respiratory tract disease (e.g. removal of tracheal foreign body) and suction of freshly aspirated materials.[4]

Indications/contraindications

It's prudent for the feline practitioner to appreciate that lower airway endoscopy should not be considered to be the first ancillary procedure in these patients. Rather, the suspicion of diseases and the real need for endoscopy to reach the proper diagnosis should only be performed after collecting a full clinical history and performing a full clinical examination, including response to previous treatments (antibiotics, anthelmintics, bronchodilators, etc.). Further tests could include haematology, biochemistry, D. immitis Ab/Ag testing, faecal parasitology and thoracic radiographs. Lower airway endoscopy may then become irrelevant, when radiographic findings include the presence of metastases, mediastinal mass, suspicion of cardiac diseases, pleural disease or diaphragmatic hernia.

Laryngeal examination is indicated for cats presenting with airway signs such as dysphonia, stridor or inspiratory/expiratory dyspnoea. Laryngoscopy can be performed easily and should always be incorporated as a routine part of any upper or lower airway endoscopic examination. The differentiation between laryngeal problems and lower airway disease based solely on history and clinical examination is not always obvious in cats. Laryngeal disorders can induce some coughing, but as soon as the laryngeal inlet is reduced, the main signs are dyspnoea and stridor. In cats, the most common laryngeal disorder is laryngeal oedema; tumours and paralysis are less common.[4]

Tracheoscopy is rarely performed on its own as such; however, when performing tracheobronchoscopy the trachea will always be investigated as part of a lower airway endoscopic examination. Tracheoscopy alone would be indicated when a tracheal disorder is suspected based on clinical signs and confirmed on imaging. When tracheal compression or displacement is shown to be due to the presence of an extra- or intrathoracic mass, especially with a mediastinal space-occuying lesion/mass, endoscopy would not be helpful for the diagnosis nor choice of treatment.

Bronchoscopy should be considered in those cases of suspected (or confirmed) lesions of the mainstem bronchi, segmental bronchioles/airways (e.g FB, neoplasia) and/or for visually-guided intervention and airway sampling. Severe cardiac arrhythmia, heart failure or severe hypoxia are contraindications to tracheobronchoscopy.

Patient preparation and anaesthesia

Prior to proceeding with tracheobronchoscopy, the endoscopist should be well versed in normal anatomy and appearance of the feline airways as this will allow for rapid and efficient evaluation, and collection of samples.[5] The rapidity of the procedure when performed by an experienced endoscopist is one of the key factors in a successful outcome.

Lower airway endoscopy requires general anaesthesia and is, therefore, considered medium-to-high risk in many feline respiratory cases due to the relatively small and higher airway responsiveness of the cat compared to that of the dog. Moreover, as soon as a cat presents as a dyspnoeic patient, the procedure is considered even more of a potential risk of acute respiratory embarrassment during induction, maintenance and recovery phases of general anaesthesia.

Equipment

For cats, a small diameter (e.g. 3–4 mm), fexible

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fibreoptic or video bronchoscope is preferred over rigid endoscopes. While rigid endoscopes can be used for tracheoscopy, they are much less useful in feline practice as they do not allow examination beyond the carina and there is also risk of airway perforation in the hands of inexperienced operators. Flexible bronchoscopes will also have a 'multi-use' biopsy channel in which an adapter can be attached to allow for concurrent oxygen administration, passing biopsy forceps and saline for BALs. Bronchoscopes will usually only have two-way distal tip deflection (up and down). Ancillary equipment used in bronchoscopy includes cytology brushes/aspiration catheters, foreign body retrieval forceps, transbronchial aspiration needles and biopsy forceps.

Often, animals that are candidates for bronchoscopy have compromised respiratory function. There is no such 'one size fits all' anaesthetic protocol and each case needs to be treated individually. Most cases tolerate a low-dose acepromazine (0.01 mg/kg), an opioid (e.g. buprenorphine 0.02 mg/kg) and bronchodilator (e.g. terbutaline 1 mg/ml, 0.01 mg/kg IV, IM or SQ), the latter to help reduce bronchospasm and improve oxygenation. General anaesthesia is most easily maintained by total intravenous anaesthesia (TIVA) with incremental doses of propofol or alfaxalone, In cats, due to the sensitive nature of the larynx, topical anaesthesia with 1% lidocaine spray is required.

Technique
The bronchoscope should be sterilised before use, either by cold sterilisation in 2% glutaraldehyde solution such as Cidex (Johnson & Johnson) or Med-DisTM (Medichem International Ltd), F10 or using ethylene oxide gas sterilisation. If cold sterilisation is used, the instrument must be rinsed and channel flushed thoroughly with sterile water before use.

It's essential for the operator to be familiar with endobronchial anatomy due to the limited amount of examination time spent within the airways between 're-oxygenation' phases. Common examination method is to evaluate the larynx, trachea, entire right side, then entire left side in a standard order. Following a 'roadmap' will help with endobronchial orientation and ensures a more complete investigation of each lung lobe.[5]

The larynx (both structure and function) should be under a very light plane of anaesthesia. It is vital to have an assistant 'announce' phase of respiration to ascertain appropriate abduction on inspiration and adduction on expiration. Unfortunately, cats have airways that are too narrow to allow for the passage of a bronchoscope through a T-adaptor and endotracheal tube. Instead, the bronchoscope is usually advanced directly into the airway under direct visualisation.

The trachea should then be examined down to the level of the carina. It should contain no mucus/foam and should be a uniform pink colour with a smooth wall with easily visible submucosal capillary complexes.

The carina marks the bifurcation of the trachea into the left and right main stem bronchi. The right main stem bronchus is usually straight ahead of the bronchoscope and the left main stem bronchi usually require some manoeuvring to the operator's right to facilitate entry. For this reason, airway foreign bodies (especially grass blades) are more commonly seen in the right main stem bronchi. Each segmental airway should be evaluated for changes in colour, shape, size, and signs of 'bubbling', purulent discharge, excessive mucus, or blood. If the operator becomes 'lost', the bronchoscope is retracted back to the carina (landmark) to re-establish position.

Airway sampling/bronchioalveolar lavage
Once the airways have been thoroughly evaluated, samples should be obtained for cytology and microbiology as gross changes are not pathognomonic for specific disease. Airway sampling is best achieved via directed BAL. Samples are obtained by 'wedging' the tip of the bronchoscope within a terminal bronchus. Warmed sterile saline is flushed through the biopsy port of the bronchoscope and immediately aspirated. BAL samples should be obtained from at least two sites (usually left and right side) as well as any focal abnormalities.[4]

The retrieved saline is submitted for both cytology, bacterial culture /sensitivity and mycoplasma PCR.

Postoperative management
The patient is intubated immediately post-procedure and maintained on 100% oxygen until stable and recovered from anaesthesia. It's essential that patients are closely monitored on recovery and supplemental oxygen administered, either by flow-by or O2 tent, as required.

Postoperative complications

The most significant complication encountered post-procedure is upper airway obstruction. This may be due to laryngeal oedema, inflammation and swelling resulting from biopsy or pre-existing lesion, accumulation of mucus/airway secretions or bronchococonstriction. In cases where upper airway swelling/inflammation is anticipated, intravenous dexamethasone may be administered IV (0.05–0.2 mg/kg) before extubation. Additional doses of terbutaline may also be considered to counteract bronchoconstriction.

Conclusion

Bronchoscopy is generally considered a safe procedure, provided there is use of correct equipment for patient size, adequate patient risk assessment, close anaesthetic monitoring and adequate operator experience.

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WSV18-0038

NUTRITION AND DIAGNOSTIC IMAGING (LECTURES GIVEN IN MANDARIN CHINESE)

NEW SONOGRAPHY TECHNOLOGY IN VASCULAR IMAGING

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MEASUREMENT IN ULTRASONOGRAPHY: DOES IT MATTER?

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Organ **size**estimation using ultrasound is subjective. Interpretation of the ultrasound examination based on size alone is not advisable. When there is suspected change in the size of a certain organ, it should be interpreted with other signs of disease such as change of echogenicity and presence of clinical signs. The important practical aspect of size measurement in ultrasound is for future comparison in follow up examinations. This could be useful to determine whether there is a progression of disease or response to treatment. There is no reliable way to measure the size of the liver. Rounding of the margins and excessive caudal extension of the liver suggest hepatomegaly. A small liver is suspected when there is poor visualization of the liver. The size of the spleen in dogs and cats varies depending on many factors. Sonographic determination of splenomegaly is often subjective. A more reliable way to measure the size of spleen is through radiography. In cats, the spleen is always small and an enlargement of the spleen is easier to recognize. Although a positive correlation of kidney length and volume with body weight has been reported, it is not widely used clinically due to the wide range of standard deviation. However, linear kidney measurement in cats is more reliable as there is less variation of the body size. It is more difficult to determine mild enlargement of the kidneys when there is no alteration of the echogenicity and echotexture. Slightly smaller kidney size is commonly seen in older dogs and cats due to chronic renal disease. A mild increased size of the renal pelvis has been reported to be normal, secondary to intravenous fluid administration, or associated with disease condition such as pyelonephritis and ureter obstruction. Marked dilatation of the renal pelvis (hydronephrosis) is due to obstruction of the ureter. The width of the pancreas in dogs and cats is 1-3 cm and 1 cm respectively. A detectable pancreatic change of echogenicity or contour

is usually accompanied with an increase in the size of the pancreas. Unfortunately, size change does not always mean that there is pancreatic pathology. The canine prostate size is variable. Its size increases with age and is small in neutered males. Although there are studies indicating prostate size estimation using ultrasound is reliable, it is still very subjective as differentiating between normal and diseased prostate is difficult.

The volume of the gallbladder and urinary bladder varies in both dogs and cats. Some of them appear large but have no clinical significance. The wall of the gallbladder should appear as a thin echogenic line in normal dogs and cats. Thickening and the presence of a measurable gallbladder wall most likely indicates edema or inflammation of the wall. The diameter of the common bile duct in dogs and cats is < 3mm and < 4mm respectively. Dilation of the common bile duct most likely indicates obstruction. The urinary bladder wall should be < 2.3 mm for dogs and < 1.7 mm for cats. Increased urinary bladder wall thickness should be interpreted with other changes and history. Thickness of the gastrointestinal tract in normal dogs and cats has been reported. Generally, the gastric thickness of dogs and cats should be less than 5mm and 3.6 mm respectively. The thickness of the small intestines in cats is more consistent and mostly < 2.5 mm. As for dogs, the thickness of the small intestines varies depending on the body size. However, it should be less than 6 mm for the duodenum and 5 mm for jejunum. The thickness of the wall of the colon is 2-3 mm in dogs and 1.4-2.5 mm in cats. Increased wall thickness without disruption of the five layer appearance of the gastrointestinal tract is usually due to inflammatory bowel disease except when there is increased thickness of the muscularis layer, which may indicate lymphosarcoma. A normal adrenal gland size has been reported in dogs. The maximal diameter of the adrenal gland has been found to be the most reliable indicator of its size. As a general rule, a diameter of 7.4 mm has been suggested to be used as the upper limits of normal. Unfortunately, overlapping in size between normal dogs and those with adrenal gland abnormality exist. Approximately 25% of dogs with pituitary dependent hyperadrenocorticism (PDH) do not have adrenal gland enlargement, and about 20% of dogs without any evidence of adrenal diseases had adrenal size more than the proposed upper limits of 7.4 mm. Enlargement of the canine adrenal glands is suggestive of PDH, neoplasia or nodular hyperplasia. In many instances, abnormalities of the adrenal glands lead to an increase in size alone without any nodules or masses. In cats, a study using 10 normal adult cats showed that the normal size to be 4.3 ±0.3 mm.

As a general rule, size measured should be interpreted with care, and should always correlate with clinical signs.

WSV18-0074

NURSES (HILLS)

CARDIOPULMONARY RESUSCITATION PRACTICAL FOR NURSES

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UPDATE ON CARDIOPULMONARY RESUSCITATION (CPR)

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In CPR time is of the essence and therefore it is important that all staff know what to do, how to do it and where everything is that is required in treating the patient. It is also important that staff know what is normal and what is abnormal in the patient. The emergency cart must be well stocked and checked regularly to ensure drugs and equipment is maintained. The definition of Cardiac Arrest is an abrupt cessation of spontaneous and effective ventilation and systemic perfusion (circulation) which leads to inadequate oxygen delivery to tissues.

In 2012 The Reassessment Campaign on Veterinary Resuscitation (RECOVER) was designed to systematically evaluate the evidence on the clinical practice of veterinary CPR with 2 overarching goals. First to devise clinical guidelines on how to best treat CPR in dogs and cats, and second to identify important knowledge gaps in veterinary CPR that need to be filled in order to improve the quality of recommendations, and thus the quality of patient care in the future. An Advisory board of experts in the field as well as over 80 Veterinarians were consulted throughout the process.

One of the findings in the guidelines was that delaying the start of CPR has shown significant reductions in survival to discharge and the neurological status of the patient. Agonal breaths can be misinterpreted as spontaneous breathing. In human studies less than 2% of patients on whom CPR is started when not completely confirmed are actually harmed as commonly the patients respond to the stimulation associated with CPR. If there is any doubt CPR should be started immediately.

Preparing for CPR

An organised, cohesive, knowledgeable and well trained team will lead to better outcomes in CPR. It was recognised that the factors that can affect the outcome of CPR are environmental and personnel factors. Environmental factors that may impact performance include well-designed, straightforward checklists; algorithm charts; cognitive aids; and well-stocked, easy to access, organized crash carts.

Personnel factors include high-level team member training, specific leadership training, and appropriate intervals of retraining and debriefing. Improved outcomes require consistent improvement in resuscitation education and the implementation of support systems to allow the streamlined delivery of CPR in the clinic setting. Above all else the Veterinary team should practice CPR in their clinic on a regular basis at least every 3 – 6 months so everyone knows what their role is and the techniques that will be used. This is simple to achieve by using a stuffed toy dog, cat or even teddy bear!

Crash Cart

An emergency box or "crash cart" is a vital part of emergency procedures as it contains all the vital equipment and drugs required to deal with an emergency in the one location. The emergency box can be as simple as a large tool box with the basic necessary items or a large storage cart on wheels with multiple draws. The emergency box should be located in the treatment area or surgery of the clinic and divided into sections of similar equipment.

On top of the cart would be all resuscitation equipment such as an Ambu bag, Pulse Oxmeter, oxygen supply, suction unit and ECG unit. The top draw could contain airway equipment such as Laryngoscope, endotracheal tubes, stylets, tracheostomy tubes, lubricating jelly, ties and swabs. The second draw could contain circulation equipment such as Intravenous catheters, tape, tourniquet, feeding tubes, swabs and fluids. The bottom draw could contain emergency drugs, syringes and needles. Carts may be divided into further sections with more drawers if available and to the specifications of the Veterinarians in the veterinary clinic.

Equipment and usage

• Ambu Bag

An Ambu bag is a self-inflating device that is used to provide artificial ventilation to the patient. An oxygen source is connected to the bag and the bag then attached to the endotracheal tube. Ambu bags are preferable to using an anaesthetic machine for assisted ventilations as there could be residual anaesthetic gases in the machine and the circuit must be closed. Using an Ambu bag also creates a shorter distance for the oxygen to travel to the patient.

• Oxygen Supply

100% oxygen is ideally required for assisted ventilations. This may be via an anaesthetic machine or from a direct oxygen source (such as an Ambu bag) with a flowmeter attached.

• Pulse Oximeter

A Pulse Oximeter is a vital piece of equipment in monitoring the percentage of arterial haemoglobin-oxygen saturation. This is important in assessing the need for supplemental oxygen and also when it is no longer required. It consists of an infrared sensor placed on the tongue, lip or paw to obtain a reading. Normal values are between 98 – 100%

• Electocardiogram (ECG)

An ECG may be used in advanced life support after basic life support has been established. An ECG shows the heart rate and rhythm and the presence of arrhythmias that may have developed and therefore appropriate therapy can be administered. Ventricular fibrillation is the most common arrhythmia and an Electrical Defibrillator if available may be used to help stimulate and regulate the electrical activity in the heart.

• Suction Unit

A suction unit may be used to clear the airway of fluids and debris which may include mucous, blood, vomit or saliva and hence aid in endotracheal intubation.

Other Equipment

Endotracheal Tubes, Stylets, Swabs & Ties, Lubricating jelly, IV Catheters, loaded syringes e.g saline, feeding tubes or urinary catheters, tracheostomy tubes, chest drains and 3 way stopcock taps, bandage material and tape, gloves, syringes and needles.

• Fluids

Fluid therapy is the primary method of circulatory support in the emergency patient. The goal of fluid therapy is to restore circulating volume, perfusion and oxygen to the tissues to a desired level (e.g. normal heart rate, mucous membrane colour and capillary refill time) without causing a volume overload. A volume overload could lead to pulmonary and cerebral oedema. The correct type of fluid and the rate of administration will depend on the patient's condition. Crystalloid fluids are isotonic fluids (being of equal osmotic strength) that pass readily through cell membranes. These fluids will help to balance out between the Extracellular space and Intracellular space. Hartman's and 0.9% Sodium Chloride are crystalloids and are given where there is a need to replace the extracellular fluid and for volume expansion. They are inexpensive and easily available. A downside to crystalloids however is that they do not remain in the vascular space for very long.

Colloids are also isotonic fluids but they contain higher molecular weight particles that will remain intravascular for a longer period of time. Therefore the volume of fluid required will be less. Examples of colloids are Voluven and 5% Dextrose. Hypertonic saline draws fluid from the

extracellular space into the intracellular space causing volume expansion. It is most commonly used in cases of hypovolaemic shock (decreased circulating volume) as it creates rapid volume expansion. It must always be followed by crystalloid fluid administration to restore equilibrium of the extracellular and intracellular fluid. An example of hypertonic saline is Dextrans. Mannitol is another fluid that may be used in the emergency situation. Mannitol is an osmotic diuretic and is used to treat cerebral oedema. It draws fluid from the extracellular space as hypertonic saline does. It helps by increasing cerebral blood flow and decreasing fluid and swelling of the brain therefore decreasing intracranial pressure. 50% Glucose should also be included in the Emergency cart for use in cases of Hypoglycaemia where the glucose level of the patient is extremely low. The rate of fluid administration will depend on the condition of the patient. In CPR fluid overload has been found to be a problem when there is circulatory compromise and the use of fluids will depend on the individual case. As a general guide slightly higher than maintenance rates at 5 – 10ml/kg/hr can be used however only if the patient is hypovolemic. Fluid may be given as a bolus in severe cases to rapidly restore circulating volume.

Therefore the crash cart should contain both 500ml and 1000ml bags of Hartman's, 0.9% Sodium Chloride and if possible Haemaccel or 5% Dextrose. Hypertonic saline, Mannitol and 50% Glucose in 500ml bags are required also. Infusion sets of 20 drops/ml and paediatric giving sets should also be included.

Emergency Drugs

Recommended route of administration for most emergency drugs is Intravenous (I/V) or Intra-Osseous for the fastest absorption rate however if these routes are not possible the Intra-Tracheal route can be used however there is not any evidence on optimal dose, volume or diluents. Any drugs administered during CPR via the IV route must be followed by a saline bolus of 20 – 30mls to ensure the full effect. The following drugs, doses and routes of administrations are guides only and your Veterinarian will decide on what may or may not be used and the dose and route of administration for each case.

Adrenalin 1:1000 1mg/ml

Dose rate of 0.01mg/kg intravenously or double dose rate if given intratracheal for two doses on every other cycle of basic life support. If further doses are required then the dose is increased to 0.1mg/kg. Administer on every other cycle of basic life support. Used in cases of Ventricular Asystole (No electrical activity present in the heart) and also in cases of severe bradycardia (slow heart rate). Adrenalin causes vasoconstriction and improves coronary and cerebral blood flow and venous return to the heart. It increases the heart rate

and the force of contractions of the heart. It is most commonly administered intravenously but can also be given intratracheal through a feeding tube placed down the endotracheal tube. The dose rate does need to be doubled by this route and may already be made up as a 1:10000 solution (9mls of saline to 1ml of 1:1000 adrenalin). Lower doses are recommended to start with as high does may cause ventricular fibrillation. It's effect is immediate if given intravenously.

Vasopressin

Dose rate of 0.3 micrograms/kg (1ml/25kg) IV given instead of Adrenalin for the first one or two doses then administer Adrenalin for subsequent doses. May be given Intra-tracheal at a dose rate 5 times the IV dose. Vasopressin is a nonadrenergic vasopresser that causes peripheral, coronary and renal vasoconstriction. This in turn increase cerebral and coronary blood flow and can be used instead of Adrenalin to similar effects. Current studies in human medicine do not find that Vasopressin is superior to use of Adrenalin however some studies have shown that it may be more effective in animals particularly those with severe hypovolaemia. The RECOVER guidelines recommend that Vasopressin is used for only the first or second doses and that Adrenalin is used thereafter.

Atropine Sulphate 0.6mg/ml

Dose rate of 0.02 – 0.05mg per kg intravenously, intramuscularly or

sub-cutaneously. Used in cases of bradycardia. It will increase the cardiac output and heart rate by blocking vagal stimulation in the heart. It is also used as an antidote in cases of organophosphate poisoning. Care must be taken not to overdose as this can cause sinus tachycardia. Its effect is at its peak 3 – 4 minutes after intravenous administration. Can be given intratracheal again at double the dose rate intravenously and should be followed with a few short breaths to enhance delivery into the patients system.

Lignocaine 2% 20mg/ml

Dose rate of 2 – 6 mg per kg equating to 1ml/10kg intravenously. Used in cases of severe tachycardia and ventricular fibrillation. It is a local anaesthetic and therefore suppresses the ventricular electrical activity of the heart. It can be given intratracheal at double the intravenous dose rate.

Frusemide (Furosemide) 50mg/ml

Dose rate of 2- 4 mg per kg in dogs and 1 – 2 mg per kg in cats intravenously or intramuscular. Used to treat cerebral and pulmonary oedema. It is a diuretic and hence helps to remove fluid. Care must be taken that the patient does not dehydrate and develop an electrolyte imbalance.

Dopamine 40mg/ml

Dose rate of 5 – 10 ug/kg/min for cardiac uses. Used for the treatment of acute heart failure and to correct the hemodynamic imbalances that can be present following shock. It increases cardiac output, organ perfusion and renal blood flow.

Dobutamine 12.5mg/ml

Dose rate of 5 – 15mcg/kg/min. Used for the treatment of low cardiac output and myocardial failure. It can also be used in shock patients when fluid therapy alone has not restored arterial blood pressure, cardiac output or tissue perfusion. It is a short acting drug.

Visual aids such as dose rate charts, protocols and checklists will also improve the outcome for CPR. You do not have time to be working out the correct dose for that animal in CPR so having a dosage chart with the weight of an animal for the drugs Adrenalin, Atropine and Lignocaine in particular is highly recommended.

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WSV18-0253

WSAVA DENTAL GUIDELINES

HOW THE WSAVA DENTAL GUIDELINES CAN IMPROVE YOUR PRACTICE

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Marketing

Improving your practice with the WSAVA dental guidelines

b.a. nIEMEIC, dvm, dipl. AVDC, Dipl. EVDC, Fellow AVD

Many veterinarians have not heard of the World Small Animal Veterinary Association; much less know they are members. The WSAVA is an association of associations, counting over 200,000 members and 100 associations. Therefore, if you are reading this, you are likely a member of the association and have access to all of the educational and promotional material on the website and any CE provided.

Why are the guidelines important?

Dental disease is the most common problem in small animal medicine. There are rarely any outward clinical signs, however these conditions do cause significant pain and infection. Further, these conditions create many local and systemic ramifications. Therefore, dental disease is significantly undertreated. Further dental education is sadly lacking in most veterinary curricula. The combination of all these issues allows numerous myths and misconceptions to remain. This includes the overuse of antibiotics and anesthesia free dentistry.**What are the Dental Guidelines?**

The committee includes not only Diplomates of the American and/or European Veterinary Dental Colleges from 5 continents but also specialists in anesthesia and analgesia as well as nutrition. Finally, there are two animal welfare advocates on the committee.

The guideless contain sections on oral pathology, anesthesia and pain management, as well as the universities role in improving dental education. Further is a section on the importance of and how to perform a proper oral exam, which includes instructions on the use of a free on line charting system. Step by step instructions with full color images detail the basics of dental prophylaxis, dental radiology and extraction. Minimal equipment recommendations are made for the various areas as well as a thorough review on dental homecare. While non-anesthesia dentistry is discouraged throughout the text, it's inappropriateness is the subject of its own section. Finally, the animal welfare impact of untreated dental disease is introduced.

This document is tiered by the socioeconomic position of various countries, to improve the minimum standards of care in a realistic fashion while still promoting best practice. The authors have strived to create a document that is not only available world-wide for free, but also written in a very accessible way.

Why is promoting the dental department important?

1. Oral disease is by far the most common problem in veterinary medicine and there are generally only subtle to no clinical signs. However, patients afflicted with dental disease are quite often painful despite the lack of clinical signs. In addition, these disease processes cause significant localized and systemic medical problems. Ignorance abounds regarding dentistry both in the general public as well as in the veterinary field. This results in most patients being under treated. Therefore, proper dental therapy is financially rewarding and good medicine.

2. Over the last decade or so, there has occurred a significant loss of traditional revenue streams due to many factors. Vaccine revenue has been markedly reduced by new studies. In addition, flea and heartworm prevention as well as other prescription revenue has been lost due to online prescriptions. Finally, increased reliance on the internet or other information decreases the client trips to the clinic

How to Increase Dental Compliance and Revenue

Dental revenue can be improved in four distinct ways. However, they do not stand alone; all of them should be included in the marketing plan. In fact, they are synergistic, by increasing more than one, they positively affect each other, further improving gains.

1. The first and most cost-effective way to attain this goal is to increase the number of dental prophylaxis procedures performed.

    1. Client education: This is best performed by enlightening the population about dental disease. This should come not only from the veterinarian, but the entire staff. This includes technicians and possibly most importantly, receptionists. By educating the veterinary staff, you educate the clients and provide more dentistry. This ideally is in person, but if time is an issue, handouts or qualified websites can be effective as well. There are can be in person, or via handouts and/or websites. Client educational videos are available at www.dogbeach-dentistry.com

    2. Superior, new equipment: Once the marketing plan is underway and the days

are full, superior equipment will speed procedures. A new drill, ultrasonic scaler, elevator, or curette can markedly cut down on surgical time and increase the number of procedures performed a day. If a practice can do one more procedure a day 5 days a week at an average of say $400 it will pay off $4,000 worth of equipment in a month. Moreover, this will result in shorted anesthetics.

3. Continuing education/training: By learning better techniques veterinarians and technicians can speed the dental procedures benefiting the practice and the staff. The staff can be more efficient, which will also allow for the possibility of additional procedures. Furthermore, this efficiency will decrease operator stain and stress. Finally, proper performance of dental procedures should result in less surgical trauma and superior patient care.

2. The next way to increase income is by increasing the per dental procedure charge. Increase the number of treatment options for the clients. This does not mean doing things like root canals, jaw fracture repair and major oral surgery since what most DVM's charge for these it is not efficient time usage. By spending that time doing office calls the practitioner will increase income with less stress. A more efficient way to do this is by offering superior "basic" care. This should include:

    1. Dental radiology,

    2. Periodontal pocket treatment

1. i. Closed root planning/subgingival scaling

2. ii. Perioceutic (doxirobe/clindoral)

    1. Barrier sealants

    2. Regional anesthesia

    3. Proper pain management

    4. Bonded sealants

All of these will greatly increase income without a significant investment of time or money. Practitioners, who have mastered the basics, can consider proceeding to composite restorations and periodontal flap surgeries.

1. Clinics can markedly improve their dental and income by improving their pre-operative testing protocol. Furthermore, perform the pre-operative testing the day the cleaning is recommended, this will help lock clients into the procedure.

A. Complete blood panel (renal, hepatic, CBC, T4)

B. Urinalysis

C. Chest radiographs

HCM is often not ausculted

Over 50% of patients over 6 have significant findings on chest films

1. Provide superior (and necessary!) post-operative treatment

   1. Pain management: Opiates, NSAIDS, Local Anesthetics (nerve blocks)

   2. Homecare products such as tooth brushes, rinses, and dental diets

   3. Rechecks

5. Specific cases where income can be increased

A. Persistent deciduous teeth are a very common problem in small animal patients, especially toy breeds. Most clinics will do this and charge for it, but in general they will way under charge and under treat. These are large teeth that are time consuming extractions. By keeping the teeth, the clients can understand why the extraction is expensive. In addition, proper pain medication and radiology will increase the fee to a reasonable level.

B. Fractured teeth with pulp exposure are a very common occurrence in veterinary medicine (approximately 10% of dogs have a broken tooth with pulp exposure). All teeth that are fractured with pulp exposure are either painful, infected, or both. Therefore, all teeth need to be treated via root canal therapy or extraction. This does "bother the dog" and therapy is critical. If a minor tooth, extraction is a viable option. If it is a major tooth and the client is to be referred for root canal therapy, the patient should be placed on pain medications and/or antibiotics and a minimum database performed.

C. Worn teeth with root canal exposure need to be treated with root canal therapy or extraction. Teeth without root canal involvement should be radiographed to ensure lack of endodontic infection and then treated with composite bonding if indicated.

D. Discolored (intrinsically stained) teeth. A study by Hale in 2001 reported that only 40% of discolored teeth have radiographic signs of endodontic disease. However, when physically examined, it was discovered that 93% of the teeth were in fact non-vital. Therefore all discolored teeth should be treated as dead and infected (root canal therapy or extraction).

E. Feline tooth resorptive lesions are reported to be present in up to 60% of all cats greater than 6 years of age. These are VERY painful lesions and require therapy.

These are diagnosed with an explorer along the gingival margin. Full mouth dental radiographs are indicated when lesions are found as they will generally have additional lesions. These teeth need to be extracted.

F. Periapical Abscess can be treated by root canal therapy or extraction. If electing to perform an extraction, remember that they are surgical procedures and should be charged as such. By calling it oral surgery it changes client perception of the procedure. Dental Radiographs and pain management including local anesthetics should be administered.

G: Oral masses are incredibly common in small animal dentistry (especially dogs). All growths no matter how small and normal appearing should be sampled and submitted to the lab for histopathologic analysis. In my experience about 1% of these biopsies will turn out to be malignant and need additional therapy. In addition they should all be radiographed to evaluate for bony involvement. This will help the pathologist to determine level of aggressiveness.

H. Uncomplicated Crown Fractures are a very common finding in large breed dogs (at least 50%). This occurs when a piece of the crown is broken, which exposed the dentin, but not the root canal. Occasionally, these teeth can become infected through the dentinal tubules which will go undiagnosed without dental radiology. However, teeth with no to small pulpal exposures tend to be the ones with clinical abscessation. Even if these are not infected, they are at least transiently sensitive and require restoration.

- Ø Treatment

- Ø Dental radiographs are WNL

ØBonded sealant

ØRecheck radiographs in 9-12 months

- Ø Radiographic evidence of endodontic disease

ØRoot canal therapy

ØExtraction

Key Points of periodontal disease from the DG

- Periodontal disease is by far the most common medical condition in small animal veterinary patients.

- Plaque forms within 24 hours, calculus within 3 days and gingivitis begins as early as 2 weeks.

- Periodontal inflammation is caused by subgingival plaque; therefore, control of plaque needs to address both supra- and more importantly subgingival plaque to be effective at controlling disease.

- Calculus (or tartar) is essentially non-pathogenic

- The first sign of periodontal disease is bleeding

on probing or brushing which occurs prior to a color change

- Periodontal infections have been linked to numerous systemic maladies including:
    - Diabetes Mellitus
    - Heart, lung, liver, and kidney disease
    - Early mortality
- Periodontal disease has been associated with numerous severe local effects including:
    - Oronasal fistulas
    - Oral cancer
    - Mandibular fractures
    - Ocular infection and blindness
    - Osteomyelitis and osteonecrosis
    - Class II perio-endo lesions

Key Points of periodontal therapy from the DG

- A professional dental cleaning is an involved procedure with numerous steps.
- All periodontal therapeutic procedures must be performed under general anesthesia.
- Each step must be properly performed to achieve a positive outcome.
- Sufficient time must be allotted for the procedure to have significant clinical benefit.
- Subgingival scaling is the most important step of a professional dental cleaning.
- A complete oral exam and charting is a critical part of the procedure.
- Daily homecare is recommended since plaque accumulates in 24 hours.
- Without homecare, the efficacy of professional periodontal therapy is severely limited.
- Tooth brushing is the gold standard and is most effective on rostral teeth.
- Passive homecare methods may or may not be effective, and any provided benefit will be mainly on the caudal teeth.
- Standard dry dog food is not beneficial for oral health.

How will members benefit from the DG?

The sections on oral pathology provide current diagnostic and treatment recommendations for common oral pathology. The text is supported by numerous full color pictures as well as dental radiographs. Since this is available on line for free, it can facilitate client communication. This will improve dental compliance, thus improving patient care and practice income.

The anesthesia & analgesia section contains instructions and recommendations for pre-anesthesia testing, drugs, and monitoring. This is the latest information and is a valuable resource for the practitioner. Further, this section details the most current level of safety, which should further increase compliance

The numerous mentions of the inappropriateness of NAD will greatly aid practices in decreasing this wholly ineffective practice. The arguments against this procedure are presented in not only the dental prophylaxis section, but also in the anesthesia and welfare areas. This combination, together with a listing of all the professional associations who oppose it will aid in client discussions.

Finally, but perhaps most importantly, is the section on the welfare aspect of untreated dental disease in small animal medicine. This well referenced section, penned by non-dentists, highlights the plight that our pets face on a daily basis when dealing with untreated dental conditions. By using the term "animal welfare concern" we can improve the acceptance of recommendations on a personal as well as association level. Together we can strive to improve oral care for pets worldwide.

WSV18-0108

SVA PRACTICE MANAGEMENT

USING YOUR CLINIC'S DATA TO IMPROVE COMPLIANCE TO VETERINARY RECOMMENDATIONS

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USING YOUR CLINIC'S DATA TO IMPROVE COMPLIANCE TO VETERINARY RECOMMENDATIONS

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What is Compliance?

The AAHA Compliance report in 2003 defined compliance as;

The pets in your practice receiving the care you believe is best for them.

Areas where we know compliance rates are poor include Preventative healthcare, Repeat medications, Elective procedures, and Dietary recommendations

Improving Compliance requires the three R's; Recommending; Reinforcement; Reminding

The Steps to full compliance;

1. A clear recommendation
2. Reinforcement from the team
3. Follow up if not acted upon (Reminder)
4. Reinforcement from the team
5. Follow up if not acted upon ………

A few tips on improving compliance

A verbal recommendation is not enough; A written Recommendation always adds more weight

Recording the recommendation enables Reinforcement from the team

Setting a Reminder stimulates Follow up

Understanding your system

Most practice management software includes a system of "Reminders" or "Recalls", understand how to get these to work for you in your clinic. It will depend on your system, however, most allow reminders and these can often be linked to "Sales items" or can be set "manually"

"Selling" a recommendation often creates additional visibility because it then appears on the client's invoice which is visible to front of house staff and can stimulate discussion and reinforcement when the client pays their bill.

Case Study 1 - Recommending and Recording

Background: A UK based clinic where a review of practice data showed that only 35% of vaccinated patients had a record of having received dental advice at the time of vaccination, although all would have had their teeth checked as part of the annual health exam.

The Goal:

To ensure that all patients had their dental health status both determined and recorded at the annual health exam, and that when appropriate follow up reminders were set

A review of current situation with the clinical team identified 3 groupings for patients dental health that could be recorded;

1. Dental health good, no action required
2. Dental problems identified, remedial treatment advised
3. Significant dental problems identified, immediate remedial action recommended

We created 3 new sales items and 2 associated reminder types to allow all 3 groups to be recorded and counted. Using a Sales item ensured that the recommendation would be included on client invoice, and visible to front of house staff

A series of meeting was held to communicate the modified recording process to all staff and a process of monthly monitoring commenced.

Each month progress was reviewed with the vet team and experiences were shared which helped identify the exceptions and omissions, and these lessons were shared with all staff with the result that recording levels quickly improved, and the reminder follow up resulted in a higher proportion of patients receiving the dental care that they required.

Reminders and Recalls - The most important "tool" in the box We most typically use reminders for events such as the Annual Health-check, Routine Vaccinations, Internal and External parasite control

We find that despite the fact that we would expect to see every pet at some future date, on average in UK clinics only 50 to 80 % of patients have an active recall or reminder set for any preventative health treatment.

In many regards we should not be surprised at this figure, because reminders are easily missed, for example when a 6 month old puppy, who was already vaccinated elsewhere visits for the first time for neutering or when a new client visits with a sick animal visits for the first time and the vaccination is not due

43RD WORLD SMALL ANIMAL VETERINARY ASSOCIATION CONGRESS
AND 9TH FASAVA CONGRESS

Because we expect to see every patient again, before they leave, we should always check;

1. When do we expect to see them again?
2. What for?
3. Is the reminder correctly set?
4. Does the client know to expect it?

Case Study 2 - Routine Reminders and Recalls

Background: UK based clinic with 4 branches. In the UK typically only 64% of dogs are vaccinated (UK has no Rabies or high fatality risks) yet a review of practice data showed that only 59% of active canine patients were vaccinated.

Goal:

To ensure that all canine patients had their vaccination status determined, and the appropriate reminder set

A review of current situation with the clinical team identified 5 groupings for patients;

1. Vaccinated by the practice, reminder set
2. Vaccinated elsewhere, reminder set
3. Vaccination not required (medical reason)
4. Vaccination not required (domestic reason)
5. Vaccination declined by owner

3 new sales items and associated reminder types were created to allow all 5 groups to be recorded and counted and the revised process was communicated to all staff

Monthly monitoring using a "review and learn" process identified exceptions and omissions and these lessons were shared together with improvement ideas across all branches

The result was that over time the proportion of patients whose vaccination status was recorded now exceeds 95%, with the result that overall vaccination rates now exceed 75%, meaning that more patients are protected.

Get the "Recall" habit

There are lots of other areas where we can adapt our recall and review habit to improve compliance, such as following up on the first or sample bag of a new diet, following up on lapsed prescriptions for long term medications, overdue weight checks, and geriatric screening.

Checklist for change;

At every consultation;
- Always identify the next meeting
- Talk about it before they go
- Ensure the appropriate recall is set
- Warn the client what to expect
- Measure your practice's performance
- Review progress, Acclaim, Learn, Instruct
- Plan how to celebrate your success!

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TIBIAL PLATEAU ANGLE – HOW TO MEASURE IT & WHY YOU NEED TO ON YOUR CRUCIATE LIGAMENT PATIENTS

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I recommend measuring tibial plateau angles (TPA – Fig 1) on all patients prior to surgery regardless of the stabilizing procedure being performrmed. Unpublished data have shown that the strain on the CrCL increases as the TPA increases (Hulse, DA, 2009). It is logical then that extracapsular sutures ("ExCap") would be under progressively higher strain as the TPA increases. High strain upon the ExCap suture could increase the risk of premature suture stretching or breakage. While one study showed that TPA did not affect the outcome of ExCap stabilization treatment, that study did not look at dogs with TPA > 34°. I currently recommend that ExCap suture stabilization be avoided in dogs when the TPA exceeds 30°.

In order to properly position a patient for radiographic measurement of TPA, I advise heavy sedation or general anesthesia. The patient is the placed in lateral recumbency with the limb of interest in the 'down position'. The radiographic objective is to obtain a PERFECT medio-lateral radiograph of the entire tibia, but with the radiographic beam centered upon the stifle. It is important that the hip, stifle and tarsus each be resting upon the same surface (or at least resting at the same level) in order to avoid rotating the tibial out of the perfect medio-lateral orientation, Next, the "up" leg is pulled forward via hip flexion – the movement of this limb in the sagittal plane will help preserve the perfect mediolateral position of the 'down' tibia. In contrast, abduction of the upper limb tends to pull the 'down' limb out of the perfect medio-lateral position. Finally, remember to center the radiographic beam upon the stifle, but collimate it to include the talus (Fig 1). When properly positioned, the radiograph should show the intercondylar tubercles of the proximal tibial (collectively referred to as the "tibial eminence") arising from a "horizon" created by the superimposed medial and lateral tibial plateau surfaces.

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BACTERIOLOGICAL EVALUATION OF PYOTHORAX IN DOGS AND CATS

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Pyothorax is accumulation of exudates in the pleural cavity, also known as empyema. It is characterized by accumulation of purulent fluid due to bacterial or fungal contamination. Clinical signs of pyothorax are not specific and commonly include dyspnea, tachypnea, anorexia, pyrexia, and exercise intolerance. Duration of the clinical signs varied from 1-300 days, with a median of 33 days in cats and 40 days in dogs. Disease severity is influenced by the chronicity of the infectious. In order to facilitate establishment of treatment guidelines, the **American Thoracic Society** divides pleural infections into three stages: an exudative stage, fibropurulent stage, and organizational stage characterized by formation of a pleural peel (scar tissue). Although numerous retrospective studies, case reports, have been published on the subject, no data exist on the actual incidence of pyothorax in dogs and cats. Furthermore, the actual route of pleural infection often remains unknown with identification of an underlying cause reported in only 2–22% of dogs and 35–67% of cats. Diagnosis is based on clinical signs, thoracic X-ray, US and CT-scan, fluid sampling, cytological evaluation and bacterial/fungal culture and sensitivity. Definitive diagnosis is based on cytology examination of pleural effusion or subsequent aerobic and anaerobic bacteria/fungal culture results.

The isolated bacteria vary and are influenced by the primary source of contamination. The etiology of pyothorax includes; penetrating wound, primary bacterial pneumonia that may progress to parapneumonic effusion; migrating foreign body, esophageal perforation and lung absces. In cats bite wound were considered as the most common cause of pyothorax with pasteurella contamination. A study evaluating the risk factors of 80 cats suffering from pyothorax, cats were 3.8 times more to live in a house load, but only 15% (20/128) had signs of bites. Another study of 27 cats with pyothorax in Australia has revealed that 78% of the cases had mixed oropharingeal isolation including **Pasteurella**, **Salmonella**, and **Mycoplasma** Species in kittens. 56 % of the cats suffered from pneumonia indicating the parapneumonic spread may be more common in cats as the source of the pleural contamination. Esophageal rupture may occur due to foreign body, neoplasia by **Spirocerca lupi**. The incidence of inhaled foreign bodies seems to vary depending on geographical location, climate, and vegetation, and the type of activity in which the dog is involved; hunting, working dogs or just dogs

living in the country side were grass own are common. In a study evaluating 182 dogs with documented grass own inhalation, only in 3 dogs grass own was confirmed. Grass owns migration may be the source of **Nocardia** and **Actinomyces** spp. that are usually in organic matter.

Primary antimicrobial therapy is based on cytological evaluation and later on culture and sensitivity. Cytologic findings did not match microbiological findings

in 13 of 44 (30%) dogs. Empirical treatment with penicillin based antibiotics combined with floroquinalones and sulfadiazine (TMS) have shown that results of antimicrobial susceptibility testing suggested empirical antimicrobial selection was associated with a 35% risk of inefficacy. Several bacteria may produce sulfur granules in the septic pleural effusion. A study of 46 dogs with pyothorax a filamentous, branching organism was detected cytologically in 18 dogs, 11 of which had sulfur granules. Bacteria isolated from these 18 dogs included *Prevotella spp, Clostridium, Bacteroides spp*, *Corynebacterium spp*, *Staphylococcus spp, Enterobacter spp, Fusobacterium spp Nocardia and Actinomyces spp*. The 2 latter are Gram positive, creating endospres that became reactive in optimal environment. They are not always identified in normal stain as they are partially acid fast therefore may need special staining (e.g Zen Nielsen) to identified them in a smear. When these bacteria are highly suspected a combination of clindmacyn and TMS should administrate. CT-scan and surgical intervention may be needed as pulmonary absces is suspected as source of infection. Intrapleural antimicrobial treatment is not recommended in human empyema. Only one limited study in veterinary medicine has shown shorter thoracic tube placement in dogs treated intrapleurally with antibiotics (ampicillin and /or metronidazole) (4.8 vs. 6.8 days). However, the sample size was small and did not reach significant statistical difference.

SVA FELINE

FELINE DIAGNOSTIC LAPAROSCOPY: GOING BEYOND THE 'LAP SPAY'

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Introduction

Laparoscopy offers a minimally invasive alternative to laparotomy for exploring the abdominal cavity in our feline patients. The widespread acceptance of laparoscopy stems mainly from the successes and technological advances in a variety of procedures in the human field compared to those of traditional open surgery. Though many surgical procedures performed in human beings will have little applicability in cats, many of the procedures used for diagnostic purposes will offer value due to its relative ease, effectiveness, and decreased of morbidity compared to more traditional interventions.

Diagnostic Laparoscopy: Is it for the Feline Practitioner? Does Laparoscopy Present Any Advantage to the Patient? What are the Advantages for the Owners

Often times in feline practice, the clinician is faced with owner hesitation in putting a cat through traditional exploratory surgery for the sake of diagnostic purposes. With that in mind, clinicians will often have to rely on indirect and incomplete information provided by blood tests and imaging studies to arrive at a 'most likely' diagnosis. The psychological barrier for pursuing surgical biopsies seems greatly reduced when a minimally invasive alternative is offered as an alternative to open surgery.

It's been well established in the human field that laparoscopic procedures greatly reduce patient morbidity, post-operative pain and post-operative recovery times. In both human and veterinary literature, it's been well documented that larger incisions are considerably more painful compared to smaller incisions.[1-3] Recovery times (and **how well** the patient recovers) are also important factors when deciding on minimally invasive surgery, not only for the patient, but also their caregivers during the convalescence period. In addition to reduced incision size, cytokine and other inflammatory mediator release are greatly reduced, not only due to minimal trauma during tissue manipulation, but also directly due to carbon dioxide insufflation. The latter has been shown, in vitro, to cause a marked cytosolic acidification in peritoneal macrophages which suppresses cytokine release for up to 24 hours.[4]

Can the Practitioner Benefit From It?

Over the years, placing an endoscope into a cavity (or 'hole') has brought a tremendous amount of diagnostic information to the clinician. Laparoscopy gives the internist an extra opportunity to carry out a thorough visual inspection of the abdominal cavity, in a highly magnified and illuminated environment.[5,6] This is particularly advantageous in very small patients particularly when attempting to gain access into relatively inaccessible spaces (e.g. between liver and diaphragm). Laparoscopy allows for procurement of excellent quality (and size) tissue samples[5-7] in a controlled environment (e.g. controlling haemostasis).

The idea of minimally invasive surgery, as well as the idea of offering the state-of-the-art technology, will certainly push cat

owners to request for minimally invasive procedures.

Disadvantages:

I've described the desirable advantages to **kitty keyhole surgery**...so what are the disadvantages? One drawback is the initial financial outlay and training required to acquire the skills and safely perform these procedures in feline patients. Another drawback is the overly compliant abdominal cavity in the cat, coupled with a small working space for triangulation and reduced depth perception which can make abdominal access more challenging compared to dogs.[5,6] In addition, there's a need to development of a '6th sense' due to lack of direct tactile feedback by relying on instruments as an extension of our fingers. For example, the use of a palpation probe can be used not only to manipulate organs but it's also used palpate textures and 'ballot' organs (e.g. gall bladder for signs of inflammation/thickness) as additional information beyond gross appearance. The key to success to making the transition over to 'the other side' is to be **patient** and taking a step-by-step approach when acquiring new skills, receiving formal tuition from experienced endoscopists (e.g. university training centres), and refrain from being too ambitious when being released into the real world!

Indications for Diagnostic Laparoscopy

Full evaluation of abdominal organs for signs of disease, biopsy liver, pancreas, lymph nodes, kidney, small intestine ('laparoscopic-assisted' full-thickness), abdominal mass evaluation, and cholecystocentesis.[5,6]

Contraindications

Cardiovascular instability, inappropriate equipment, inexperienced/untrained surgeons, diaphragmatic hernia, extreme obesity, septic peritonitis, adhesions, coagulopathy, any condition in which conventional surgical intervention is indicated.

History/Clinical Examination/Pre-surgery Diagnostics:

As for any surgical candidate, a full clinical history, clinical examination, and appropriate pre-surgery diagnostics should be performed prior to any exploratory intervention. Ultrasonography by a skilled imager is extremely useful to help isolate a specific area, and extent, of disease. Diagnostic imaging is considered to be a **complementary** part of any medical investigation and should ideally be considered prior to any laparoscopic assessment. A full clotting profile should also be performed prior to surgery (PT, APTT, and total platelet count); however, if this is not possible, then performing an activated clotting time, total platelet count and buccal mucosal bleeding time should be performed as a bare minimum. Despite the latter recommendation, clinical experience (and experience of fellow colleagues) suggest that abnormal bleeding times may not necessarily preclude performing diagnostic laparoscopy as abnormal clotting times do not always correlate with excessive bleeding at biopsy sites. There are studies in human medicine that indicate that in vitro coagulation tests do not accurately predict the probability of hepatic bleeding times.[8,9]

Liver Biopsy

Once the abdomen has been thoroughly examined, a suitable area on the liver is selected for collecting a biopsy using a 5mm laparoscopic 'clamshell' biopsy instrument.[5-7] This can be placed through the same accessory cannula as the blunt probe, thus avoiding the need to place an additional port. These types of instruments obtain much larger specimens compared to fine needle or core biopsy samples. If excessive bleeding occurs, the palpation probe is used to apply direct compression over the biopsy site until a clot has formed. If bleeding is still uncontrolled then laparoscopic forceps can be used to place a piece coagulation material into the biopsy defect.

Pancreatic Biopsy

The pancreas can be biopsied from the distal-most portion of the right limb for diffuse disease, or directly into a lesion, using a 5mm endoscopic biopsy punch forceps. It's vital to avoid the pancreatic ducts in the proximal portion of the limb, next to the duodenum. The same technique for collecting a liver biopsy, applies to pancreatic biopsies.

Cholecystocentesis

Bile aspirates for cytology and culture/sensitivity are important when performing gastrointestinal investigations in cats, particularly those suspected of having triaditis. Laparoscopic cholecystocentesis is considered safe and easy to perform. The author will typically use a spinal needle (18-20G x 3-6" or 20-22G depending on fluid viscosity) with an inner stylet that can be removed once the lumen of the gall bladder has been penetrated. The needle entry site on the external abdominal wall should be identified by ballottement visualised through the endoscope and should be caudal to the diaphragm. Depending on the intracystic pressure, it can be challenging to penetrate the wall with the needle. A quick forceful 'jab' will usually facilitate entry. Complete aspiration of all contents should be attempted to reduce the risk of bile leakage and peritonitis. In the author's experience, this is a rare occurrence.

Intestinal Biopsies

Biopsy of the small intestine is usually performed using a laparoscopic-assisted technique. A 'mini-laparotomy' is usually performed by exteriorising a small bowel segment through a port incision. As an alternative approach in cats (compared to dogs), the author will often perform intestinal/lymph node biopsies at the end of the exploratory procedure using a wound retractor (Alexis 2cm-4cm)[10] placed in the caudal camera port. Because pneumonperitoneum will be lost, it is advisable to perform intestinal biopsies at the end of the procedure.

Post-operative care

Most cats (if no intestinal biopsies were obtained), will often be discharged the same day as the procedure. If intestinal biopsies were collected, then overnight observation is advised as per open surgery until the patient is eating with no signs of post-operative complications such as intestinal wound dehiscence.

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NUTRITION AND DIAGNOSTIC IMAGING (LECTURES GIVEN IN MANDARIN CHINESE)

MEASUREMENT IN ULTRASONOGRAPHY: DOES IT MATTER?

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THE THORAX EXAMINED BY THE RADIOLOGIST: HOW TO AVOID MISTAKES

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Two most commonly mistakes that can be avoid easily in reading thoracic radiographs occurs during the radiographic process, which are taking too few views and inappropriate positioning. **Too few views**

A minimum of 2 orthogonal views are needed for a thoracic study. A minimum of 3 views are needed for a metastatic check for metastatic neoplasia to the lungs. As aspiration pneumonia commonly occurs in the right cranial and right middle lung lobes, a left lateral view is needed to diagnose this condition. A recent trend of performing 3 views (2 laterals and a VD) is recommended for all disease condition in thorax. A DV view is needed for the evaluation of the caudal lobar vessels in the investigation of cardiac disease. At our institute, we performed a DV, a VD and a lateral views for study of cardiology.

Inappropriate positioning

Shape and size of the cardiac silhouette is very sensitive to positioning. Any obliquity of the DV or VD views will distort the cardiac silhouette and give us a wrong impression of cardiomegaly. Misdiagnosis of right cardiomegaly is commonly make with shifting of the cardiac silhouette to the right secondary to oblique positioning. The spine and the ribs are not optimize for reading. Wrong diagnosis of spinal fracture in cases of motor vehicle accident is common.

A designated person for quality control for all images taken is essential in any hospital. At our institute, the radiology residents and radiologists approve all studies. Occasionally, patients need sedation or general anesthesia to achieve optimal positioning. Patient not suitable for sedation or general anesthesia may not have a good positioning radiograph and making reading difficult.

The other commonly make mistakes after radiography are mistaken normal structures as abnormal, fail to recognize breed variation, fail to recognize normal anatomical variation and fail to recognize physiology effect on radiographs.

Mistaken normal structures as abnormal

The caudal esophagus is commonly seen as a soft tissue tubular structure with a distinct ventral border, on the left lateral recumbency view. This may be mistaken as esophageal foreign body. Sternal lymph node is normally seen on a right lateral recumbency view but not the left lateral recumbency view. Thus is it crucial to be able to detect a sternal lymph node in both lateral recumbency views to make a definitely of mild enlargement of the sternal lymph node in cases of patients with peritoneal disease. Radiographic images are 2 D image of a 3 D structure. Superficial structures such as skin tag, nipples and wet hair may be seen as the lung parenchyma. One of the challenges of reading thoracic radiograph is to differentiate the superficial structures such as nipple from a real pulmonary soft tissue nodule.

Fail to recognize breed variation

Cardiac Silhouette: The appearance of the cardiac silhouette is breed and body conformation dependent. The most common error in reading thoracic images is overestimation of the size of the cardiac silhouette. This is especially true for the barrel-chested dogs when there is a heart murmur detected during physical examination. The cardiac silhouettes of the deep-chested dogs are taller and the barrel-chested dogs are slightly shorter and rounder. Oblique positioning of the VD sometimes will make the cardiac silhouette appear larger on the right and smaller on the left. There is a positive spinal trachea angle, mainly in deep chested dogs. The reduction of spinal trachea angle is normal due to left ventricle enlargement and presence of a heart base mass. In small breed and barrel chested dogs, the trachea normally runs parallel with the spine, thus it may has a zero spinal trachea angle. One of the common mistake is to look at this small spinal trachea angel in small breed dog and make a misdiagnosis of left sided cardiomegaly secondary to mitral valve insufficiency. Pleural indentation is commonly seen in Basset Hound on both VD and DV views. This mimics retraction of lung lobes, thus mistaken as presence of pleural effusion.

Fail to recognize normal anatomical variation

Presence of pericardiac fat will occasionally lead to enlarged cardiac silhouette. This is especially true in the VD view of cats. Sometimes, the cardiac silhouette could not be see with the abundant of pericardial fat. In older cats, the cardiac silhouette will have a more horizontal position and a bulge may be present at the aortic arch. This should not be mistaken as an aortic aneurysm. Widening of the cranial mediastinum is one of the radiographic signs of cranial mediastinal lymphadenomegaly. In some cases of obese or brachycephalic dogs, the mediastinum could be very wide due to presence of abundant mediastinal fat. This is sometimes misinterpreted as a mediastinal mass. The cranial sub segment of the left cranial lung lobe is located slightly more cranial to the right cranial lung lobe, crossing right ward over the mediastinum and part of it appears to be in the "cranial aspect of the right hemithorax". On the lateral view, this could be mistaken as a large pulmonary bulla.

Fail to recognize physiology effect on radiographs

It is common to detect a small amount of gas in the mid thoracic esophagus in healthy animals. When patients are under sedation or general anesthesia, there may be a slightly increased in the gas in the esophagus. Thus the status of sedation and general anesthesia of the patients should be considered when reading thoracic radiographs. This is easily confused with megaesophagus. Obese animal normally hypoventilate, thus will produce expiratory thoracic radiograph. This will lead to increased soft tissue opacity of the caudodorsal lung field, mimicking pulmonary edema. In some cases, the less expended lungs appear to be "retracted" from the thoracic wall. This could be mistaken as presence of pleural effusion. In deep chested dogs, they tend to be able to inhale easily even though on lateral recumbency. Thus inspiratory radiograph with mild degree of over expansion of lungs may occur. This could be seen as a radiolucent area at the apex of the cardiac silhouette. The differential diagnosis for this is pneumothorax.

As a conclusion, number of views and positioning should be taken into consideration even before interpretation of a thoracic study. The knowledge of normal anatomy variation appearance on radiograph, especially with breed variation is essential. Last but not least, the physiological state of the patient will influence the appearance of the lungs and esophagus, and should be taken into consideration when interpreting thoracic study.

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SVA PRACTICE MANAGEMENT

CHOOSING THE RIGHT ORGANIZATIONAL STRUCTURE FOR YOUR CLINIC

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CHOOSING THE RIGHT ORGANISATIONAL STRUCTURE FOR YOUR CLINIC

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I am often asked "What is the best organisational structure for a practice"

My answer is always, "well that depends!" What this talk is about is just what it depends upon.

Built on my experiences of helping practices with organisational change this talk hopes to;

- Review the most common organisational forms we find in practices today

- Review the strengths and weaknesses of each form

- Highlight the common issues that I find

- Identify the corrective actions required

In managing our practice (just like any other business) there are three principal roles;

Strategy formulation, usually performed by "Directors" whose role is to set out the goals, strategies and policies of the practice

Management control, usually performed by managers whose role is to develop and manage the business processes that will deliver the strategies, policies and goals of the practice

Task control, usually performed by suitably skilled technicians, whose role is to ensure individual tasks are performed efficiently and effectivly

All of us will spend some of our time in all of these roles
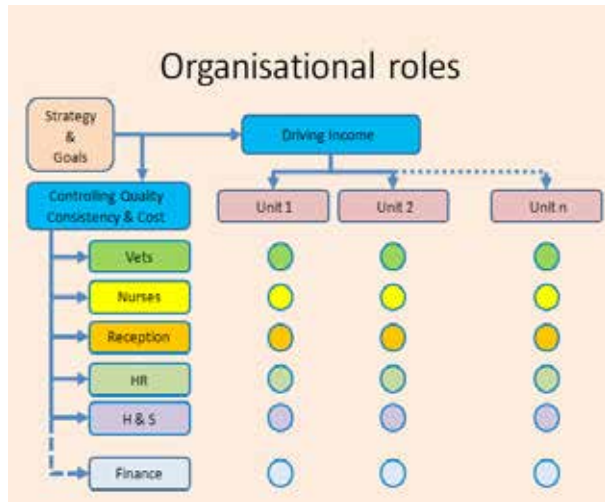
In applying these roles to our practice we can differentiate between;

Those tasks that are concerned with the need to drive income and growth

AND

Those that are concerned with ensuring we consistently achieve the desired quality of output at the lowest total cost to the organisation

Income driving roles should reflect the income streams of the practice,

For example – Large animal, small animal, equine

Or   - Main site, branch 1, branch 2

Or   - 1st opinion work, referral work, out of hours

Whereas consistency and cost roles will apply to the deployment of resources or skills

For example – Vets, Nurses, Reception, HR, Finance, Health and Safety, etc.

For most practices non of these roles are likely to be full time, and so individuals can hold many roles

Owners must decide which roles they wish to keep, and those that they wish to pay someone else to do for them

However, owners need to remember that all of the roles will always be required and cannot be ignored, so if they choose to keep one, they must make the time to perform it!

The result of their choice will determine the appropriate structure for their practice

Owner centric

The most common form of structure for small practices is the owners centric or "hub and spoke" organisation

Positives

The owner retains control of all functions

The owner can react quickly to changing circumstances

The owner can personally influence all elements of the organisation

Very effective during new practice start up

Negatives

The owner gets swamped in detail as the practice grows and cannot perform all of the roles required in an appropriate timescale

Staff can choose to pass all problems straight back to the owner

Rarely works effectively with multiple owners because the decision making process is either unclear or takes too long

Is quickly outgrown, but owners have a reluctance to change because of the loss of control

Once outgrown will cause good staff to leave, and it then becomes very difficult to transform

Delegated non-clinical

The first step from owner centric

Delegation of non-clinical issues to an "Administration Manager"

Positives

Removes admin burden from owners

Enables the practice to bring in or develop "managerial skills"

Owner(s) retain control of key clinical roles

Negatives

Often misunderstood by owners who fail to delegate with sufficient authority to enable the form to work

Hence;

This is the perceived state by the owner's of many owner centric practices!

Delegated non-vet

The next logical step

Delegation of all staff roles and functions other than veterinary surgeons to a "Practice Manager"

Commonest form amongst mid size practices

Positives

Allows further development of the managerial role

Reduces admin burden on owners

Owners (usually vets) retain the management of the

assistant vets

Negatives

Owners fail to delegate with sufficient authority and responsibility

Clinical issues overlap between roles which can cause problems

Practice Manager often finds they have many bosses with many views!

Owners can undermine their manager by dealing with non-vets directly

Delegated business operations

Increasing in popularity with larger practices

Business Manager could be a "Managing Partner" or an "external" manager

Has led to a "partnership share" for some "non-vet" managers

Positives

Removes the "many heads" effect where there are multiple owners

Allows owners who are "clinical specialists" to focus on their key interest and skills and away from "day to day" activities

Can be used in conjunction with many sub structural forms, for example with a Head Nurse or Head receptionist

Negatives

Requires owners to understand their roles as shareholders, directors and technicians, and play them accordingly

Requires effective delegation of authority and responsibility

Departmental structure

Used by practice groups with many business units

Includes as many profit centre managers as there are profit centres

Some roles may not be "full time" depending on size

Some roles may be performed by "owners"

Positives

Organisational structure reflects the organisational needs

Creates a strong and cohesive management team

Negatives

Requires well developed "managers" to be successful

Choosing the right structure?

Review where you are today;

Evaluate current performance, Interview key staff, Assess how effective they feel, What issues frustrate them today?

Honest assessment of current strengths and then agree where you need to be in the future;

Creating the right organisation

1. 1.   Select the appropriate structure to reflect business situation and needs

2. 2.   Owner(s) decide which roles they wish to retain and have time to perform to an appropriate standard

3. 3.   Delegate to their staff the other roles

4. 4.   Delegate with sufficient authority and responsibility

5. 5.   Hire or train appropriate staff

6. 6.   Continually review their roles with a view to delegating further

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SVA SOFT TISSUE SURGERY

DIAPHRAGMATIC HERNIA

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Most (85%) diaphragmatic hernias are a result of trauma. However, not all patients have a history of trauma, nor do they always present with respiratory symptoms. The diagnosis is usually straight forward for those with a history of trauma and respiratory compromise, however it is not uncommon that a patient will present signs such as chronic weight loss, gastrointestinal signs or symptoms related to a hepatopathy or other organ dysfunction. Trauma may have taken place months or years prior, unbeknownst to the owner. The hernia can also be an incidental finding during imaging performed for seemingly unrelated reasons.

In cases of acute traumatic herniation, the time to address the hernia should be carefully considered. Rapid control of the airway and ventilation must be weighed against the value of further patient stabilization. Stabilization over several hours to days prior to intervention may be prudent to minimize anaesthetic and surgical risk. The trauma that has resulted in the hernia has very often caused other injuries. Attempts should be made to resolve hemodynamic instability prior to anaesthesia and surgical intervention. An otherwise stable patient is much more likely to survive the repair. In cases of chronic herniation without decompensation the surgery can be scheduled at the surgeon's convenience.

Ventilation must be provided following anaesthetic induction in all cases of diaphragmatic hernia. The pleuroperitoneal pressure gradient has been abolished and respiratory attempts under anaesthesia will be particularly ineffective. Most anaesthetics impair respiration to some extent and as such decompensation following induction is possible without adequate support. ETCO2 should be monitored throughout these procedures to ensure ventilation is adequate. Inspiratory pressures should be initially limited to 10 or 15cm of water but increased up to (but not beyond) 20cm of water as needed. A typical starting ventilation rate of 6-8 breaths per minute is recommended and adjusted accordingly based on ETCO2.

Once the abdomen has been entered, both sides of the diaphragm should be evaluated. It is not uncommon to have multiple hernias or tears. Circumferential and radial tears of the musculature generally predominate. The falciform fat and liver are most frequently herniated however the small intestine, stomach, spleen, omentum, pancreas, colon, cecum and uterus can also be herniated. Gentle traction on the herniated organs usually facilitates reduction however chronic hernias can

have significant thoracic adhesions. Acute herniations surgically managed within 10 days will not have had time to develop adhesions and reduction is often straight forward. However even acute hernias can be difficult due to congested and friable splenic or hepatic tissue. Torsed vascular pedicles should be noted and de-rotated under controlled circumstances as appropriate or excised if this appears safer. In cases of chronic herniation with extensive adhesions visualization of structures from the thoracic cavity can improve safety. The surgeon should be prepared to perform a caudal sternotomy as required.

During reduction of the hernia the free volume within the thoracic cavity will increase and available volume in the abdominal cavity will decrease. In acute herniations this is generally of less consequence. Care should be taken to evacuate the chest very slowly such that the lungs are not placed under excessive strain. Aggressive ventilation beyond 20cm of water to re-expand the lungs sound be avoided. Negative pressure in the pleural space will facilitate re-expansion of the lungs more slowly and safely. Re-expansion injury can occur in chronic and acute cases secondary to mechanical and reperfusion injury. All efforts should be made to re-expand the lungs slowly. The increased abdominal contents may cause a 'loss of domain' necessitating emptying of the bladder, removal of falciform fat and omentum, and even removal of the spleen should this appear to be contributing significantly. Tension relief of the abdominal wall or an expanded closure with a graft is also a potential option. In cases with abdominal tension an indwelling urinary catheter should be placed to allow post-operative measurement of intrabdominal pressure.

The closure of the diaphragm itself can be performed with synthetic monofilament typically between 3-0 and 0 USP. Suture patterns vary but include simple interrupted, mattress, cruciate, simple continuous patterns (the latter leaving less knot ends that could lacerate hepatic parenchyma). Suturing is generally started dorsally and completed ventrally. Prior to placing the last 1-2 sutures a chest tube can be advanced through the defect and into the thorax. This can then be used to gently remove air from the thoracic cavity with caution not to inflict excessive re-expansion trauma. This tube can then often be removed. In cases where ongoing fluid or gas accumulation are anticipated an indwelling chest tube can be placed.

Post-operative monitoring should include careful evaluation of respiration and blood gas analysis where possible. Adequate analgesia and supportive care provided, the extent of which to be determined on a case by case basis. Abdominal pressures should be below 10mmHg, with medial or surgical interventions required beyond this pressure.

Potential complications include pneumothorax, haemothorax, pulmonary re-expansion injury, or other sequelae arising from the original trauma. Post-operative reflux and oesophageal ulceration is seen in some cases. Extended medical management for reflux can become necessary. Although the recurrence rate is generally low, the client should be instructed to monitor closely, and re-evaluation/imaging performed as needed.

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REPRODUCTION

PREPUBERAL AND POSTPUBERAL GONADECTOMY: TECHNIQUES, ADVANTAGES AND HEALTH CONSEQUENCES IN DOGS AND CATS

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Introduction

Gonadectomy is the surgical removal of both gonads under general anesthesia. In the female, gonadectomy (or spaying) is generally performed through removal of both ovaries (ovariectomy = OVX) or of ovaries and uterus (ovariohysterectomy = OVH) via an abdominal approach. Whether or not bitches and queens should be spayed, what is the best surgical procedure and what is the best age to perform it are issues which have many welfare implications, as well as advantages and disadvantages. Prepubertal gonadectomy is commonly performed on immature male and female animals aging from 6-to-14 weeks and has been routinely used at many shelter and academic institutions in United States and Europe over the last quarter of a century to control pet over-population. Although regarded as a safe technique, prepuberal gonadectomy may carry some health concerns. Postpuberal gonadectomy still offer several advantages if performed early in life although the growth of reproductive organs and the potential presence of pathologic conditions may occasionally complicate the surgical approach. Also, the well known advantage due to a reduced risk of mammary neoplasia is often considerably reduced when gonadectomy is delayed beyond the third cycle in bitches and queens.

Surgical technique – The midline incision should be done in the cranial third of the abdomen for bitches (ovaries are more difficult to exteriorize than the uterine body) and in the middle third of the abdomen for queens (uterine body more difficult to exteriorize than the ovaries). The length of the incision may be increased depending on uterine conditions (an enlarged uterus normally requires a longer incision). Once the ovary has been located the proper ligament is clamped and the ovary is pulled up. When pulling up the ovary the ovarian suspensory ligament is stretched and may be simply broken with the index finger. A hole is made in the mesovarium avoiding the ovarian vessels (normally caudal to the vessels), then 2 or 3 clamps are placed on the ovarian pedicle. An incision is made between the 2 clamps (or – if 3 clamps are used - between the clamp closest to the ovary and the adjacent one), the ovary is removed and the pedicle is ligated directly below the remaining clamp (in case of a young bitch or queen) or the third clamp (the one most distant from the ovary) is removed and the ligature is placed in the groove caused by the clamp. Removal of the ovary can also be done without using clamps but simply having the surgeon holding firmly the ovary and an operator ligate proximal to the ovary.

To remove the uterus the broad ligament is exteriorized, inspected and severed with the thumb or index finger, or is ligated if highly vascularized. Once all potential sources of broad ligament bleeding have been eliminated (2 or better) 3 clamps are placed on the most caudal aspect of the uterine body which is then severed between the proximal and middle clamp. The uterine stump is ligated with a single ligature (for queens or small bitches) or a ligature is placed on each uterine artery. The caudal clamp is released and the remaining groove is used to facilitate placing the ligature on the uterine body. Resection of the uterine body can also be done without using clamps but simply having the surgeon hold firmly the uterine body and an operator ligate distal to the uterine body. The uterine stump is grasped with a hemostat and the clamp is released while checking for absence of bleeding, after which the uterine stump is replaced into the abdomen. Suture material is normally absorbable.

Ovariectomy vs Ovariohysterectomy - Whether to remove only the ovaries or also the uterus is for many veterinarians a true dilemma. The "Anglo-Saxon" or "British" school of thought has always had the approach of removing everything based on the concept that what is removed cannot cause a disease, while the "Latin" approach has always been that of removing only the ovaries since the uterus quickly undergoes atrophy following OVX, and perhaps the risk of developing urinary incontinence could be lower because of anatomical reasons. Research done in Utrecht in which 138 OVH'ed bitches and 126 OVX'ed bitches were followed up for 8-11 years, has clearly showed that between the 2 groups of dogs: a) there is no difference in short-term as well as long-term surgical complications; b) there is no difference in incidence of cystic endometrial hyperplasia, pyometra or any other uterine disease; c) there is no difference in incidence of urinary incontinence[1]

As a matter of fact, the only differences between the two approaches are the degree of invasiveness and length of the surgery, and therefore length of anesthesia, all of which are higher in the case of OVH. This causes a higher risk of surgical complications and a a higher stress for the animal as well as a higher cost for the owner. The following conclusion of Okkens et al.[1] "There is no indications to remove also the uterus in elective castration procedures of healthy bitches, and therefore ovariectomy is to be considered the procedure of choice" is currently shared by the majority of US as well as European authors[2,3,4]. Therefore, it is currently considered unethical to perform ovariohysterectomy

instead of ovariectomy, unless there are specific health reasons. Although such data are available only for bitches, it is likely that the same situation is true also for queens. In case of specific indications for OVH, it is advisable not to remove the cervix because of its important role in isolating the abdominal cavity from outside even in the castrated female. Hysterectomy should not be performed as bilateral development of ovarian cysts has been anecdotally reported with bitches having to undergo laparotomy again to remove the cystic ovaries. Also, if a previously hysterectomized small litter size bitch is mated during heat by a large size dog her vagino-cervical suture may rupture with a consequent coital peritonitis5.

In which stage of the reproductive cycle?

While no specific choice of time during the adult life is necessary when castrating male dogs or cats, performing gonadectomy in adult females may have has some health implications with regard to the reproductive cycle stage in which the female is at the time of surgery. This is particularly true for bitches, as when such surgery is performed in proestrus or estrus there is an increased risk of short-term post-surgical complications (see below), while when it is performed in diestrus there is an increased risk of false pregnancy (due to the sudden decrease in serum progesterone causing a peak in serum prolactin concentration which sets in mammary function and maternal instict). In bitches gonadectomy should best be performed during anestrus (2.5-5.5 months following onset of proestrus); serum progesterone assay may be of help in ruling out diestrus when history is not available. Although no specific study has ever looked at what is the best stage for performing gonadectomy in felines, the queen is not known to experience any such problems depending on when in her reproductive stage she is castrated due to lack of clinically evident effects of estrogens on vascularization of the reproductive tract and absence of false pregnancy in felines.

Advantages of gonadectomy

There are several health advantages due to gonadectomy in male and female dogs and cats6. The removal of the ovaries is associated in both bitches and queens with a reduced risk of mammary and uterine diseases (mammary neoplasia and pyometra, respectivey), as well as absence of ovarian diseases (ovarian tumors, ovarian cysts), progesterone-related diseases (false pregnancy, feline mammary hypertrophy), pregnancy-related diseases (unwanted pregnancy, pregnancy complications, abortion) or parturition-related diseases (dystocia, uterine prolapse, and in the bitch only subinvolution of placental sites) and in bitches also estrogen-related diseases (vaginal hyperplasia/prolapse, persistent estrus, bone marrow aplasia). Bitches gonadectomized prior to puberty have a 95%

reduction of the risk of developing mammary tumors as opposed to bitches spayed after 1st heat (92% risk reduction), after 2nd heat (74% risk reduction) or bitches spayed after 2.5 years of age or left intact (no difference in risk)7. Incidence of pyometra in adult intact bitches vary from 15% to 25%, but the risk tends to increase with increasing age8. Libido, aggressiveness, urine marking and roaming are never displayed in male dogs and cats neutered prior to puberty, while they may persist at some degrees in a few cases when a male animal is neutered as an adult9. From a behavioural point of view it is commonly believed that, apart from not showing reproductive behavior, neutered animals have a more relaxed, somewhat lazy attitude. In male dogs there is a significant reduction of all prostatic conditions6 and in male cats the decreased (or lack of) roaming attitude certainly decreases the risk of death due to trauma. Gonadectomy may play an important role in reducing pet ovepopulation.

Changes due to pre- and post-puberal gonadectomy

Physical changes

In dogs, growth plate closure is delayed when surgery is performed prior to puberty, but the delay is significantly longer when neutering is done at 7 weeks as compared to 7 months. The rate of growth is unaffected but both radius and ulna become longer regardless of the age at neutering. Food intake is not affected nor is weight gain or back-fat depth during the first 15 months or until the age of 18 months when comparing 7 week- to 7 month- neutered puppies 10,11, but weight gain later in life has been reported in non-working bitches. Although it is well known that neutering predisposes (non-working) dogs to develop a significant increase in body weight, the time at which gonadectomy is performed in these animals probably does not affect the clinical manifestation of this problem. External genitalia do not develop fully: penile and preputial immaturity and decreased radiodensity of the os penis are frequently observed (which rarely if ever constitute a problem). Vulvar development may be insufficient in bitches gonadectomised at 7 weeks. However, the incidence of infolding of vulvar lips leading to perivulvar dermatitis and chronic vaginitis is not different when compared to bitches neutered at 7 months12. When penile protrusion was attempted at the age of 22 months, it could be done in all intact cats, in only 60 % of cats neutered at 7 months and in none of the cats neutered at 7 weeks13.

In cats neutered at 7 weeks or 7 months of age physeal closure is delayed 5 to 7 months resulting in a 10% longer size of long bones when comparing neutered to intact cats. Physeal (Salter-Harris) fractures have been reported in neutered dogs and cats at 12-16 months of age14. Salter-Harris fractures are a potential complication when prepubertal gonadectomy is performed, but appear to be as an extremely rare condition in both

dogs and cats. No decrease in urethral diameter (and therefore no increased risk of lower urinary tract disease) has been observed in neutered male cats (regardless of age at neutering) compared to the intact population, with no difference in urethral closure pressure in neutered vs intact female cats, while there was a significant decrease in the diameter of the pre-pelvic urethras in queens neutered at 7 weeks[13]. Glucose metabolism (glucose half life, peak serum insulin concentrations, peak insulin response following intravenous glucose administration) was not altered in neutered (regardless of age at neutering) compared to intact cats when evaluated at 6, 12, 18 months of age, while at 24 months of age neutered male cats had a significantly later peak serum insulin response when compared to intact cats [13].

Behavioral changes

Effects of prepuberal gonadectomy on behaviour vary depending on species and sex and have not been completely investigated yet. In general, excitability and degree of activity are increased in males and females gonadectomised at 7 weeks or 7 months[15,16,17]. However, inappropriate behaviors as reasons for relinquishment have not been reported in dogs neutered at an early age wen compared to those neutered as adults, and in Labrador/Golden Retriever crosses raised as guide dogs for the blind, neutering at 7 weeks or 7 months does not affect the degree of training success. No effect of early age neutering on playfulness, excitement or frequency of vocalization has been reported in cats. In cats castrated prior to 5 months of age there is a decrease in activity and an increase of shyness towards strangers [15,16]. In adult bitches castration may exhacerbate dominance regardless of their attitude prior to surgery. A significant increase in the degree of reactivity has been observed in German sheperd bitches 5 months following OVX[18]. Owners should realize the importance of evaluating the behaviour of their bitches prior to deciding whether to castrate them as castration may not be the appropriate treatment for behavioural problems[17].

Obesity - Obesity is reported as a common side effect of neutering[7]. No difference in food intake, weight gain or back-fat depth was reported for the first 15 months following neutering when a total of 32 neutered and intact dogs were compared. However, food intake increases significantly in bitches during the first 90 days following OVX/OVH with respect to bitches receiving a sham-laparotomy. Larger studies indicate that, irrespective of age at surgery, spayed female dogs are about twice as likely to become obese as intact animals. The risk for obesity does not change in animals gonadectomized prepuberally. It is unknown whether there is a predisposition to obesity, but if this is the case gonadectomy seems to have the same stimulus regardless of age. Obesity is also a side effect of

neutering in cats, as intact animals have a lower weight and a lower fat thickness than cats neutered at 7 weeks or 7 months. The resting metabolic rate is lower and body condition scores are higher in gonadectomised (irrespective of age at neutering) compared to intact cats. Appetite increases significantly in gonadectomized cats 3 days after surgery. In castrated tomcats, a significant increase in body weight 35 weeks post-castration was preceded by significant increases in serum concentrations of Insulin Growth Factor I, prolactin and leptin in castrate toms 1, 7 and 11 weeks following surgery, respectively[19].

Urinary Incontinence - A decreased capacity of the external urethral sphincter is observed following gonadectomy in bitches but not in queens. Average urethral closure pressure in intact bitches is 18.6+10.5 cm H20, 12 months following gonadectomy is 10.3+6.7 cm H20 in continent bitches, and may fall to 4.6+2.3 cm H20 in incontinent animals. Urinary incontinence (UI) is a common sequel of spaying in bitches, with the interval between spaying and diagnosis of incontinence varying from days to months or even years. Incidence of UI in the normal canine population varies from 0.3 to 2.0%, while it increases to 5-10% in the spayed bitches population. In a long-term study on risks and benefits of early age neutering in dogs, the incidence of UI was reported to increase in bitches neutered prior to 3 months of age when compared to that of bitches neutered after 3 months, therefore the age of 3 months was indicated as a threshold afer which female puppies could be safely neutered[15]. However, many authors think that going through puberty may have a beneficial effect as it allows the reproductive system to reach its final stage of growth thereby perhaps limiting the incidence of side effects such as UI as well as those due to insufficient growth of external genitalia.

Osteoporosis – Osteoporosis (loss of trabecular bone) is the most important complication of menopause in women and is thought to be due to a lack of estrogen stimulation causing reduced secretion of calcitonin. Loss of trabecular bone has been observed in Beagle bitches 11 months following OVH [20], although at present it is unclear whether this bears any clinical significance presumably because of the short life-span of most of the dogs used as companion animals.

Surgical complications - Generic surgical risk may be significant in females due to the laparotomy access and of little if any significance in males. Side effects of gonadectomy in bitches and queens have been reported[11, 21-25] and include haemorrage/granuloma of the ovarian or (more commonly) uterine pedicle, suture dehiscence/infection/abscess/edema, peritonitis, evisceration, formation of suture fistulas, retention of a cotton gauze, ureteral ligation with secondary hydronephrosis, formation of vesicovaginal fistula with

secondary hydroureter, ovarian remnant syndrome with or without secondary uterine stump inflammation (Table n° 1). Incidence of these conditions varies depending on OVX or OVH from 7% to 27% in the bitch and 33% in the queen. There is no correlation between incidence of complications and age of the animal, ability of the veterinarian or presence of reproductive diseases. There is no breed predisposition. The complication observed most frequently seems to be (vaginal or intra-abdominal) haemorrhage, which is much more common in large (80% incidence) as opposed to small (20% incidence) size bitches. Vulvar blood loss may occur following OVH (blood coming from the uterine pedicle, the suspensory ligament or the broad ligament) as well as following OVX (blood coming from the ovarian pedicle). Some complications may be due to the stage of the reproductive cycle in which spaying is performed: intraoperative bleeding is more common during proestrus and estrus (due to high estrogen concentrations) while false pregnancy may follow when spaying is performed during diestrus (due to rising prolactin concentrations following an sudden progesterone fall).

	Surgical complications	Occurs after	Incidence (a)
Short term complications	Haemorrhage of ovarian/uterine pedicle (bitch, especially if spayed during heat) (b)	OVH, OVX (*)	80%
	Vaginal haemorrage	OVH, OVX (*)	15%
	False pregnancy	OVH, OVX	reported
	Suture dehiscence, infection, ascesso, edema	OVH, OVX	14-20%
	Peritonitis; evisceration	OVH, OVX	reported
Long term complications	Ovarian remnant sindrome	OVH, OVX	17-43%
	Ligature of ureteris, uretero-vaginal fistula	OVH	11-17%
	Granuloma of the utero/ovarian pedicle	OVH, OVX	6-28%
	Fistula due to use of non-absorbable suture material	OVH, OVX	3-20%
	Cotton gauze in abdomen	OVH, OVX	reported
	Infection of the uterine stump	OVH	reported

Table n° 1 – Short- or long-term complications due to ovariectomy (OVX) or ovariohysterectomy (OVH) in bitches and queens. Based on personal experience and data from literature (a): total number of short- and long-term complications; (b): haemorrhage of the uterine or ovarian pedicle becaude of incomplete ligature or ruptrue of blood vessels). (*) the risk of complications is higher following OVH than OVX.

Heat after gonadectomy in bitches and queens is a well know surgical complication of both OVX/OVH. Ovarian remnants are more commonly found on the right side, and may (but this is not proven) occur more frequently following OVH than OVX because of the fact that the abdominal incision tends to be more caudal in OVH therefore making it more difficult to reach the ovaries. Ureteral obstruction, due to the inclusion of the ureter into the ligature, or the development of a uretero-vaginal fistula are reported only following OVH. Granulomas of the ovarian (less common) or uterine (more common) pedicle may be due to the use of non-absorbable suture material, while inflammation of the uterine pedicle may occur if the most caudal part of the uterus is accidentally caught in the suture during OVH performed

for a presenting complaint of pyometra. Pyometra may develop following OVX if an ovarian remnant is left in place and the bitch/queen resumes regular cycling following surgery, or if progestins are administered for medical reasons later in life.

Anesthetic and surgical issues for early-age patients

Early age patients should be in healthy conditions and normally hydrated before anesthesia and surgery. Also, proper immunization for the most common infectious diseases should have been previously performed. Although vaccination should be a prerequisite, vaccination failures may occur and death of immunized young dogs/cats entering a veterinary facility for castration purposes has been reported during the 7-day period following surgery. From a surgical point of view, neutering at a very early age is a very simple and quick, low-risk procedure. The incidence of short-term post-surgical complications is lower in young (<12 weeks of age) than in young adult (>24 weeks of age) animals and most of the complications are minor problems such as swelling of the abdominal suture. The following guidelines should be followed when performing surgical procedures in very young animals:

• In order to avoid hypoglycaemia fasting prior to surgery should not be longer than 1 hour; also, eating should be allowed as soon as the patient is able to stand

• The use of heating pads and a warm environment during and after surgery is recommended in order to avoid hypothermia which occurs very commonly in young patients; hypothermia prolongs recovery time and slows the metabolism of anesthetic drugs

• Controlling post-operative pain is fundamental to help return to normality thereby stimulating appetite. Anti-inflammatory drugs are know to cause physeal cartilage damage following chronic use, but they may not be harmful following (although there is no information about side effects due to single treatments)

• The opioid petidine provides analgesia and sedation without causing bradycardia; petidine must be given exclusively IM because releases histamine causing pain when given IV

• Avoid bradycardia-inducing drugs (such as alpha-agonists like medetomidine)

• Avoid long-acting drugs (such as acepromazine or tiletamine-zolazepam) as they prolong recovery time

• Use short-acting drugs for induction of anesthesia (such as propofol or alfaxalone) to avoid drug residues during the recovery phase

• Benzodiazepine can be used to increase sedation (thanks to its cardiovascular sparing effect)

• If possible use only volatile anesthesia during surgery

• Atropine or glycopyrrolate are not adviced as they decrease respiratory secretions by reducing the serous component thereby potentially causing the development of dangerous mucous plugs.

• Buprenorphine can be associated as a single shot as it gives a long analgesic effect without CVS depression

• The association ketamina-midazolam can be safely used in kittens as it stimulates the central nervous system without causing any depression of the cardio-respiratory function

Conclusions

Prepubertal neutering is probably an acceptable technique for dogs and cats when it is performed at a shelter. Health risks are minimal and there are advantages due to the lower incidence of side effects when compared to performing it in adult animals. However, the choice of neutering a very young (<3 months) or even an adult pet should be thoroughly discussed with the owner carefully highlighting advantages and disadvantages. Once a decision on neutering has been taken, the option of delaying neutering until the anestrus following puberty in bitches should be considered because of the potential beneficial effects on the development of the urogenital system

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AQUATIC PET MEDICINE AND WORLD RABIES DAY

INTERNATIONAL EFFORTS TO VALIDATE DAY-1 COMPETENCY IN AQUATIC VETERINARY EDUCATION

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The global demand for well-trained aquatic veterinarians represents a huge opportunity for veterinarians willing to expand the services they offer clients. While aquatic animals (fish, amphibians, reptiles, non-agricultural birds and mammals, etc.) are frequently considered exotic or minor species, aquatic veterinary medicine is a well-defined, but poorly recognized veterinary discipline. As the fastest growing veterinary disciple globally, services to aquatic pet (ornamental) and aquaculture (farmed fisheries) represents a largely untapped opportunity for many veterinarians wanting to expand their practices. By example, globally captured and farmed seafood production is now larger than any other animal protein-producing industry (Figure 1) an industry considered by many to be one of the few industries that will feed an ever-growing human population that is expected to reach 9-10 billion by 2050. (Table 1).
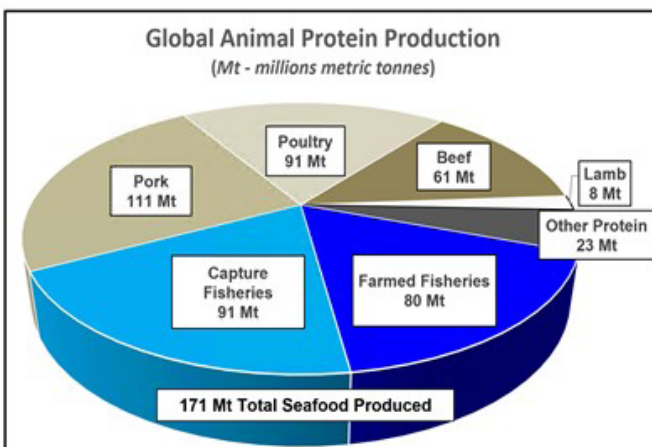
Figure 1.:Current production global animal protein producing industries. Data from FAO (2018) and FishStatJ database (accessible at: http://www.fao.org/fishery/statistics/en).

Similarly, in many countries the number of ornamental (pet) aquatic animal is growing, and frequently exceeds the number of cats or dog owned (AVMA 2012; APPA, 2017; Statista, 2017). But more importantly, by example, in the USA, while 30-35% of households in the U.S. have dogs (60 million) and/or cats (47 million), more than 15 million households (12%) own pet fish almost as many that own all other types of minor species pets, collectively (Figure 2).

Category	2011	2012	2013	2014	2015	2016
Production						
Capture						
Inland	10.7	11.2	11.2	11.3	11.4	11.6
Marine	81.5	78.4	79.4	79.9	81.2	79.3
Total capture	92.2	89.5	90.6	91.2	92.7	90.9
Aquaculture						
Inland	38.6	42.0	44.8	46.9	48.6	51.4
Marine	23.2	24.4	25.4	26.8	27.5	28.7
Total aquaculture	61.8	66.4	70.2	73.7	76.1	80.0
Total world fisheries and aquaculture	154.0	156.0	160.7	164.9	168.7	170.9
Utilization*						
Human consumption	130.0	136.4	140.1	144.8	148.4	151.2
Non-food uses	24.0	19.6	20.6	20.0	20.3	19.7
Population (billions)*	7.0	7.1	7.2	7.3	7.3	7.4
Per capita apparent consumption (kg)	18.5	19.2	19.5	19.9	20.2	20.3

Table 1. *Capture fisheries is progressively declining (from overfishing), while farmed fisheries (aquaculture), considered by many to be one of the few industries able to expand to feed a growing human population, continues to grow exponentially. Data from FAO (2018); * excludes aquatic mammals, reptiles, and plants; b & c estimated for 2014-2016 by the UN in 2015.*
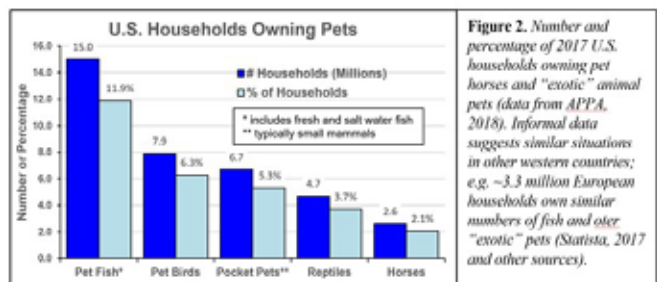
Figure 2. *Number and percentage of 2017 U.S. households owning pet horses and "exotic" animal pets (data from APPA, 2018). Informal data suggests similar situations in other western countries; e.g. ~3.3 million European households own similar numbers of fish and oter "exotic" pets (Statista, 2017 and other sources).*

Unfortunately, increasing impacts of diseases have had enormous impacts on these industries.

Numerous efforts are underway ensure that sufficient numbers of veterinarians are available to support aquaculture industries and producers, government agencies that support or regulate aquaculture, and a myriad of other supporting industries that provide therapeutic products, laboratory diagnostic services, and a number of other services. However, little attention has been given to ensure that veterinary school curricular provide the core (Day-1) knowledge, skills and experience (KSEs) or competency, to service the needs of ornamental (pet) owing, or aquaculture (seafood producing) clients. Without this infrastructure, sustainable and economically viable aquaculture will simply not thrive.

There is therefore a critical need for a well-trained aquatic veterinary workforce[1]

The work presented here therefore outlines the

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progress on efforts to: 1) identify what core competency (knowledge, skills and experience/education or KSEs) are needed for a well-trained aquatic veterinary workforce; 2) evaluate and validate that these KSEs meet the needs of this workforce in delivering services to aquaculture and ornamental aquatic animal producers and owners; and, 3) determine what educational opportunities exist in veterinary school curricular, and how well they address the needs of these industries.

While efforts by several entities address aquatic veterinary education needs, that of the World Aquatic Veterinary Medical Association (WAVMA) stands out. Developed in 2010 the Aquatic Veterinary Certification (CertAqV) Program identifies the core (Day-1) knowledge, skills and experience (KSEs) needed. It also recognizes those veterinarians with sufficient KSE (obtained through a variety of educational opportunities) as competent to practice aquatic veterinary medicine. Specifically, this program identifies nine core subject-matter areas (Modules) that are directly relevant to providing aquatic veterinary services to aquaculture and/or ornamental (pet) producers and animal owners. They primarily focus on finfish, molluscan and crustacean medicine, although the principles are easily applicable to any aquatic taxa, including mammals, birds, reptiles and amphibians.

These Modules include both preclinical and clinical topics that an aquatic veterinarian needs to be familiar with, including:

Preclinical:

1. Anatomy and physiology of aquatic species/taxa;

2. Environmental factors affecting aquatic animal health;

3. Structure and function of aquaculture and ornamental (pet) industries

Clinical:

4. Pathobiology and epidemiology of important aquatic animal diseases;

5. Clinical and laboratory diagnostics used for diagnosing aquatic animal diseases;

6. Availability and appropriate use of therapeutic and biologic agents in aquatic veterinary medicine;

7. Public health, zoonotic diseases and seafood safety issues important in aquatic veterinary medicine;

8. International and national legislation, regulations and policies affecting aquatic veterinary medicine; and

9. Principles of welfare and humane treatment of aquatic animals.

Importantly, in developing the CertAqV Program it was recognized that if creative, adaptive thinking is an integral part of a veterinary curriculum, that 80-90% of the general medical principles learned during veterinary school can easily be applied to aquatic veterinary medicine.

[1A] veterinary workforce includes veterinarians and para-veterinarians (veterinary technicians or nurses, fisheries biologists, and other research scientists who provide important and vital laboratory and other supportive services) who collectively provide the services needed to ensure the health and welfare, and implement optimal measures for the prevention, control and eradication of animal diseases. In most countries, these individuals need to be licensed or registered (DeHaven & Scarfe, 2012).

While some aquatic veterinary organizations and veterinary schools are examining which of these nine Modules are covered in their educational programs or curriculum, whether these meet the needs of aquatic veterinarians in private practice has not yet been validated.

Customarily veterinarians have relied on extracurricular continuing education and professional development (CEPD) programs after graduation or post-graduate degrees, to refine or improve their knowledge, skills and experience (KSEs) to practice in any chosen field. While aquatic CEPD programs are abundant (e.g. Hartman, et al., 2006), little attention has been given to discover how veterinary school curricular address aquatic veterinary medicine.

An early, small study suggested that a number of European universities (not all were at veterinary schools) offer aquatic animal health courses suitable for veterinarians (Weber et al., 2009). Two follow-up surveys, one of European veterinary schools (De Briyne, 2014, personal communication & unpublished data), and another (Scarfe, 2014, personal communication & unpublished data) that focused on N. American and asked questions related to the WAVMA CertAqV Program subject matter, revealed some interesting additional preliminary information: around 60% of veterinary schools in both regions included some aspects of aquatic veterinary medicine into at least one course; 50-60% of these courses were required of all students; most courses were given every year; some of these courses had been taught for >20 years; and, the majority of courses emphasized finfish (vs. other aquatic species).

While no veterinary school covered all Day-1 subjects identified by WAVMA as important to practice aquatic veterinary medicine, these studies and other information suggested that a large number of veterinarians (perhaps 5-10,000) in North America, Western Europe and perhaps Australia/New Zealand, may have sufficient KSEs to support an adequate aquatic veterinary workforce, whereas other regions need improvement. Building on this information we are in the process of evaluating and validating the KSEs needed to practice aquatic veterinary

medicine, and discover what is covered in veterinary curricular throughout the world.

Most academic curricula, syllabi, and courses are developed through a top-down process, where administrators determine what they think students need. However, we have and will continue to utilized a unique bottom-up approach, that involves using input from practicing full-time aquatic veterinarians to identify the key Day-1 KSEs that would be expected of new graduates. This DACUM (Developing a Curriculum) approach has proven to be very effective, relatively quick, and a low-cost approach to accurately develop occupational standards for any job, or work-related tasks. Because of its low cost and effectiveness, it has been and continues to be used by educators and trainers in over 40 countries (Adams, et al, 2015), including veterinary medicine (e.g. Miller, et al., 2004).

The DACUM process involves a panel of 6-12 expert workers the men and women with reputations for being the best at their jobs. Whether at the skilled, technical, supervisory, or professional level, these workers explain exactly what they do, that allows them to be successful (Adams, et al, 2015).

To evaluate the WAVMA CertAqV Modules, we used six aquatic veterinarians who are actively engaged in private aquatic veterinary medicine practice. They participated in a 3-day workshop during which they completed an occupational analysis that identified key General Areas of Competence (GAC equivalent to CertAqV Modules), and the essential competencies (KSEs) within each GAC, that are necessary to practice competent aquatic veterinary medicine. The resulting occupational analysis identified 18 General Areas of Competence and 189 individual competencies essential for the Day-1 practitioner of aquatic veterinary medicine.

Finally, to validate that these are what any aquatic veterinarian needs to practice, 3-5 additional aquatic veterinarians, from different global regions, will be used to validate that these apply to needs in the Americas, Europe, Africa and the Asia-Pacific. This approach is to ensure that significant GACs have not been omitted, weight each competency in relation to its GAC, and ensure needs of of different countries is accommodated. While still in progress, the resulting occupational analysis will serve as the basis for evaluating whether existing veterinary curricula adequately cover sufficient information, and will be useful for developing model curricula to ensure a veterinarian desiring to practice aquatic veterinary medicine has sufficient Day-1 KSEs.

As in all veterinary disciplines, most applied knowledge and skills are refined and honed in the first years of private practice, when delivering services to animal owning clients. This is when veterinarians draw on a vast number of medical principles learned during their

veterinary degree that apply to any species, and apply them to hone their knowledge and skills in dealing with species not traditionally addressed in the veterinary curriculum. This continues through life-long learning, and most countries require veterinarians to document a minimum number of hours of continuing education and professional development (CEPD), as a condition to legally practice veterinary medicine. Aquatic veterinary medicine is no exception.

With the assistance of the Council on International Veterinary Education and collaboration from other national and international veterinary organizations, we hope that efforts in 2019 and beyond, will help evaluate aquatic veterinary education opportunities throughout Africa, Asia, Eastern Europe, South America, and perhaps the Middle East.

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WSV18-0090

SVA EXOTICS

RESCUE AND REHABILITATION OF WILDLIFE IN SINGAPORE AND SOUTHEAST ASIA

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Rescue and Rehabilitation of Wildlife in Singapore and Southeast Asia

Wildlife Reserves Singapore (WRS) is the parent company of the Jurong Bird Park, Night Safari, River Safari and the Singapore Zoo. Through its parks, well-equipped medical facilities and dedicated team of animal management and healthcare specialists, WRS strives to provide the highest standards of care for her living collection. This includes elements of medical management such as disease diagnosis, medical and surgical treatment and prophylaxis. Evidence-based management is central to our health and husbandry practices, and decision-making processes.

Besides managing the four wildlife parks, WRS is also the designated rescue and rehabilitation centre for local wildlife in Singapore, and by doing so; contributes to the management and protection of native biodiversity in the city state. Wildlife Reserves Singapore is a member of several native species working groups and collaborates with key stakeholders and other likeminded organizations (e.g. government agencies and various conservation NGOs) in the rescue, rehab and release of native species.

The increasing human density in Singapore results in increased human-wildlife interactions, and WRS receives injured animals, as well as animals rescued from conflict situations. It does so through two of its veterinary hospitals, i.e. the Wildlife Healthcare and Research Centre (WHRC) in Mandai and the Avian Hospital in Jurong Bird Park. Both hospitals are equipped with the full range of diagnostic and therapeutic equipment, including a full surgical suite, digital radiographs, endoscopes etc. There is a separate treatment area in each hospital for wildlife patients, so that there is no mixing of wildlife and animals belonging to the WRS collection. Each hospital is also staffed by at least one veterinarian and one veterinary nurse every day of the year.

In 2017, the WHRC received 941 wild animals from around Singapore (i.e. 361 mammals, 20 birds, 560 reptiles), while the Jurong Bird Park Avian Hospital received 756 birds. WRS is also involved in regional conservation efforts through funding support, as well as capacity building initiatives. This presentation will illustrate on the institution's role in wildlife conservation in Singapore with specific case examples and touch on efforts in the Southeast Asian region.

WSV18-0290

INTEGRATIVE MEDICINE (LECTURES GIVEN IN MANDARIN CHINESE)

IMPROVING NEUROLOGICAL OUTCOME WITH ACUPUNCTURE AND REHABILITATION

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Introduction

Intervertebral disc disease (IVDD) is commonly seen in dogs in veterinary practice. The clinical signs of IVDD are back or neck pain, trouble walking, lameness, trouble urinating, paresis and/or paralysis. The most common types of thoracolumbar IVDD, are caused by herniation of the nucleus pulposus into the spinal canal (Hansen's Type I), and protrusion of the intervertebral disc into the spinal canal, with the dorsal annulus still coving the disc material (Hansen's Type II). Acupuncture and rehabilitation can be effective at returning to dogs to an ambulatory state, and return of normal function, when used alone or in combination with Western medicine and surgery, for both types of IVDD. Several studies have shown promising effects of using electroacupuncture at treating IVDD in dogs and rat models. This presentation explains the Traditional Chinese Veterinary Medicine (TCVM) etiology and effective treatment modalities, which include physical rehabilitation used in IVDD cases.

TCVM Etiology and Pathophysiology in IVDD

The nervous system is related to the Kidney (bones and spinal cord), the Liver (joints and smooth flow of Qi and blood), and the Spleen (muscle strength). IVDD are often also considered as a Bi syndrome and are usually accompanied by a Wei (weakness) syndrome. There are 2 Excess Patterns and 3 Deficiency Patterns that are associated with various forms of IVDD. The Excess conditions are invasion of WindColdDamp and QiBlood stagnation, which are often associated with acute trauma in chondrodystrophic dogs (Type I). The Deficiency Patterns, often associated with chronic, Type II IVDD in nonchondrodystrophic breeds, include Qi/Yang Deficiency, Yin Deficiency and combined YinQi Deficiency.1

TCVM Treatment for IVDD

1) Acupuncture:

Acupuncture has been proven to be an effective therapy for IVDD.2-8 A general acupuncture treatment plan for a patient with IVDD is as follows:

a) **Dry needle:** GV-20, Liu-feng, BL-60, KID-3, SP-10, LIV-3, BL-40, ST-41, GV-1

b) **Electro-acupuncture:** (20-40 Hz for 10 minutes, 80-120 Hz for 10 minutes, 200 Hz for 10 minutes) at the following pairs of acupoints:

- GV-14 to Bai-hui or GV-1
- Left BL-11 to right Shen-shu
- Right BL-11 to left Shen-shu
- Hua-tuo-jia-ji at or proximal and distal to the suspected or diagnosed disc space, bilateral
- ST-36 to GB-34, or ST-36/GB-34 bilateral
- KID-1 to BL point proximal to IVDD lesion, or KID-1/Liu-feng, bilateral

c) **Aqua-acupuncture** (Vitamin B12) at Hua-tuo-jia-ji at or proximal and distal to the suspected or diagnosed disc space, KID-1, BL-40, LIV-3, LI-4, Liu-feng

d) **Hemo-acupuncture:** use acupuncture needle or 24G hypodermic needle to puncture Jing-well points on the affected limbs and Wei-jian acupoint and get a few drops of blood.

2) Scalp Acupuncture:

a) Motor area: A line starts at GV-22 and extends cranial and ventral to TH-23 on the lateral eyebrow

- GV-22: In the small triangular area formed by the ridges of the frontal crest
- TH-23: In the depression on the rim of the orbit at the end of the eyebrow were it extended to the lateral canthus

b) Sensory area: Starts at GV 20 and extends cranial and ventral to Nao-shu

- GV-20: On the dorsal midline on a line drawn from the tips of the ears level with the ear canals
- Nao-shu: Over the temporalis muscles ⅓ the way along a line from the cranial ear base to the lateral canthus

c) Long insertion methods: use 32-38G acupuncture needle (½" for small dogs and cats; 1" for big dogs) to penetrated subcutaneously the entire motor line (from GV-22 through TH-23) and sensory line (from GV-20 through Nao-shu). Rotate, lift and thrust, or electrical stimulate the needles every 2-3 minutes.

3) Herbal Medicine

General herbal dosage for dogs is 0.5 g per 10-20 lb body weight BID for 2 to 4 months, and then as needed.

a) Da Huo Luo Dan or Double P II (Da Hua Luo Dan modification) is the primary herbal medicine used to treat IVDD. It may cause loose stool in some cases. It can be used as long as the gut is able to tolerate it. Do not give if patient is sensitive to herbal medications or is prone to

diarrhea.

b) Add Bu Yang Huan Wu for Qi or Yang Deficiency (rear weakness, pale and wet tongue, and deep/weak pulse). Better tolerated than Double P II, so can use instead of Double P II if animal is prone to diarrhea.

c) Add Di Gu Pi San for Yin Deficiency (cool-seeking, or rear weakness, red/dry tongue, and fast/thin pulse).

d) Add Hindquarter Formula for Qi and Yin Deficiency (cool-seeking, rear weakness, red or pale tongue, and fast/weak pulse).

e) Add Stasis Breaker if a tumor or mass is present in the spinal cord

f) Add Jie Gu San for fractures of the vertebra.

Physical Rehabilitation for General Practitioners

In conservative cases of acute and recurrent thoracolumbar disk disease, it is important to perform minimal intervention and avoid motions that might risk further herniation of disk material for one month or longer. Patients with cervical or thoracolumbar disk disease are more likely to have proprioceptive and motor deficits, requiring greater attention to balancing and strengthening activities.

A. General exercise:

a) Gentle massage of musculature in the cervical, forelimb, paraspinal, and hindlimb for 10-20 minutes to warm up the muscles or to relieve muscle tensions, BID-TID

b) PROM with bicycling movements all joints of affected limbs, 10-15 reps, BID-TID

B. Proprioception and balancing training:

c) Assisted Standing with abdominal support (e.g. sling, physioball beneath abdomen, standing in water level to the greater trochanter) as needed on a stable, high-traction surface; gradually decreasing support as the patient was able to stand better

d) Weight Shift (weight bearing, balance): Stand behind the dog with the hands placed on either side of the pelvis to support the dog for rear limb weight shift (place on the chest for front limb weight shift). With the limbs in a square standing position, the dog is gently pushed in one direction, and then the other in a slow, rhythmic fashion. Gently push 10-15 times, BID-TID. Make sure this is done on a surface with good traction with the four legs underneath the dog in a normal standing position

e) Challenged Standing (improves weight bearing, balance/proprioception): Have the dog stand on any irregular surface such as couch cushions, foam, trampoline, etc. While standing, can provide gentle pushing to challenge balance, but not too much to lose balance. Repeat 5-10 times during each session, BID-TID.

f) Figure Eights (balance, spinal ROM, weight shifting): Walk the dog on an outline of the number 'eight' on the ground. Start with SLOW speed and BIG Eights. Watch the weight-shifting and weight-bearing carefully. Repeat 5-10 times during each session, BID-TID.

g) Weave Poles (core strengthening/range of motion): Place the cones or bottles close enough together that the dog has to bend its spine sufficiently during the exercise. This distance is typically slightly less than the body length of the dog. Lead the dog carefully through the cones so that the head, neck, and body flex as the poles are negotiated. Repeat 5-10 times during each session, BID-TID.

h) Obstacle Course (balance/proprioception): Place various objects in a hallway or in a line and slowly walk the dog over the obstacles on a short leash. Obstacles can be pillows, cushions, rolled towels, PVC pipes, broom handles, air mattresses, steps, dog beds, etc. Repeat 5-10 times during each session, BID-TID.

C. Strengthening (typically 4 or more weeks postoperatively):

i) Sit to Stand (rear leg strength/ROM): Using a treat or other cue, the dog is placed in a square sitting position. She is then allowed to push up to a standing position. Be certain that the dog sits and stands squarely and symmetrically. Placing an affected leg against a wall or having the dog sit in a corner helps to encourage symmetrical sitting and standing. Repeat this motion 5-10 times during each session, BID-TID. Give only as much support as needed to help encourage the dog to use its muscles

j) Stair Climbing (ROM/strengthen legs/core): Begin with wide, low stairs; avoid open stairs. Gradually increase number and steepness of stairs. Initially, walk dog upstairs 5 steps , and downstairs 5 steps. Repeat 3-5 times, SID-BID. Gradually increase the steps up to 10 each time.

k) Cryotherapy at lesion site as needed for 20 min after exercise

* Caution: Choose only 2-4 exercises per session. Other exercises may be performed as part of a home exercise program. Doing all exercises may result in lameness and pain early in the program

Outcome Measurement

Using a 0-5 grading scale to evaluate clinical neurological signs of IVDD (Table 1), it can be a valuable tool to help choose the mode of treatment, determine the prognosis, and assess the success of treatment. 9 For the most optimum recovery, it is best to use TCVM with decompressive surgery for cases with grades 4 and

5. IVDD with Grades 1 to 3 may be successfully treated with TCVM alone.2-8

A 2010 study evaluated the effectiveness of acupuncture in comparison to decompressive surgery, and a combination of both surgery and acupuncture, in forty dogs that had long standing clinical signs of IVDD (>48 hours). The dogs were re-graded six months after onset of clinical signs, and were considered a success if they returned to ambulation (i.e. they decreased from grade 4/5 to grades 1/2). This research demonstrated that electro-acupuncture had a greater success (79% or 15/19 dogs) than did decompressive surgery alone (40% or 4/10 dogs). Dogs that had both decompressive surgery and electro-acupuncture had an intermediate response (72% or 8/11).2 This study indicates that the duration of clinical signs prior to treatment appears be an important factor in determining if decompressive surgery will benefit the patient. Therefore if the clinical signs of IVDD have persisted for over 48 hours, and the animal is a grade 5 for a prolonged amount of time, electro-acupuncture is the treatment that shows the most benefit to these patients. In addition, if the client is unable to afford surgery, TCVM may be the only potentially effective treatment option.2

Summary

Intervertebral disc disease is commonly seen in small animal clinics. Traditional Chinese Veterinary Medicine (TCVM), including acupuncture, food therapy and herbal medicine, can be an effective singular therapy, or part of integrated therapy with Western medicine and surgery, based on a grading scale of clinical signs and type of IVDD.

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Table 1. Neurological grading scale in canine intervertebral disc disease

Grade	Grading Scale of Clinical Signs *	Recommended Therapy
0	Normal	None
1	Cervical or thoracolumbar pain, hyperesthesia	Acupuncture, herbal medication
2	Ataxia, paresis (muscle weakness) with decreased proprioception, ambulatory (able to walk).	Acupuncture, herbal medication, exercise program
3	Severe paresis with absent proprioception, not ambulatory (not able to walk).	Acupuncture +/- surgery, herbal medication, physical rehabilitation
4	Paralysis{ XE "Paralysis" } (not able to stand/walk), decreased or no bladder control, deep pain perception present	Acupuncture and surgery, herbal medication, physical rehabilitation
5	Paralysis, urinary and fecal incontinence, no deep conscious pain perception	Acupuncture and surgery, herbal medication, physical rehabilitation

NURSES (HILLS)

EMOTIONAL INTELLIGENCE IN THE WORKPLACE

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EMOTIONAL INTELLIGENCE IN THE WORKPLACE

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Developing Emotional Intelligence

Emotional Intelligence – an overview

There are two different kinds of intelligence: the rational one and the emotional one. Our performance in the workplace, and out of it, is determined not only by IQ (Intellectual quotient), but also emotional intelligence, sometimes, also named of emotional quotient (EQ). According to Mayer and Salovey (1997), the emotional intelligence is the capacity of realising and expressing the emotion, assimilating it to the thought, understanding and reasoning with it and being able to regulate it in you and in the others".The leading research on the concept originated with Peter Salovey and John Mayer starting in the late 1980s. In 1990, their seminal paper defined the concept as intelligence. Mayer and Salovey continued to research the concept, and created an emotional intelligence test called the MSCEIT (Mayer-Salovey-Caruso Emotional Intelligence Test). The term "emotional intelligence" was popularised by Daniel Goleman (1995).

Emotional Intelligence Fundamentals

There are four fundamental aspects of EI as measured by the Emotional Competence Inventory:

Competency	Definition
Self-Awareness	Your ability to accurately perceive your emotions and stay aware of them as they happen. This includes keeping on top of how you tend to respond to specific situations and certain people.
Self-Management	Your ability to use awareness of your emotions to stay flexible and positively direct your behaviour. This means managing your emotional reactions to all situations and people.
Social Awareness	Your ability to accurately to pick-up on emotions in other people and understand what is really going on. This often means understanding what other people are thinking and feeling, even if you don't feel the same way.
Social Skill	Your ability to use awareness of your emotions and the emotions of others to manage interactions successfully. Letting emotional awareness guide clear communication and effective handling of conflict.

Recognising and Appreciating the Emotional Strengths and Weaknesses of Others

You will probably find that there are mixed levels of emotional intelligence amongst your colleagues and this in itself can cause issues and conflicts because they may not manage their emotions in the same way as another, or how you would manage your own emotions. You need to recognise where on the scale of emotional intelligence each of those people in your team lies and respond to their emotional states accordingly.

Your emotional responses will differ to those of your colleagues because we all have different personalities and emotional strengths and weaknesses. Something that motivates you may not evoke the same drive from another staff member. Likewise, something that concerns another another staff member, e.g., a sick family member, may affect their behaviour towards others in the workplace or it might affect their performance and ability to do their job properly. The emotions this person is experiencing are potential causes of conflict, particularly if other staff members do not understand what is driving this behaviour.

You need to be able to recognise the emotional strengths and weaknesses of others within your team and the emotional states that they produce. The state we are in determines how we perceive something that is happening to us or around us which results in the emotion we feel towards it. The emotion we feel to the same stimulus may be completely different depending on the state we are in.

Benefits of Emotional Intelligence to the Workplace

Encouraging your workforce to develop their own emotional intelligence helps them to build productive relationships not only in the workplace, but enhances their personal relationships as they gain confidence to manage their emotions and relationships rationally and thoughtfully. It develops their prospects for promotion and consequent rise in salary as well a boost to their overall self-confidence.

The following chart that demonstrates the potential workplace outcomes of developing emotional intelligence within an organisation, was researched and compiled by Dr Benjamin Palmer and Professor Con Stough from Swinburne University and is based upon their seven-factor model of emotional intelligence.

Emotional intelligence skill	Definition	Workplace outcome
Emotional self-awareness	The skill of perceiving and understanding one's own emotions	· The capacity to identify and understand the impact one's own feelings is having on thoughts, decisions, behaviour and performance at work · Greater self-awareness

Emotional expression	The skill of effectively expressing one's own emotions	· Creating greater understanding amongst colleagues about yourself · Creating trust and perceptions of genuineness amongst colleagues
Emotional awareness of others	The skill of perceiving and understanding others' emotions	· Greater understanding of others, how to engage, respond, motivate and connect with them · Interpersonal effectiveness
Emotional reasoning	The skill of utilising emotional information in decision-making	· Enhanced decision-making where more information is considered in the process · Greater buy-in from others into decisions that are made
Emotional self-manage-ment	The skill of effectively managing one's own emotions	· Improved job satisfaction and engagement · Improved ability to cope with high work demands · Greater interpersonal effectiveness · Enhanced productivity and performance
Emotional management of others	The skill of influencing the moods and emotions of others	· The capacity to generate greater productivity and performance from others · The capacity to generate a positive and satisfying work environment for others · The capacity to effectively deal with workplace conflict
Emotional self-control	The skill of effectively controlling strong emo-tions experienced	· Emotional well-being · The capacity to think clearly in stressful situations · The capacity to deal effectively with situations that cause strong emotions

You can read more at: http://www.eiconsortium.org/measures/genos.html

Use Emotional Intelligence to Maximise Team OutcomesWhen team members have a strong relationship they are more likely to sustain positive emotions and a positive mind set. This maintenance of positive energy then breeds ideas and creativity which results in innovation and increased productivity.

The satisfaction the team members enjoy from their successes increases motivation for further success and a further increase in productivity. As the positive mood and emotions continue, the members of the team then seek to take on new challenges with other teams, increasing the collaboration growing the cohesion of the community within the organisation. The more the workers collaborate, share success and satisfaction as a whole, the less competition there is for allocation of resources as it becomes a shared ownership for the benefit of the whole organisation.

Individual members of the work force start to see themselves as part of the fabric of the organisation instead of individual workers and view themselves as 'we, the organisation', and not just 'I'. The positive environment creates a work place that is fun, satisfying, productive, supportive and innovative, and one in which the work force takes ownership and responsibility for the part they play in its success. Any negative emotions emanating from an individual are quickly negated by the wave of positivity around them.

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43RD WORLD SMALL ANIMAL VETERINARY ASSOCIATION CONGRESS AND 9TH FASAVA CONGRESS

WSV18-0233

HOW TO ASSESS PERIOPERATIVE PAIN IN DOGS AND CATS

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RECOGNITION AND ASSESSMENT OF ACUTE PAIN IN CATS

Acute pain is the result of a traumatic, surgical, medical or infectious event that begins abruptly and should be relatively brief. This pain can usually be alleviated by the correct choice of analgesic drugs, most commonly opioids and non-steroidal anti-inflammatory drugs (NSAIDs). For successful relief of pain, one must first look for it and recognize it. It is recommended that assessment of pain is incorporated into Temperature, Pulse and Respiration (TPR) examinations, making pain the 4th vital sign we monitor. Cats that have been injured or undergone surgery should be monitored closely and pain must be treated promptly to prevent it from escalating. Treatment must be continued until the acute inflammatory response abates. The degree of trauma dictates the intensity and duration of the inflammatory response but treatment may be required for several days. Feral cats require preemptive administration of analgesics based on the severity of the proposed surgical procedure rather than based on their behaviour; in addition, interactive pain assessment is not possible in this population.[6]

Neuroendocrine assays measuring β-endorphin, catecholamines and cortisol concentrations in plasma have been correlated with acute pain in cats; however, these are also influenced by other factors such as anxiety, stress, fear and drugs.[7] Currently there is no validated pain scoring system for use in cats. Objective measurements such as heart rate, pupil size and respiratory rate have not been consistently correlated with signs of pain in cats – therefore we depend on subjective evaluation based on behaviour.[8]

Pain assessment and recognition

Take into consideration the type, anatomical location and duration of surgery, the environment, individual variation, age, and health status. The cat should be observed from a distance then, if possible, the caregiver should interact with the cat and palpate the painful area to fully assess the cat's pain. A good knowledge of the cat's normal behaviour is very helpful as changes in behaviour (absence of normal behaviours such as grooming and climbing into the litter box) and presence of new behaviours (a previously friendly cat becoming aggressive, hiding or trying to escape) may provide helpful clues. Some cats may not display clear overt behaviours indicative of pain, especially in the presence of human beings, other animals or in stressful situations. Cats should not be awakened to check their pain status; rest and sleep are good signs of comfort but one should ensure the cat is resting or sleeping in a normal posture (relaxed, curled up). In some cases cats will remain very still because they are afraid or it is too painful to move, and some cats feign sleep when stressed.[9]

Facial expressions and postures: these can be altered in cats experiencing pain: furrowed brow, orbital squeezing (squinted eyes) and a hanging head (head down) can be indicators of pain. Following abdominal surgery a hunched position and/or a tense abdomen is indicative of pain. Abnormal gait or shifting of weight and sitting or lying in abnormal positions may reflect discomfort and protection of an injured area. Comfortable cats should display normal facial expressions, postures and movement after successful analgesic therapy. Figure 1 provides examples of normal postures and facial expressions and those that may be indicative of pain.

Behavioural changes associated with acute pain in cats: reduced activity, loss of appetite, quietness, hiding, hissing and growling (vocalization), excessive licking of a specific area of the body (usually involving surgical wounds), guarding behaviour, cessation of grooming, tail flicking and aggression may be observed. Cats in severe pain are usually depressed, immobile and silent. They will appear tense and distant from their environment.[10]

Dysphoria versus pain: thrashing, restlessness and continuous activity can be signs of severe pain in cats. However, these can also be related to dysphoria. Dysphoria is usually restricted to the early postoperative period (20–30 min) and/or associated with poor anaesthetic recoveries after inhalant anaesthesia and/or ketamine administration and/or after high doses of opioids. Hyperthermia associated with the administration of hydromorphone and some other opioids may lead to anxiety and signs of agitation in cats.

Figure 1. Illustrations of normal postures and facial expressions and those that may be indicative of pain. (A) A cat with a normal posture – the cat's head is up, the cat is alert and the eyes are open. (B) A cat resting after surgery in a normal relaxed and curled up position. (C) This cat is 'flat out' and tense after surgery – also note the facial expression. (D) and (E) These cats have had abdominal surgery; the hunched posture and low hung head are suggestive of pain. Note also that the eyes are either held shut or half closed and appear "slanted" or "squinted" compared to the cat in Figure 1A.

Fig 1A

anaesthetic drugs affect them. Thus, evaluation of pain in dogs is primarily subjective and based on behavioural signs.

Pain recognition

Behavioural expression of pain is species-specific and is influenced by age, breed, individual temperament and the presence of additional stressors such as anxiety or fear. Debilitating disease can dramatically reduce the range of behavioural indicators of pain that the animal would normally show e.g. dogs may not vocalize and may be reluctant to move to prevent worsening pain. Therefore, when assessing a dog for pain a range of factors should be considered, including the type, anatomical location and duration of surgery, the medical problem, or extent of injury. It is helpful to know the dog's normal behaviour; however, this is not always practical and strangers, other dogs, and many analgesic and other drugs (e.g. sedatives) may inhibit the dog's normal behavioural repertoire.

Behavioural signs of pain in dogs include:
- change in posture or body position (Figures 2 and 3)
- change in demeanour (Figure 4)
- vocalization
- altered reaction to touch
- altered interaction with people (e.g. reduced interaction, aggression)
- altered mobility (e.g. lameness, reluctance to move)
- reduction in appetite.

Pain assessment protocol

The most important step in managing acute pain well is to actively assess the dog for signs of pain on a regular basis, and use the outcomes of these assessments (through observation and interaction) along with knowledge of the disease/surgical status and history of the animal to make a judgement on the pain state of the dog. It is recommended that carers adopt a specific protocol and approach every dog in a consistent manner to assess them for pain. Dysphoria should be considered where panting, nausea, vomiting or vocalization occurs immediately following opioid administration.
- Observe the dog in its kennel/bed and consider its demeanor and posture
- Approach the dog and interact with it, calling its name, and consider its response
- Touch the dog (around a wound/damaged tissue as appropriate), and consider its response (normal, aggressive, flinching etc.).

Where a dog is judged to be in pain, treatment should be given immediately to provide relief. Dogs should be assessed continuously to ensure that treatment has been effective, and thereafter on a 2–4 hourly basis.

Pain measurement tools: these should possess the key properties of validity, reliability and sensitivity to change.

1. **RECOGNITION AND ASSESSMENT OF ACUTE PAIN IN DOGS**Acute pain occurs commonly in dogs as a result of a trauma, surgery, medical problems, infections or inflammatory disease. The severity of pain can range from very mild to very severe. The duration of pain can be expected to be from a few hours to several days. It is generally well managed by the use of analgesic drugs. The effective management of pain relies on the ability of the veterinarian, animal health technician and veterinary nurse to recognize pain, and assess and measure it in a reliable manner. When the dog is discharged home, owners should be given guidance on signs of pain and how to treat it.

Objective measurements including heart rate, arterial blood pressure and plasma cortisol and catecholamine levels have been associated with acute pain in dogs;[11] however, they are unreliable since stress, fear and

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Pain is an abstract construct so there is no gold standard for measurement and since the goal is to measure the affective component of pain (i.e. how it makes the dog feel), this is a real challenge. This is further compounded by the use of an observer to rate the dog's pain. Few of the scales available for use in dogs have been fully validated. Simple uni-dimensional scales, including the Numerical Rating Scale (NRS), the Visual Analogue Scale (VAS) and the Simple Descriptive Scale (SDS) (Figure 5), have been used.[12,13] These scales require the user to record a subjective score for pain intensity. When using these scales, the observer's judgment can be affected by factors such as age, gender, personal health and clinical experience, thus introducing a degree of inter-observer variability and limiting the reliability of the scale. However, when used consistently, these are effective as part of a protocol to evaluate pain as described above. Of the three types of scales described (and there are others in this category), the NRS (0 to 10) is recommended for use due to its enhanced sensitivity over the SDS and increased reliability over the VAS.

Composite scales include the Glasgow Composite Measure Pain Scale and its short form (CMPS-SF),[14,15] and the French Association for Animal Anaesthesia and Analgesia pain scoring system, the 4A-Vet.[15] The CMPS-SF, validated for use in measuring acute pain, is a clinical decision-making tool when used in conjunction with clinical judgement. Intervention level scores have been described (i.e. the score at which analgesia should be administered), thus it can be used to indicate the need for analgesic treatment. The instrument is available to download online.[16] The 4A-Vet, which is also available online,[17] is available for use in cats and dogs, although evidence for its validity and reliability have not yet been demonstrated. The Colorado State University (CSU) acute pain scale for the dog[18] combines aspects of the numerical rating scale along with composite behavioural observation, and it has been shown to increase awareness of behavioural changes associated with pain. The University of Melbourne Pain Scale combines physiologic data and behavioural responses.[19] Japanese Society of Study for Animal Pain Canine Acute Pain Scale (written in Japanese) is a numerical rating scale combined with behavioural observation and can be downloaded from the website.[20] All of the composite scales above are easy to use and include interactive components and behavioural categories.

Figure 2. (A) Post-laparotomy (B) severe dermatitis

Fig 2A

Fig 2B

Figure 3. Gastro-esophageal pain

Figure 4. (A) Pancreatitis, painful

(B) pancreatitis, pain free

Fig 4A

Fig 4B

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i) Simple Descriptive Scale (SDS)
No pain, Mild pain, Moderate pain, Severe Pain
Categories may be assigned numbers for data collection purposes; however, they are not numerical values.

ii) Numerical Rating Scale (NRS)
0, 1, 2, 3, 4, 5, 6, 7, 8, 9, 10.
0: no pain; 10, maximum pain possible

iii) Visual Analogue Scale (VAS)
|------------------------X--|
No pain   maximum pain possible

Using the scales: The observer makes an assessment of the amount of pain a dog is experiencing based on their observation of and interaction with the dog, and their clinical judgement. A category (SDS) or number (NRS) is selected, or a mark made on the line (VAS) to reflect that judgement.

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WSAVA GLOBAL NUTRITION

NUTRITION DISASTERS AND HOW TO SPOT THEM

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The basics of critical care nutrition include an initial patient assessment, the prescribing an appropriate diet whilst being mind-full of the diagnosis and disease severity, deciding on a method of delivery of that diet, setting goals for the nutritional intervention, and finally assessing whether these goals are in fact being met. The goals of critical care nutrition are to meet resting energy requirements as well as to supply sufficient essential and conditionally essential amino acids and all other micro and macro nutrient needs of that patient. A plethora of methods are available to us to achieve this, including a variety of diet and feeding tube options for the still preferred enteral nutrition route as well as multiple solution options and peripheral and central line options for parental nutrition. In some cases, partial parenteral and enteral nutrition best supplies the patient's nutritional needs.

WSV18-0247

AQUATIC PET MEDICINE AND WORLD RABIES DAY

SELECTED CASE STUDIES IN ORNAMENTAL FISH MEDICINE

N. Saint-Erne[1]

[1]World Aquatic Veterinary medical Association, PetSmart, Phoenix, USA

This presentation will include case studies of diseases and treatments of aquarium fish by three different aquatic veterinarians.

WSV18-0293

WSAVA GLOBAL PAIN

EPIDURAL ANESTHESIA - AN IN-DEPTH HOW-TO

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The administration of drugs with analgesic properties via the epidural route provides effective anaesthesia and analgesia for procedures and painful pathologies involving the pelvis and pelvic limbs, the tail, perineum and abdomen. Furthermore epidural administration can be used to provide analgesia of the thorax. The advantages of epidural administration of analgesics and local anaesthetics are well known: it reduces the need for other anaesthetics, improves haemodynamic function and muscle relaxation, facilitates intestinal surgery and accelerates recovery, reduces the likelihood of adverse effects associated with systemic administration of opioids.

Contraindications:

Infection or neoplasia at the injection site; bleeding disorders (coagulopathy, thrombocytopenia); hypovolemia/hypotension (if using local anaesthetics); septicaemia; (controversial); traumatic or congenital anatomical abnormalities – if landmarks cannot be identified

Equipment
- Medium to large dogs: 20-gauge spinal/Tuohy needles in varying lengths (1.5 – 3.5 inches)
- Small dogs and cats: 22-gauge spinal/Tuohy needles in varying lengths (1.5 – 3.5 inches)

Positioning

Patients may be positioned in either sternal or lateral recumbency depending on the patient's medical condition or clinician's preference. Sternal recumbency has several advantages as it is easier for the veterinarian to keep the spinal needle in the correct planes and orientation, it also allows use of the "hanging drop" technique for confirmation of the correct needle placement. The pelvic limbs may be positioned in one of two ways while the patient is in sternal recumbency:
- Rostral extension of the pelvic limbs – increases lumbo-sacral (LS) and L6 – L7 distance.
- "Frog-legged" with the pelvic limbs resting on the stifle and the feet extended posteriorly. This position may allow easier palpation of anatomical landmarks in obese patients.

A lumbo-sacral epidural may be administered with patients positioned in lateral recumbency, with the pelvic limbs taped or held in a rostral position by an assistance. This position may be preferable in cases not amenable to sternal positioning, such as femoral fracture or severe

pelvic fractures.

Anatomic landmarks

- **Lumbo-Sacral Epidural:** The LS intervertebral space is located by palpation of the cranial border of the iliac crests using the the thumb and third finger of the non-dominant hand; an imaginary line between them runs over the L6-to-L7 space, which is palpated with the index finger. The index finger is placed on the patient's midline and palpates the LS space as a depression on the midline. The index finger is also used to confirm the proper space by palpating cranially (the dorsal spinous process of L6 is larger than L7) and caudally (the sacrum does not have intervertebral spaces).

- **Caudal Epidural:** Palpate the space between the sacrum - Cd1 or Cd1 - Cd2, which can be easily identified by having a team member move the tail up and down

Preparation

The injection site should be clipped to ensure hairless margins laterally to the aspects of the ilial wings, cranially to L3-4 and caudally to S3-Co1. The skin is prepared aseptically with in a standard pre-surgical fashion to avoid infection/abscess of the epidural space and discospondylitis.

Technique

In most instances the pre-emptive approach to pain management in surgical patients' results in epidural injections performed after induction to general anaesthesia before the start of surgery. Epidural injections and catheter placement may under certain circumstances be performed on sedated patients (most commonly in the intensive care unit), with superficial infiltration of local anaesthetic to facilitate the procedure. Strict aseptic technique should be followed throughout the epidural injection or epidural catheter placement. Sterile gloves as well as a facemask and hair covering should be worn. A sterile fenestrated drape should be placed over the intended needle insertion site. A sterile table drape can assist in maintaining a sterile field with which to place all required equipment and drugs. Under sterile conditions, the needle is introduced perpendicular to the skin (with the bevel of the needle directed cranially) while the index finger of the palpating hand remains in the L-S intervertebral space to ensure accurate positioning. Adjustments to the angle of insertion can be made as required to facilitate correct placement in the epidural space. Once the needle has traversed through the skin and subcutaneous tissue the stylet is removed. The hub of the needle is then filled with sterile saline to facilitate the "hanging drop" confirmation technique. The needle is then advanced further. As the needle advances, a ''pop'' maybe felt when it pierces the ligamentum flavum, and the needle is introduced into the epidural space. Once the ligament is penetrated, the "hanging drop" solution is the fluid is

aspirated into the needle shaft by the sub-atmospheric epidural pressure. In dogs, I recommend advancing the needle all the way to the floor of the epidural space in and then withdrawing 1 to 2 mm; in this way, the position of the needle is ensured in the epidural space and being off midline can be ruled out. In cats, the presence of the spinal dura mater beyond L7 makes it likely that CSF is obtained if the needle is advanced to the floor; therefore, it is best avoided. Instead, flicking of the tail, movement of the hind limbs, or twitching of the skin in the area of the L-S intervertebral space is commonly observed in cats when the needle enters the epidural space and pricks the spinal cord or cauda equina, without subsequent adverse effects; however, for this reason, smaller gauge spinal needles are recommended in cats. To verify correct placement of the needle, several tests can be performed. A plastic or glass syringe, specifically designed to offer minimal resistance, can be attached to the needle, and air can be injected to detect "loss of resistance" on injection because of the sub-atmospheric pressure of the epidural space.

Advanced confirmation techniques

Epidural pulse wave measurement: For the epidural pressure waveform method, the epidural needle or catheter is connected to a pressure transducer, volume is injected into the space, and waveforms are observed on the monitor. The presence of the injected fluid in the epidural space facilitates transmission from CSF pressures and allows arterial pulsations to be visible.

Electro-location: A shielded nerve stimulator or Tuohy needle is primed with 0.2 to 1 mL of saline, and connected to a peripheral nerve stimulator set to deliver a current at 1 Hz, with a pulse width of 0.2 m sec. Initially the current is set at 1.2 mA as the needle is advanced into position. Confirmation of epidural needle placement is confirmed when twitches were observed in the pelvic limbs and/or tail. The lowest mean (range) current reported to elicit pelvic limb twitches is 0.72 mA (0.4–1.0 mA); lowest mean (range) current reported to elicit tail twitches is 0.58 mA (0.4–1.0 mA).; tail twitches were reliably lost at a mean current of 0.37 mA (0.2–0.8).

Ultrasound: Ultrasound-guided epidural injection has been described in dogs.[1]

Injection

Prior to injection, the hub of the needle should be observed for the presence of CSF or blood. If CSF is obtained during epidural attempts, withdrawing the needle slowly may reposition the needle back into the epidural space. It is not recommended to inject an epidural dose intrathecally. It is recommended to reduce the dose by 25% to 50%. If blood, is obtained during epidural attempts, withdraw the needle and redirect your approach or consider an alternative location (e.g. L6 – L7, between the sacrum - Cd1 or Cd1 - Cd2) using a

new needle. DO NOT inject drugs intended for epidural administration if blood is observed as inadvertent IV injection of LA can cause systemic toxicity (convulsions, cardiovascular depression). Bupivacaine (and to a lesser extent ropivacaine), may cause cardiac arrest due to increased cardiotoxicity. Therefore, it is recommended to always aspirate before injection.

Size differences

The spinal cord typically ends at the level of the L6 vertebra in medium/large adult dogs, however, in cats and in small or young dogs, the spinal cord and meninges may extend to the level of the L7 making piercing of meninges and leakage of CSF more likely compared to medium/large dogs.

Drugs

Adetailed review of current drugs used for epidural anaesthesia and analgesia by Steagall and colleagues (2017)[2] is freely available on-line: https://www.frontiersin.org/articles/10.3389/fvets.2017.00068/full

The most commonly used drugs for epidural administration are opioids, local anaesthetics or a combination of the two drugs.

- **Local anaesthetics:** The site of action for local anaesthetics administered in the epidural space is primarily the spinal nerve roots. Local anaesthetics result in autonomic, sensory and motor blockade. Bupivacaine is the most commonly used local anaesthetic drugs due to their longer duration of analgesia of two to four hours. Ropivacaine has the advantage of being less arrhythmogenic and toxic for the CNS and cardiovascular system.
- **Bupivicaine 0.5% Dosing:** 0.5-1.0 mg/kg
- **Ropivicaine 0.75% Dosing:** 1.0-1.65 mg/kg
- **Opioids:** The site of action for epidural administered opioids is the opioid receptors in the dorsal horn of the spinal cord. They provide segmental analgesia without sensory, sympathetic or motor blockade. Morphine is the most widely used as it is the least lipid-soluble of the commonly used opioids and, therefore, has the slowest onset of action (30 to 60 minutes) but the longest duration of action (up to 24 hours). Preservative-free morphine is recommended.
- **Morphine Dosing:** 0.1 mg/kg (diluted with saline to 0.3 mL/kg)
- **Local anaesthetic/opioid combinations:** The combination of opioids with local anaesthetics may be beneficial because affinity of opioid drugs for their receptors in the spinal cord is increased by local anaesthetics

Volume

In small animals, a total epidural volume of injectate that approximates 0.2 mL/kg but does not exceed 6 mL for animals has been recommended.[3] For drugs that do not cause sympathetic or motor blockade, such as the opioids, it is not necessary to adhere to this rule. Volumes greater than 0.2 ml/kg which contain local anaesthetic may spread cranially and potentiate motor blockade of the diaphragm (phrenic nerves).

Volume Guide:
- **Anaesthesia/analgesia caudal to the diaphragm:** 0.2 ml/kg (combined volume opioid + LA)
- **Opioid ONLY analgesia to the thorax:** 0.3 ml/kg (DO NOT combine with LA at this dose)

Complications

Reported complications with the epidural administration of opioids and local anaesthetic agents are rare, but may include respiratory depression, pruritus (reported in humans), hypotension, nausea, vomiting, delayed hair regrowth and urinary retention. Urinary bladder management should include emptying the bladder at the time of surgery (expression or urinary catheterization) and monitoring bladder size every four to six hours postoperatively until the patient is able to urinate.

Indwelling Epidural Catheter Placement Technique

Catheterization of the epidural space provides the opportunity for repeated or constant delivery of analgesics to the spinal cord, and is usually accomplished by using commercial kits.

STEPS:
- Anatomical landmarks are confirmed and the patient is prepared as previously described.
- The length of epidural catheter to be placed inside the patient is then pre-measured by carefully placing the catheter over the sterile drape against the patient. For severe cranial abdominal pain the tip of the catheter should be advanced to the level of L1-2 or L2-3. For pelvic origin pain, the catheter is advanced only to the level of L5-6. Remember to include the distance from the skin surface to the epidural space in the estimation of catheter length needed. Mark the catheter with a sterile pen (often included in the catheter kit) or utilise the reference markings if present on the catheter (brand specific).
- The mark just created will not be visible during placement, so to assist in accurate placement a second mark exactly the same length of the Tuohy spinal needle is placed on the catheter. The tip of Tuohy spinal needle is placed at the mark created above and a second mark is placed on the catheter at the level of the catheter hub.
- The epidural space that has been chosen for insertion is then carefully palpated again and the thumb of the non-needle placing hand is firmly embedded into the depression between L7-S1 or L6-7. The Tuohy spinal needle is then inserted into the desired epidural space. Correct placement is then confirmed using the previously mentioned techniques.
- Once a positive placement has been confirmed, the catheter guide is placed on the epidural catheter and the appropriate tip is inserted into the Tuohy needle hub. The catheter guide is then gently seated into the Tuohy needle hub and the catheter is gently advanced to the tip of the needle. Be careful not to disrupt the placement of the tip of the Tuohy needle. Gentle resistance should be felt as the catheter tip

makes the turn at the tip of the Tuohy needle on its way cranially. After this the catheter should advance smoothly with minimal resistance. If difficulty is encountered in attempting to advance the catheter past the tip of the needle, then withdraw the catheter to ensure the tip is within the barrel of the Tuohy needle once again. Then gently put cranial pressure on the hub of the Tuohy needle and move it cranially 5 to 25mm and attempt to advance the catheter once again. If resistance is encountered again, then the needle placement is likely incorrect. The catheter should be withdrawn, and needle placement rechecked. If a positive site for advancement of the catheter with minimal resistance is not found after 2-3 successive attempts then needle placement should take place with fluoroscopic guidance.

- Once the catheter advances with minimal resistance then the catheter should be advanced until the second mark on the catheter is entering the catheter hub. Advance the catheter 10-25mm further and then gently withdraw the Tuohy needle, the catheter guidewire, and the catheter guide over the catheter and completely remove them from the catheter.

- The second mark on the catheter should now be visible outside the patient. If the first mark is not visible then withdraw the catheter gently until the first mark is visible outside the patient.

- The catheter is then cut to allow 10 to 20cm of catheter to remain outside the patient.

- The catheter tip is then gently inserted into the tapered end of the injection adapter until resistance is met, withdrawn 1-2mm and then the hub is tightened.

- The floating luer end of the 0.22 micron filter device is then attached firmly to the hub of the catheter adapter after removing the plastic coverings of both devices. The luer tip catheter injection port is then firmly attached to the other end of the 0.22 micron filter device after removing the plastic coverings of both devices.

- Catheter placement is then verified by attempting to inject a small quantity of preservative free 0.9% sterile saline through the filter device and into the catheter. Because of the small inside diameter of the catheter, the resistance to injection is large. As long as injectate continues to flow, then catheter placement is confirmed. Radiographic verification is recommended to confirm correct placement of the epidural catheter tip.

Carefully attach a clean tape butterfly to the catheter near the skin-catheter interface and suture or staple the tape butterfly to the patient. A second clean tape butterfly is attached to the filter device and stapled or sutured to the patient.

- The skin around the catheter-skin interface is wiped again with an antimicrobial skin preparation and a small amount of sterile antimicrobial ointment (betadine or chlorhexidine) is placed at the catheter-skin interface. An occlusive plastic skin drape is then placed over the entire area to secure the catheter and prevent accidental removal.

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WSV18-0039

HOMEMADE DIETS: WHEN AND HOW TO USE THEM

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HOMEMADE DIETS: WHEN AND HOW TO USE THEM

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Commercial diets are frequently used due to their cost and convenience. The number of calories provided by commercial food varies depending on the region but can be very high in some countries (up to 90% in the USA). Some pet owners home cook for their pet. There are several reasons as to why. Some of those reasons[1] include palatability (some dogs and cats do not find overall commercial diets appetent, even though there are hundreds of options in the market), mistrust of processing or ingredients, a wish to feed pet according to their own feeding habits/philosophy (vegan, organic, etc), wish to include/exclude certain ingredients, or a wish to have a tighter control over what is being fed to their dog or cat. Finally, some owners choose home cooking because there are no commercial diets that are adequate, for example, when a pet has multiple diseases.

Independently of the reason, a homemade diet for a dog or a cat should be formulated in a way to ensure that all nutritional and energy needs are met.

How are homemade diets formulated?

The formulator needs to know

1) the nutritional and energy requirements of the species and life stage of the pet,

2) any maximum/safe upper limits set for specific nutrients for species and life stage, and

3) the nutritional and energy content of the foodstuffs chosen to formulate the diet.

Nutrition and energy needs are compiled by the NRC[2] (2006) from experimental data. Different associations (like AAFCO in the USA, or FEDIAF in Europe) base their recommendations for commercial diets on the NRC values. These are for healthy pets, and any modifications required for disease needs to be checked in the existing literature. The NRC also reports safe upper limits for those nutrients that can have deleterious effects when fed in excess (such as vitamins A and D).

In order to meet these requirements and avoid toxic levels, the formulator will combine different ingredients (plus any required supplements). The nutrient and energy content of these ingredients is obtained from databases, usually public ones that each country maintains. These are usually less complete than pet food manufacturers' internal database and do not contain ingredients that humans do not commonly eat. A home cooked diet is as good as the database, and all have serious limitations.

Risks associated with homemade diets

This is common when diets are formulated by lay people (not a veterinarian or a veterinary nutritionist) and if generic recipes are obtained from books or the internet. One study[3] found that a vast majority of diets published in books and online for healthy dogs had nutritional inadequacies, plus almost no guidance on calorie intake. Similar results were observed in diets for pets with cancer[4] and kidney disease[5].

Other problems associated to "generic" online and book recipes include outdated strategies or nutritional requirements, over-complexity or simplicity, and use of dangerous ingredients (such as garlic). And they do not have the main benefit of homemade diets: customization to the pet.

Homemade diets properly formulated are prone to be changed over time[6] ("diet drift"), and these changes can result in the diet becoming inadequate for long term feeding or for the disease it was formulated for. The reasons owners give for these changes include lack of palatability, inability to obtain a specific ingredient, etc.

Even in properly formulated diets, database limitations include an element of risk. Homemade diets are not analyzed after preparation, and their formulation relies on theoretical values provided by the database. This means that the following assumptions are made: that he database reflects accurately the nutritional composition of the ingredients used; and that digestibility, bioavailability, and energy density of the ingredients is the same as in humans (most databases used for homemade diets are for people). Moreover, databases for human foodstuffs do not include concentration of some important nutrients for pets such as taurine and choline. Also, some ingredients are missing from the database, or we have incomplete entries. Moreover, the rarer the ingredient, the less reliable the database.

Finally, homemade diets, when properly done, are usually more expensive than commercial dry food, plus they require an important time and space investment.

Pros of homemade diets: when to use them

Homemade diets are a good option in several situations, as long as they are complete and balanced, and the patient has frequent veterinary visits. Homemade diets have the ability to be fully customized to patient and

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owner, and this is particularly useful when there are no commercial diets available for the patient.

Finicky patients

Homemade diets might be better consumed by dogs and cats with capricious appetite, although this is not always the case. The owner must have realistic expectations, especially when the pet's disease requires protein or fat moderation.

Concomitant diseases

In some cases, the combination of diseases in one pet makes finding a commercial option difficult, although there are more and more "multifunction" diets in the market. Homemade diets, in these cases, allow provision of nutritional modifications without sacrificing important nutritional strategies for one or more conditions.

Electrolyte alterations

One study[7] reported the usefulness of low potassium homemade diets in dogs with chronic kidney disease and hyperkalemia. Other electrolyte disturbances where homemade diets might help include idiopathic hypercalcemia in cats, although we do not have published data at this time.

High digestibility

Homemade diets can be more digestible than commercial diets and this can be helpful in some gastrointestinal diseases (such as short bowel syndrome.

Ultra-low fat diets

Despite the presence of low fat diets in the market, especially for dogs, there are some patients with fat sensitive diseases that will require more aggressive fat restriction, such as some instances of hypertriglyceridemia, chronic pancreatitis, and lymphangiectasia. It is very important that these diets still provide enough essential fatty acids, thus, their formulation requires skill.

Resources

In order to obtain a homemade diet, it is recommended to contact a boarded veterinary nutrition specialist. In the US, the specialists are part of the American College of Veterinary Nutrition (www.acvn.org), whereas in Europe they are part of the European College of Veterinary and Comparative Nutrition (http://www.esvcn.eu/college/animal-owners). Other countries can have their own veterinary nutrition specialization organization, and the veterinary schools or the veterinary licensing bodies can also be contacted for information. Many services can work remotely through a referring veterinarian.

Patients eating homemade diets should be visited at least twice yearly (or even more, depending on the health status of the patient) to undergo a complete nutritional evaluation[8] assessing the pet, the diet (in detail), and the environment, to ensure the homemade diet is being used properly and to assess if adjustments are needed. The WSAVA website has useful tools to perform this evaluation (http://www.wsava.org/Guidelines/Global-Nutrition-Guidelines).

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SVA SOFT TISSUE SURGERY

SURGICAL MANAGEMENT OF GASTRIC DILATION-VOLVULUS

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If you would like a copy of this surgical procedure on DVD go to www.videovet.org.

Key Points

• Survival is generally determined by early and appropriate presurgical management

• Patients referred for surgery should be decompressed prior to referral with continued decompression provided during transport

• Incisional gastropexy results in a fast, easy, permanent adhesion

• Ventricular tachycardia is a common postoperative complication

• Gastric necrosis signals an unfavourable prognosis

Introduction: Patients with GDV are considered critical care cases; every minute of presurgical treatment is vital to a successful outcome. Survival is generally determined by early and appropriate presurgical management; not surgery. Efficient presurgical treatment usually involves a minimum of two people. Gastric decompression and shock therapy should be done simultaneously. If this is not possible; decompression should be performed first. It is stated that gastric decompression is the single most important factor in reversing cardiovascular deficits in patients with GDV.

Decompression: Generally, orogastric intubation can successfully be performed in 80 - 90% of GDV patients. Decompression via flank needle puncture should be attempted in cases difficult to intubate or severely depressed metabolically deranged patients.

Technique: The stomach tube is measured to the last rib and marked with a piece of tape. A stiff foal or mare stomach tube with a smooth beveled tip works best (having several diameter and stiffness tubes is ideal). Apply adequate lubrication to the tube. Place a functional mouth speculum; generally a roll of 2" tape secured in the mouth with tape encircling the muzzle. As the stomach tube is passed, you will generally meet resistance at the esophageal-stomach junction. Pass the tube firmly in a twisting manner to pass the lower esophageal sphincter.

If unsuccessful, place the patient in various positions and attempt to pass the tube (i.e., elevate animal at 45 degree angle with rear feet on floor and forefeet on table, right lateral recumbancy, and left lateral recumbancy). This movement may encourage the stomach to rotate enough to allow tube passage. Be careful not to position the patient in dorsal recumbancy as this will increase abdominal visceral pressure on the caudal vena cava and may exacerbate signs of shock.

If still unsuccessful, try different diameter tubes; try a smaller diameter, more flexible tube and proceed as described above.

If still unsuccessful, attempt to remove some of the air in the stomach by placing an l8 gauge needle at the point of distention in the right flank region. Ping the area to make sure the spleen is not under the proposed trocarization site. After trocar decompression, attempt to pass the stomach tube as described above.

If still unsuccessful, sedate the dog with a narcotic (e.g., Oxymorphone) and try to pass the tube again. Mild sedation is recommended if the patient strongly resists physical restraint.

Success in passing a stomach tube depends on the skill of the operator and available assistants.

If you are successful at passing a stomach tube, but plan to refer the patient to a referral surgical center for gastropexy, transport the patient with the tube remaining in the stomach (i.e., taped to the mouth) or bring the tube out through a pharyngostomy incision or place a nasogastric tube.

If a stomach tube was successfully passed, stomach contents should be evaluated for color and presence or absence of necrotic looking gastric mucosa. This may give an impression of gastric viability.

Fluids: Shock dosage of polyionic isotonic fluid is carefully administered to expand the vascular compartment. Patients are frequently monitored during fluid administration to help determine ultimate fluid rate and amount. One or two indwelling cephalic catheters are placed.

Referral: If you are successful at passing a stomach tube, but plan to refer the patient to a referral surgical center for gastropexy, transport the patient with the tube remaining in the stomach (i.e., taped to the mouth) or bring the tube out through a pharyngostomy as described below.

Pharyangostomy placement:

a. Orally palpate the fossa lateral to the hyoid apparatus until a lateral bulge is seen

b. Make a small skin incision over the bulge and press a curved forceps (substitute for finger) through the soft tissues and skin incision.

c. Pull the stomach tube through the incision with curved forceps; then pass the tube over the arytenoid cartilages, down the esophagus, and into the stomach (measure to

the 13th rib).

Disadvantages include: heavy sedation or general anesthesia is necessary for placement of tube.

Rarely a temporary gastrostomy may need to be performed. The patient is placed in left lateral recumbancy with the right flank area clipped and surgically prepared. Heavy sedation and local infiltration of lidocaine or light general anesthesia is performed. A 4 - 5 cm incision is made in the skin over the point of greatest gastric distention (generally 1 - 2 cm caudal to the 13th rib and 2 - 3 cm distal to the transverse processes of the lumbar vertebrae). A grid technique is used to gain entrance into the peritoneal cavity. Due to severe gastric distention the stomach wall is pressed against the abdominal wall and easily identified through the flank incision. The stomach wall is sutured to the skin using a simple continuous pattern with 3-0 Maxon. This is done prior to incising into the stomach lumen. A #11 BP scalpel blade is used to puncture into the lumen of the stomach. Gas and stomach contents are expelled under pressure so stand back! The gastric mucosa is evaluated for viability. Disadvantages of gastrostomy include: the stomach is sutured in its rotated position and more time is required when definitive surgical treatment is performed due to the necessity of closing the gastrostomy.

Successful stomach tube placement: Once the stomach tube has been passed into the stomach or gastrostomy performed, the stomach is lavaged with warm water. If a stomach tube was successfully passed, the stomach contents should be evaluated for color and presence or absence of necrotic gastric mucosa. This may give an impression of gastric viability.

Surgical Treatment;: Surgical procedures utilized in the treatment of gastric dilatation-volvulus can be divided into two categories; 1) immediate decompression and 2) therapeutic gastropexy. Immediate decompression is performed with a successfully passed stomach tube secured to the patient or temporary gastrostomy as described above. Therapeutic or prophylactic gastropexy techniques are described below.

Gastric repositioning: Anatomic repositioning of the stomach is necessary to perform prior to permanent gastropexy. Repositioning occasionally occurs spontaneously at the time of gastric decompression. Knowledge of normal anatomy is necessary to understand how repositioning is performed.

A specific 'Surgical Plan' should be in mind before entering the operating room theatre. This will improve the efficiency of surgery and thus decrease overall surgery time. The 'authors' surgical plan is as follows:

Stand on the right side of the patient.

Provide generous abdominal exposure via xyphoid to pubis midline laparotomy.

Remove of all of the falciform ligament to the level of the xyphoid.

Place a 10'' Balfour self retaining abdominal retractor with full retraction.

Confirm that the omentum is draped over the exposed surface of the stomach (pathagnomonic for GDV)

Exteriorize the spleen from the abdominal cavity. Evaluate color, texture, blood flow (splenomegaly is always present and is NOT an indication for splenectomy)

Splenectomy is rarely performed but may be necessary if splenic vessels are infarcted.

If the stomach is full of air or fluid it should be emptied, if possible, prior to attempting derotation. If the stomach is full of food and several attempts to derotate (see author's technique below) are unsuccessful, perform a gastrotomy and manually remove the food from the stomach lumen. Suture the gastrotomy and attempt derotation again.

Attempt derotation by:

Standing on the patients' right side, first reach your right hand across the abdomen and place it between the left body wall and dilated stomach.

Slide your right hand along the sublumbar body wall and grasp the deep (dorsal) aspect of the stomach.

Next, place the open palm of your left hand on the exposed surface of the right side of the dilated stomach.

Using both hands simultaneously, pull the deep part of the stomach with your right hand to begin derotation whilst you push the right surface of the stomach down toward the patients sublumbar body wall with your left hand. This maneuver will be successful in the majority of cases.

See this maneuver performed on the Emergency Surgery I, Gastrointestinal Surgery I, and Soft Tissue Surgery II DVD's available at www.videovet.org.

Once the stomach is derotated, evaluate the stomach for evidence of viability abnormalities (particularly the greater curvature and fundus) and for evidence of gastric motility.

Commence your gastropexy procedure.

Incisional gastropexy: This technique is based on a 3-4cm long seromuscular antral incision sutured to a similar length incision in the transversus abdominus muscle.

With the Balfour retractors still in place visually locate the ideal position for the antral wall incision. It should be located equidistant between the pylorus and gastric incisure and equidistant between the greater curvature and lesser curvature of the stomach. A 3-4 cm sero-muscular incision is made in this antral location. An easy way to safely make the sero-muscular incision is to grasp the full thickness antral wall with your thumb and finger

at the site of the proposed incision, gently retract the wall of the stomach until you can feel the mucosa and submucosa 'slip' out of your thumb and finger. The tissue remaining between your thumb and finger is the sero-muscular layer of the antral wall. With a straight or curved pair of Metzenbaum scissors cut all the tissue remaining in your thumb and finger resulting in a perfect depth of the sero-muscular incision.

Once the antral incision is completed remove the Balfour retractors. When selecting the location on the transversus abdominus m for the gastropexy, it is important to first visualize the location of diaphragmatic muscle fibers as they radiate into the abdominal cavity and attach near the costal arch. It is important that the gastropexy site be at least 2cm caudal to the diaphragm muscle insertion. After identifying the attachment of the diaphragm, the bleeding surface of the antral incision is brought to the right body wall. With the stomach in a normal position, the bleeding antral surface is touched to the peritoneal wall approximately 3-4 cm deep to the abdominal wall incision and 2cm caudal to the insertion of the diaphragm. A blood mark is created on the peritoneum at this proposed location. This will be the site for the permanent gastropexy. The peritoneum and transverses abdominus muscle are then incised creating a mirror image defect of the antral incision. The incisional defect in the stomach is then sutured to the incisional defect in the abdominal wall. The defects are sutured in two layers using a simple continuous pattern with 2-0 or 3-0 monofilament or multifilament synthetic absorbable suture.

Belt Loop Gastropexy: This technique is based on the construction of a sero-muscular antral flap attached around a segment of transversus abdominus muscle. A horseshoe shaped incision is made in the serosal layer of the antral portion of the stomach with its base at the greater curvature. The sero-muscular portion of the stomach is identified by grasping full thickness antral wall between the thumb and index finger and "slipping" the mucosal and submucosal layers away so only the sero-muscular portion of the wall remains between thumb and finger. The sero-muscular layer is incised with scissors and the horseshoe shaped sero-muscular antral flap is dissected and elevated of the submucosal layer. The stomach is replaced in the abdominal cavity in normal position and the sero-muscular flap lined up with the transversus abdominus muscle. Once this optimal location is discovered, two longitudinal incisions (along the fibers of the transversus m.) are made in the transversus abdominus m. The segment of muscle between the incisions is undermined. The sero-muscular flap from the stomach (i.e., belt) is passed through the transversus abdominus m. (i.e., loop) and sutured to itself to complete the "Belt-Loop" gastropexy. 2-0 or 3-0 monofilament absorbable synthetic suture in a simple interrupted or continuous pattern is used to secure the flap in place. Advantages of belt loop gastropexy include: it is relatively easy to perform alone and in the middle of the night, it can be performed quickly, and it is an effective means of permanent gastropexy.

Postoperative management:

In most cases 3 to 4 days of intensive monitoring is necessary for the successful management of GDV patients. Postoperative considerations are listed below:

a. Shock is a postoperative possibility and the patient should be monitored and treated accordingly.

b. Patients are generally held off food and water for 24 hours fol¬lowing surgery. During this time maintenance fluids should be supplied using polyionic isotonic crystalloid fluid. Vomiting may occur following surgery; the NPO period should be extended accordingly. Gastritis and gastric motility disorder may be seen in post op GDV patients.

c. After 24 hours of no vomiting, oral alimentation should begin gradually with

a sequence of ice cubes, water, and finally canned dog food. This should occur over a 2-3 day period.

d. Antibiotics should be continued for 7 - 10 days.

e. Routine surgical complications such as infection, dehiscence, seroma, etc. should be watched for and treated accordingly.

f. EKG monitoring: the most common severe postoperative complication is cardiac arrhythmia. Ap¬proximately 75% of GDV patients will develop arrhythmia's in the immediate postoperative period. Arrhythmia's can be present at the initial time of presentation but most often occur within 24 - 72 hours after surgery. Ventricular premature contractions, progressing to ventricular tachycardia is most common. Etiology is unknown but shock, hypoxia, acid base alterations, endotoxins, myocardial depressant factor (MDF), reperfusion injury, release of free radicals, and hypokalemia have been identified. Occurrence of a total body potassium deficit has been proposed. Etiology of the hypokalemia includes anorexia, vomiting, tremendous outpouring of potassium rich fluids into a dilated stomach, and use of potas-sium poor fluids in treatment of shock. For this reason, ad¬ding 20-30 mEq of potassium chloride per liter of maintenance fluids during and after surgery are recommended.

g. Gastric motility: occasionally GDV patients will develop postoperatove gastric motility abnormalities. Patients with gastric hypomotility or gastric stasis should be treated with a motility modifier (i.e., metaclopramide, erythromycin, etc).

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STATE-OF-THE-ART LECTURE PRACTICAL USE OF REPRODUCTIVE HORMONES IN DOGS AND CATS

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Introduction

Reproductive hormones are readily available to veterinarians working with dogs and cats, both as small animal as well as large animal drugs. The increased importance of companion animals as family members has caused a differentiation of reproductive presenting complaints, from mostly castration a few decades ago to preventing and interrupting pregnancies, inducing heat, blocking the oestrus cycle, causing reversible sterility, treating uterine diseases or neutering-induced conditions or mammary hypertrophy and the like. These presenting complaints require in general a high degree of confidence with the clinical use of reproductive hormones, some of which have been improperly used for decades such as progestogens[1, 2]. This paper will review the most common clinical indications and applications in small animals of prostaglandins, antiprolactinics, progestogens and estrogens.

PROSTAGLANDINS

Several prostaglandin F2alpha (PGF) compounds have been available for over 30 years as veterinary compounds for use in food animals and horses. Their luteolytic and uterotonic actions make them unique and very useful in small animals for several indications related to pregnancy termination and treatment of uterine conditions. Recently, the use of prostaglandin E1 (PGE1) products such as misoprostol has become popular for their uterotonic properties.

Early pregnancy termination in bitches only (not in queens)

The abortifacient efficacy of PGF involves induction of luteolysis, stimulation of uterine contraction and cervical dilation. Initially it was thought that these actions could be achieved using dosages of 250 mcg/kg[3], but it was later demonstrated that lower doses could be just as effective[4]. In dogs, the progesterone (P4) necessary to support pregnancy is entirely secreted from the corpora lutea throughout gestation. PGF will induce luteolysis and decrease P4 concentrations to nearly non-detectable levels very easily after day 25 or 30 but also as early as day 10 following onset of diestrus[5]. The later in the cycle PGF is administered the easier and more rapid the induction of luteolysis. Use of PGF requires subcutaneous administration 2 or 3 times a day, for 6-9 days or longer, until pregnancy termination can be confirmed by ultrasound[6-9]. The dosage varies depending on the type of PGF: natural PGF should be administered using a maximum dosage of 50-80 mcg/kg twice or 3 times daily (TID with 50 mcg/kg, BID with 80 mcg/kg), starting gradually with 1/3 to ½ the dose for the first day (or the first 2 administrations)[6,10]. Cloprostenol should be used at a dose of 1-2.5 mcg/kg once daily and alphaprostol should be used at the dose of 20 mcg/kg twice daily. Side effects (which include emesis, salivation, defecation, urination and slight tachypnea) are dose dependent (i.e. displayed in 80% of bitches using doses of 250 mcg/kg natural PGF and only in 20% of bitches using doses of 50 mcg/kg natural PGF) and self-limiting, decreasing in intensity with repeated dosing. PGF do not appear to be working during the first half of diestrus in queens.

Late pregnancy termination in bitches and queens

Canine late pregnancy termination is generally adopted as a treatment when either a mismating was not observed, the female was not in the ovulatory phase or fertility of the male is unknown. Dosage of PGF compounds is the same as for early abortion, the only difference being that treatment must be continued until verification of efficacy by ultrasound as partial abortion of litters can occur if treatment is discontinued prematurely. With most dosages, 9 or more days may be required to complete fetal expulsion, although 5 to 7 days is usually sufficient[8]. Cloprostenol at the dose of 2.5 mcg/kg subcutaneously, administered three times, at 48 h intervals, starting at day 30 of pregnancy shows a high efficacy in termination of pregnancy. Cloprostenol at even lower doses (1.0 mcg/kg) has been used in combination with a dopamine agonist treatment to terminate pregnancy shortly after implantation (which in bitches and queens occurs around 15-18 days after ovulation) starting around day 23 from ovulation[11]. Mismated queens can be treated with natural PGF at a dose of 2 mg/cat IM once a day, beginning at day 33 of pregnancy, as this will induce luteolysis and terminate pregnancy by expulsion of fetuses in pregnant cats. Side effects are milder than in dogs and included prostration, vomiting and diarrhea. Cloprostenol has been used in queens with success (and with fewer side effects than in bitches) at the dose of 5 mcg/kg in combination with cabergoline administered once daily from mid pregnancy on[12].

Uterine evacuation (including treatment of open cervix pyometra) in bitches and queens

The mioconctracting action of PGF compounds is well known, and can become useful when dealing with cases of open-cervix pyometra or late abortions with incomplete fetal expulsion[13]. Dosage are the same as listed above. We frequently use PGF compounds combined with aglepristone (**see paper on clinical use of aglepristone**) when late pregnant bitches are presented

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for induction of abortion, as such combined treatments are shorter and more efficacious. Care should be taken to make sure that the cervix is open, as causing uterine contractions on a closed cervix may cause uterine rupture or force uterine content up into the oviducts. Therefore, aglepristone is administered first and then PGF are used once cervical opening has occurred.

Other clinical applications

Prostaglandin F2a (PGF) products have been used also to **induce parturition** (albeit with a continuous infusion pump) in 57-day pregnant bitches with whelping occurring normally within 2-3 days14. A **shortening of the interstrous interval** may be obtained using PGF alone or in combination with antiprolactinics. The use of a 6-10 day course of PGF starting after day 10 of cytological diestrus will achieve a complete luteolysis which will shorten diestrus and very often also anestrus. We have observed bitches in which a complete and permanent luteolysis was obtained with PGF early in their luteal phase coming back in heat after 70-90 days following the onset of previous proestrus5. If shortening of the duration of diestrus is obtained with PGF and is then followed by an antiprolactinic treatment (which may shorten anestrus – **see over**), the interestrous interval will be substantially shortened. In male dogs, an increase in semen volume and occasionally also quality (often motility) may be obtained by administration of PGF at the dose of 100 mcg/kg 15 minutes prior to semen collection; this has resulted in an increase of 270% of total sperm numbers when compared to saline treated controls, with no deleterious effect on refrigeration and freezing (Hess, 2006). PGF may also be used to obtain an ejaculate from a reluctant or inexperienced dog15.

Prostaglandin E1 - A synthetic analogue of PGE1, misoprostol, has a strong uterotonic action and may be used to help evacuating uterine content in bitches and queens with pyometra. Misoprostol is marketed as a human compound under different trade names and is available in 200 mcg tablets. In the bitch it is administered at 10 mcg/kg BID orally (1/2 tablets/10 kg16. It is very well tolerated and may be administered at home by clients. It has no luteolytic properties and little if any side effects mostly on the first day of treatment (vomiting = 25% of cases; diarrhoea = 30% of cases) 17. It can be used in both bitches and queens as an adjunct to a PGF2a or aglepristone treatment to improve uterine contractility, or to continue causing uterine contractions once luteolysis has been accomplished thus sparing the female the PGF2a related side effects. PGE1 pills may also be dissolved in saline solution and administered intravaginally 18.

ANTIPROLACTINICS

Prolactin secretion by the lactotroph cells of the anterior pituitary gland is regulated by multiple neuro-transmitters and hormones, with the major control mechanism being the activation of prolactin-inhibiting dopaminergic neurons in the hypothalamus. Prolactin is a major luteotrophic hormone and appears to be an absolute requirement for canine and feline progesterone secretion from day 30 after ovulation onwards. Dopamine agonists like bromocriptine or cabergoline are ergot alkaloids, with strong dopamine D2-receptor agonist activity, and thus can reduce prolactin secretion thereby suppressing progesterone levels. The serotonin antagonist metergoline indirectly stimulates endogenous dopamine secretion and thus can inhibit prolactin secretion as well. Cabergoline has a slow clearance, which allows for a single oral daily administration. Furthermore, its action when used at the currently accepted dosage of 5 mcg/kg is longer than 48 hours due to its particularly long (minimum 48 hours) half-life at the hypophyseal level. Bromocriptine mesylate inhibits PRL secretion during relatively short periods of time (half-life: ± 4-6 hours) and in a dose-dependent mode. In order to effectively inhibit PRL tone in a continuous fashion for therapeutic purposes, bromocryptine should be administered at least twice a day orally at doses 10-50 mcg/kg. Its lack of specificity leads to side effects on the cardiorespiratory system, causing hypotension due to vasodilatation (adrenergic type effect), or emesis due to stimulation of the Chemioreceptive Trigger Zone (CTZ). Although its effectiveness has never been questioned, bromocryptine is not approved in most countries as an anti-PRL in small animals and its use in animals may be prosecuted in countries where veterinary antiprolactinic drugs are available. Metergoline is essentially a serotoninergic antagonist with dopaminergic agonist properties when used orally at doses of 0.1-0.2 mg/kg BID. Its shorter half-life requires at least administrations twice a day. Its antiserotoninergic properties may occasionally induce central effects such as depression, nervousness, increased excitability, changes in appetite (anorexia or bulimia), psychotic effects (escaping from home, rarely aggressiveness), rarely vomition. Antiprolactinic drugs can be used in the bitch and the queen with three indications: pseudopregnancy, induction of abortion and induction of estrus 19.

Pseudopregnancy

Antiprolactinics are currently considered the treatment of choice for pseudopregnancy. The anti-lactogenic action of both metergoline and cabergoline is well known19. Their administration for 4-5 days at pharmacological doses is effective in treating pseudopregnancy signs and reducing milk production. Occasional failures can be dealt with by repeating the treatment protocol and extending it to 8 to 10 days, and also by administering at the same time metergoline (at the usual antigalactogenic dosage of 200 mcg/kg BID). Our current approach is to prescribe a 6-8 day treatment course as we feel that this decreases incidence of recurrence. Because

of the potential presence of a daily pattern of prolactin secretion, antiprolactinic treatments should best be administered in the morning to achieve a more disruptive effect on prolactin secretion20.

Induction of abortion

The abortion induction properties of antiprolactinic drugs have been well studied for dopamine agonists (cabergoline and bromocriptine), while not as much is known for metergoline. Cabergoline and bromocriptine are effective in terminating pregnancy in dogs when administered at mid-gestation (as prolactin secretion starts around day 25) or later 21,22. When administered after day 40 at oral doses of 5 mcg/kg for 5 days cabergoline is effective in causing abortion in all treated bitches21. If cabergoline administration is started earlier in pregnancy, at day 25, treatments that are effective later in pregnancy fail in most bitches and the pregnancy continues until terminated by retreatment at day 40. Combination treatment of cabergoline and prostaglandins have been used for induction of late abortion both in bitches and queen11, 12. Also, in our experience alternating PGF and antiprolactinics on consecutive days works well and allows to reduce the dosage of PGF.

Estrus induction

The estrus inducing action of antiprolactinic drugs was initially thought to be due to the lowering of prolactin concentrations, but studies done at Utrecht have demonstrated that shortening of anestrus occurs irrespective of prolactin concentrations23. All the 3 antiprolactinic products (cabergoline bromocriptine and metergoline) have been used for oestrus induction in the bitch. Cabergoline and bromocriptine have consistently given positive results, while metergoline's results have been more variable depending on dosage. Using low metergoline doses (0.1 mg/kg BID) from 100 days after ovulation until the following proestrus, the interoestrous interval will be significantly shortened. We have used bromocriptine at the dose of 10-25 mcg/kg in 5 bitches with prolonged anoestrus: 4/5 came in oestrus within 13-28 days, and all 4 conceived and whelped (unpublished data). In a study on the use of cabergoline (5 mcg/kg, once daily for up to 28 days) in 9 bitches (7 Rough Collies, 1 Shetland sheepdog and 1 English Setter) starting in mid anestrus, fertile oestrus was induced in 10/11 cycles in 24+11 days with a reduction of the interoestrous interval of 1.8+0.2 months24. In our experience, the clinical use of antiprolactinics to induce oestrus has proven to be effective in about 70-80% of cases. Occasionally a bitch may take more than 40-50 days of treatment which may cause the owner to get frustrated and discontinue the treatment.

PROGESTOGENS

Short- and long-acting synthetic progesterone compounds or progestogens have long been used in bitches and queens to control reproduction. The only short-acting product currently marketed in most countries of the World is megestrol acetate (MA), available as an oral formulation to determine oestrus suppression (short-term control) in bitches and short- as well as long-term suppression in queens. Long acting progestogens available as veterinary drugs such as medroxyprogesterone acetate (MPA) and proligestone (PROL) are used for oestrus postponement (prolonged control). The mechanism of action by which cyclicity is blocked involves disruption of pituitary-ovarian communication resulting in lowered LH and FSH release and reduced concentrations of estrogen receptors in target tissues. Duration of effect depends on degree of pituitary responsiveness which increases progressively during anestrus: therefore, early-mid anestrus treatments will be longer lasting than late anestrus treatments. Progestogens act on all target organs of P4 such as uterus (increased endometrial growth and secretion), cervix (closure), motility of reproductive tract (decreased gamete transport), mammary glands (stimulation of growth). Other hormones affected by a (short- or long-acting) progestogen treatments include, estrogens, inhibin and activin which may be decreased, growth hormone (GH) whose secretion by the mammary gland is increased, and prolactin which is inhibited during treatment and then shows a significant surge once treatment is discontinued. Insulin resistance is mediated by increased GH mammary secretion, and is particularly evident during treatment with MA. Endocrine side effects are transient and irrelevant when a young and healthy female receives a progestogen treatment with the right dosage and for an appropriate length of time. Unfortunately, overdosing has occurred many times in the second half of last century both in bitches and queens1,2, and many of these cases have been reported in the literature and cited over and again causing a widespread fear about the use of these compounds. On the contrary, a large amount of experimental data is available on the use of progestogens in bitches and queens which dates back to when these animal species had to be used for approval of marketing of P4 compounds as human drugs in the '60s and '70s of last century. When reading the literature carefully it becomes evident that all the case reports of pyometras, mammary nodules and hypertrophy, diabetes, endocrine imbalances and many others were all due to a) the use of very high dosing, b) too long treatments, or c) choice of the wrong candidate. For instance, treatment during diestrus (when endogenous P4 secretion is active) should be avoided as it could easily cause overdosing even if the correct dosage is used; serum P4 assay may be useful to identify risk patients, and should be assayed also in queens due to a relevant incidence of spontaneous ovulations in this species. Diabetic patients or females with a history of irregular cycling,

vulvar discharge, mammary nodules or liver/kidney insufficiency should not be treated. Pregnant females or pseudopregnant bitches should not be treated. Also, treatment during oestrus to suppress an unwanted heat should be done with caution and only with short-acting drugs for short periods of time, as progestongens side effects are likely to be amplified by previous estrogen exposure. Table n° 1 shows the suggested dosages of the most commonly used progestogen-based compounds in bitches and queens. The lowest effective dose should be used even if this may cause an earlier return to oestrus. Treatment durations of more than one year are probably adequate for young, healthy females, while shorter treatments should be considered for middle age females who may have subclinical uterine or mammary conditions. The use of progestogens in older females should be discouraged.

Suggested Dosage	Bitch	Queen
Megestrol Acetate	Oestrus suppression <2.0 mg/kg for 8 days in proestrus Oestrus postponement 0.05 to 0.01 mg/kg daily per os up to 1 yr	Oestrus postponement 0.02 to 0.09 mg/kg/day per os up to 1 year
Medroxy-progesterone Acetate	Oestrus postponement 1.5-2.0 mg/kg SC or IM every 13 weeks 2.5-3.0 mg/kg SC or IM every 4-5 mos 0.05 mg/kg/day per os up to 1 year	Oestrus postponement 0.01-0.05 mg/kg/day for up 1 year
Proligestone	Oestrus postponement 10-33 mg/kg SC every 3,7,5,5 months	Oestrus postponement 25-30 mg/kg SC every 5 months

Table n° 1 – Suggested dosages of the 3 most commonly used progestogen compounds in bitches for the control of reproduction. The dosage for proligestone in bitches should be intended 10 mg/kg in large size dogs and up to 33 mg/kg in medium to small size dogs

ESTROGENS

Estrogens have always been considered as potentially dangerous drugs for small animals because of their role in inducing mammary neoplasia, endometrial hyperplasia, pyometra and bone marrow aplasia in bitches [25]. However, such dangerous side effects are a feature of long-acting compounds, while short-acting estrogens such as estriol are not associated with development of full (late) estrogenic effects. Estrogens have historically been used for contraception and for the treatment of urinary incontinence.

Unwanted pregnancy

Several estrogenic compounds have been used for this purpose, but for most of them the risk of side effects has discouraged their clinical application[26]. As a consequence, estrogens are no longer considered a safe choice for canine pregnancy termination. Only estradiol benzoate, when given at low doses has proven to be fairly efficacious and relatively safe. A compound with estradiol benzoate is marketed for veterinary use in mismated bitches in some European countries, which is to be administered at the dose of 10 mcg/kg SC on day

3, 5 and 7 post breeding[27]. No short-term side effects have been reported following this protocol. However, in a retrospective study done in the UK the incidence of pyometra in the 4 months after the administration of low doses of estradiol benzoate was 8.7%, whereas the incidence of that condition in a practice situation was estimated to be < 2.0%[28].

Urinary incontinence

Long-acting synthetic compounds such as diethylstilbestrol, estradiol, estrone and other esther compounds are characterized by a dangerous action on bone marrow and uterus because of their prolonged nuclear occupance time in estrogen receptors of target tissues[29]. The following is a list of estrogens which have been used to treat canine urinary incontinence.

17-b estradiol	0.01 mg/kg MID sc/im for 3 days
Estradiol benzoate	0.01-1.0 mg MID per os
Estradiol valerate	1.0 mg/10 kg bw
Diethylstilbestrol	0.06 mg MID, tapering out to 0.01 mg
Estriol	**0.5-2.0 mg**
Conjugated estrogens	0.02 mg/kg

Of all the estrogenic compounds listed above, estriol is the only one considered as a safe drug due to its short-action which is characterized by short nuclear occupance time and minimal metabolism following absorption. Estriol does not bind to sex-hormone binding globulin, which helps in preventing development of full (late) estrogenic effects such as endometrial hyperplasia, pyometra and bone marrow suppression. Estriol is available for human use as hormone replacement therapy for women in menopause, and is marketed in several European countries as a veterinary preparation to treat canine urinary incontinence. The current formulation is in 1.0 mg pills to be given once daily for 1 week, and then either gradually increase (in case of little or no response) or decrease (in case of a very good response), with the goal being to find the lowest possible dosage Its efficacy after 42 days of 2.0 mg/day treatment was 85% in a multicentric clinical trial recently performed on 129 bitches in 4 european countries[30]. Hematological abnormalities are not observed when using dosages of 0.5-1.0 mg/day even for years, nor have they been reported in mid-term (3 months) chronic toxicity studies using 2.0, 6.0 and 10 mg doses[31]. Vulvar swelling and male attractiveness was occasionally observed in bitches treated with estriol doses of >1.5 mg/day. Some signs of estrus can be observed in bitches administered higher dosages [31]

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43RD WORLD SMALL ANIMAL VETERINARY ASSOCIATION CONGRESS AND 9TH FASAVA CONGRESS

WSV18-0285

INTEGRATIVE MEDICINE (LECTURES GIVEN IN MANDARIN CHINESE)

AN INTEGRATIVE APPROACH TO OSTEOARTHRITIS: THINKING BEYOND NSAIDS

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NURSES (HILLS)

INSTRUMENT CARE TIPS AND TECHNIQUES

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INSTRUMENT CARE TIPS AND TECHNIQUES

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Handling and Care of Instruments

Modern surgical instruments are made **of** stainless steel, an alloy of iron, chromium and carbon. The inclusion of chromium increases the resistance to corrosion. With proper care and attention, surgical instruments will last for years.

In larger veterinary hospitals it may be necessary to have a number of general surgical packs, with additional specialized instruments packed separately. A card index system, outlining the equipment needed for each procedure facilitates efficient preparation prior to surgery. Coloured tape may be used for identification of instruments from within the same set to allow for easy repackaging however, this must be used with caution as it provides a useful "hiding place" for bacteria.

Care

-Instruments should only be used for the purpose for which they were designed. Allis tissue forceps should not be used as bone holders and Metzenbaum scissors should not be used for cutting sutures!

-Instruments should always be placed on surfaces, not dropped or thrown.

-Heavy items should not be placed on top of, or adjacent to delicate equipment.

-Instruments with cutting blades should be protected to avoid dulling of the edges.

-Protective sheaths should be left in place until the instrument is ready for use.

-During surgery, the scrubbed nurse (if utilising this role) should ensure that the instruments are kept free of blood and debris by wiping with a sterile moistened swab after each use.

Checks

Instruments should be checked after each procedure.

INSTRUMENT	CHECKS
Scissors	Check tungsten carbide inlays for burrs
	Check both side for cracks
	Check correct fit of the screw linkPerform cutting test
Forceps/ clamps	Check the jaws for damages, scratches and deformation
	Check the jaws teeth, serration and tines
	Cracks in the link
	Check the teeth lock fully engage
	Teeth have same measurement and fit nicelyCheck the clamp cannot be opened
Needle holders	Check and test the lock
	Check serration for damagesCheck the rounding of inner jaw tips visually and physically with finger tips
Dissecting forceps	Check jaws for damages, deep scratches and deformation Check spring for cracks Check spring for tension Spring must not be bent

Other recommend checks;

-Ratchets should close easily and hold firmly.

-Box locks that are too loose will cause misalignment and will not hold tissue securely. When not in use, the box locks should be left secured on the first ratchet.

-The shaft of instruments, in most cases, should be straight to ensure correct alignment.

-Cutting instruments (scissors, osteotomes, chisels, periosteal elevators and rongeurs) should be checked for sharpness and chips in the metal.

-Protective sheaths should be applied for storage.

Needle holders become damaged when too large a needle has been used for the given size and type of needle holders. An appropriately sized needle will be gripped firmly with the jaws locked on the second ratchet. Some needle holders have tungsten carbide inserts, which increase strength and reduce wear on the gripping surface. These instruments are usually more expensive but they will last longer as the inserts can be replaced therefore prolonging the life of the whole instrument. Instruments with tungsten carbide inserts are identified by their gold coloured handles.

Titanium alloy is used for microsurgical and ophthalmological instruments. This alloy has a bluish colour that reduces glare under the operating lights. Such instruments are lighter and stronger than surgical steel instruments. Because of the design and physical properties of the instruments, the surgeon is able to control their movements much more easily. Extra care should be taken when handling these instruments because they are not only delicate, but are also expensive to replace. They can be placed in special trays for cleaning and storage.

Manual Cleaning

Before cleaning the surgical pack the priority is the safe removal and disposal of all sharps! This is important to avoid sharp and needle stick injuries to the theatre nurse when cleaning and handling the instruments. Common sharps to look out for in surgical kits include scalpel blades (there could be more than one used) and various needles (especially swaged onto lengths of suture material).

Always clean instruments straight after use. If thorough cleaning is not possible then at least rinse the instruments in cold water to remove gross dirt and blood. Hot water should not be used as it causes coagulation of proteins (e.g. blood). Instruments should not be soaked for prolonged periods as this is detrimental to the chromium oxide layer on the instrument surface, which may lead to staining and corrosion. If soaking is necessary a chemical cleaning solution specifically designed for instruments may be used.Instruments should be cleaned using a detergent specifically designed for instrument cleaning as ordinary soaps and detergents can damage the surface of the instrument. Enzymatic cleaners are available which break down the dirt and grease. Instruments that have movable parts should be disassembled prior to cleaning (e.g. depth gauges and drill guides etc.) All box locks and ratchets should be opened to ensure thorough cleaning.

Serrations and box locks should be cleaned with a soft bristle brush. Abrasive materials should never be used as they scratch the surface of the instrument, which may collect organic debris in later use and cause corrosion. A final water rinse will remove any traces of detergent prior to autoclaving

Ultrasonic Cleaning

Ultrasonic cleaners are effective at removing debris from inaccessible areas such as box locks, serrations and deep grooves. These cleaners act by a process of cavitation. Ultrasonic energy produces high frequency sound waves that generate tiny bubbles in the solution in the cleaner. The bubbles expand until they become unstable and collapse. This collapse generates minute vacuum areas that dislodge and dissolve debris.

The bath is filled to a level one inch above the tray. Only instrument cleaner recommended by the manufacturers should be used. The instruments are placed into a wire mesh tray (heavier, bulkier instruments at the bottom) and lowered into the bath. The tray should only be half full to avoid overloading and damage to the instruments. Instruments are thoroughly rinsed after ultrasonic cleaning to remove any surface debris and detergent residue.

Instrument Lubrication

Following cleaning and rinsing, all instruments with moving parts should be lubricated. Instrument manufacturers recommend the use of water-soluble lubricants such as instrument milk, an oil-in-water emulsion that has anti-microbial properties.

As a general rule the instruments should be submerged in the lubricant bath for thirty seconds and then left to drain without rinsing or drying. However, if the instruments are to be sterilised in a hot air oven then all traces of the lubricant should be removed as the intense heat causes the milk to form a "gum" which is difficult to remove.

Machine or mineral oils are not suitable as lubricants as they leave a sheen on the instrument surface and will inhibit steam penetration during the sterilisation process. Some instruments, such as orthopaedic drills, have their own silicone gel. This should always be used in accordance with manufacturer instructions.

Once the cleaning process has taken place, surgical instruments must be left to dry. Instruments must be laid out flat with any ratchets and hinges open to ensure adequate drying and reduce the risk of rust damage. Instruments are always air dried in a clean, low traffic area to avoid contamination. Alternatively a drying cabinet may be used. Use of a cloth or towel to dry surgical instruments will lead to accumulation of debris, which could harbour bacteria.

Sterilisation

Surgical instruments and equipment are sterilised before each and every subsequent use. The sterilisation process takes place after the instruments have been cleaned and are dry.

Heat Sterilisation

Autoclaves

This is the most widely used and efficient method of sterilisation and is also the most economical. Most items used within the veterinary practice can be autoclaved and include:

-Instruments

-Cloth drapes and gowns

-Swabs

-Most rubber articles – need to check with manufacturer/instructions

-Some plastic goods – need to check with manufacturer/instructions

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The Animal Industries Resource Centre Course materials – Certificate IV in Veterinary Nursing

SVA SOFT TISSUE SURGERY

THE 4-LIGATURE SPLENECTOMY

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INTRODUCTION

Splenectomy can be a life-saving procedure and is often necessary on an emergency basis. Unfortunately, most dogs that present with a spontaneous hemoabdomen associated with a splenic bleed have neoplasia as the underlying etiology; although benign lesions such as hematomas may also be seen. Stable dogs with non-ruptured splenic masses are also candidates for splenectomy. Spontaneous hemoabdomen is a challenging condition that requires rapid diagnosis with timely therapeutic intervention to maximize the chance of a successful outcome. Unfortunately, malignant neoplasia is the most common etiology and despite a successful short-term outcome, a guarded long-term prognosis is common. The peritoneal cavity can be considered a large potential space in which the majority of a dog's blood volume can reside. Consequently, with rupture of a highly vascular intra-abdominal organ, vascular collapse and end-organ ischemia can result rapidly. The major objectives of the veterinarian who is treating a patient with spontaneous hemoabdomen include; rapid and effective resuscitation, timed surgical intervention, rapid identification of the point of hemorrhage and efficient elimination of the source of hemorrage.

INDICATIONS

Splenectomy is indicated for removal of splenic neoplasm, rupture, torsion, infarct, abscess and hypersplenism.

PATIENT POSITIONING

The patient is placed in dorsal recumbency for routine celiotomy.

RECOMMENDED INSTRUMENTS

A Balfour self-retaining abdominal retractor is essential to maintain adequate exposure allowing complete exploration of the abdominal cavity as well as visualization of the splenic blood supply. When large amounts of blood or fluid are present in the abdominal cavity suction, using a Poole suction tube, is helpful. It is best to have a variety of sizes of hemostats available. The author recommends a minimum of 6 medium to large hemostatic forceps (Crile, Kelly or Carmalt) and 4 – 5 small hemostatic forceps (mosquito).

Ligation of individual blood vessels or clusters of vessels is performed using 3-0 or 4-0 synthetic absorbable

suture material. Common sutures include Biosyn, Monocryl, Dexon, Vicryl, Polysorb, PDS or Maxon. A secure friction knot such as a Strangle knot, Double Half Hitch or Modified Miller's knot is recommended for secure vascular ligations.

SURGICAL TECHNIQUE

A ventral midline incision from xyphoid to pubis is made to allow adequate exposure of all abdomen organs. The falciform ligament is removed and a large (10") Balfour self-retaining retractor is positioned (with the frame of the Balfour toward the cranial aspect of the incision) to provide exposure of the abdominal cavity.

The spleen is located in the cranial left quadrant of the abdominal cavity just caudal to the greater curvature and fundus of the stomach. The spleen is identified, and gently elevated through the abdominal incision. If the surgeon is dealing with a bleeding spleen (e.g., hemangiosarcoma) the exteriorized spleen is placed across the body wall to help place pressure on the splenic blood vessels. In addition, a dry laparotomy pad can be placed directly on the point of hemorrhage and gentle pressure applied. At this point a rapid and complete abdominal exploratory is performed to rule-out obvious metastasis.

Prior to splenectomy several structures should be identified. The greater curvature of the stomach, dorsal and ventral layers of the greater omentum, the gastrosplenic ligament and the left limb of the pancreas. Trace the splenic artery and vein as they course from the dorsal layer of the greater omentum into the gastrosplenic ligament. Identify the left gastroepiploic artery and vein, the many splenic arterial and venous branches into the hilus of the spleen, the short gastric vessels and the vessels continuing into the greater omentum.

The spleen receives its blood supply from 3 major sources. Three to four short gastric vessels supply the cranial aspect of the spleen. The central portion of the spleen is supplied by the major splenic artery and vein and the caudal pole of the spleen by 4-5 small omental tributaries.

The spleen can safely be removed using a technique requiring only 3 to 4 cluster ligations. Visualization of these vessels is accomplished by first elevating the spleen from the abdominal cavity. When attempting to exteriorize the spleen it is noted that its cranial pole is tethered to the greater curvature of the stomach by the 3 to 4 short gastric vessels. These vessels are identified and cluster ligated with two encircling ligatures. The vessels are transected between ligatures thus releasing the tethering effect. The spleen can now be further mobilized from the abdominal cavity allowing easy exposure of all remaining vessels.

Next the major splenic artery and vein is located and ligated prior to its bifurcation. Care should be taken to visualize the left limb of the pancreas and make certain it is a safe distance from the proposed ligation site. The splenic artery and vein are generally double ligated and depending upon size the artery can be transfixed. Finally the remaining vessels supplying the caudal pole of the spleen are cluster ligated using one or two ligatures.

During the procedure, several points should be remembered:

1) when ligating the splenic artery and vein, identify the location of the pancreas and do not occlude its blood supply

2) double ligate all major vessels

3) carefully inspect all ligated vessels for evidence of hemorrhage

CLOSURE

The Balfour retractor is removed and the abdominal incision is closed in a routine fashion.

POSTOPERATIVE CONSIDERATIONS

Postoperative care involves monitoring the patient for blood loss that may be encountered should a ligature slip from the ligated vessels.

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REPRODUCTION

BENIGN PROSTATIC HYPERPLASIA

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Normal prostatic development and function

The prostate gland is the major accessory sex gland in the male dog. It is located just caudal to the bladder in the area of the bladder neck and proximal urethra. Its purpose is to produce prostatic fluid as a transport and support media for sperm during ejaculation. Basal prostatic secretion is constantly entering the prostatic excretory duct and prostatic urethra. In the absence of micturition or ejaculation, urethral pressure moves prostatic fluid cranially into the bladder (a mechanism called prostatic fluid reflux). As the dog grows old the prostatic parenchyma is characterized by an increase in epithelial cell numbers (hyperplasia) as well as an increase in epithelial cell size (hypertrophy), but the increase in cell number is more marked[1]. This growth process begins as glandular hyperplasia in dogs as young as 2.5 years of age[2]. Intraparenchymal fluid cysts may develop in association with hyperplasia. Such cysts are variable in size and contour, contain a thin, clear to amber fluid and, if intraprenchymal, may communicate with the urethra thus leading to intermittent haemorrhagic or clear, light yellow urethral discharge.

Benign Prostatic Hyperplasia

Due to normal growth and glandular hyperplasia, the prostate of intact normal male dogs increases in weight, for the first 5 years, with a peak at 4 years of age[1]. As many as 16% of dogs have been reported to have histologic evidence of benign prostatic hyperplasia (BPH) by 2 years of age[2]. The incidence of BPH increases to over 80% with advanced age. Senile involution of the prostate occurs in animals aged 11 years or more[1]. Like in humans, the prostate of a mature dog is chronically dependent upon a continuous supply of androgen to maintain its appropriate cell content and functional activity. In particular, prostatic growth and secretion are modulated by 5-alpha-dihydrotestosterone (DHT) that is the active androgen at intracellular level.

The regulation of prostatic content of DHT is determined by the relationship between the rate of production and removal of this steroid in the prostate gland itself rather than by DHT blood levels. Normally, it is adequate to maintain a balance between prostatic cell loss and renewal such that neither involution nor proliferative overgrowth of the gland occurs. In both canine and human BPH, this balance is increasingly being shifted in favour of a large net increase in the total number of prostatic cells with advancing age. In addition, it has

been well documented that, even in young dogs, the experimental development of BPH can be induced simply by treatment of animals for 3-4 months with androgen and, in this regard, several androgens have been tested for their potential abilities to induce this disorders. One of the most serious consequence is due to the presence of fluid-filled cysts making the prostate susceptible to infection from bacteria ascending the urethra as accumulated prostatic fluid is an excellent media for bacterial growth[3]. Hematogenous spread of bacteria and spread from the kidneys and bladder via urine or from the testicles and epididimys via semen can also occur. Bacterial prostatic infection can be acute and fulminant or chronic and insidious leading to abscessation[4].

The hyperplastic prostate is highly vascularized and therefore the gland bleeds easily, which explains the common clinical sign of blood from the tip of the penis or blood in the urine. Blood loss in the prostatic urethra can be so intense that the ejaculate may appear completely red. Although presence of blood in the semen is typically considered to be a cause for infertility, dogs with blood in their ejaculates may be fertile. The reason for BPH being a common cause of infertility in the dog is probably due to the alteration of the biochemistry of the prostatic fluid whose important action of nutrition of spermatozoa is decreased. Prostatitis or abscessation are likely consequences of presence of blood in the prostate[4].

Diagnosis of canine BPH

BPH is diagnosed based on history (bloody penile discharge, difficulties in defecation/urination, poor semen quality and infertility, absence of haematological/biochimical alterations), physical exam and abdominal ultrasound (increase in prostatic size, presence of prostatic cysts), and if necessary a fine needle aspirate[3,4,5]. Urinalysis helps to rule out urinary tract diseases as a cause of penile discharge (cystitis should be treated prior to onset of BPH therapy to avoid confounding factors in the interpretation of results).

An enlarged, hypertrophic prostate may cause blood dripping from the tip of penis (the most common clinical sign), or it may grow and expand in the rectal canal, causing tenesmus and sometimes difficult defecation (less common)[6]. Other than the above signs, affected dogs are usually normal and the prostate on palpation is non-painful, symmetrically enlarged and with variable consistency. Urine may contain blood (gross or microscopic). If hyperplasia is accompanied by urethral discharge, this is typically haemorrhagic or clear but not purulent.

Prostatic enlargement may be also visualized on abdominal radiography as causing dorsal displacement of the colon and cranial displacement of the bladder[5]. On retrograde urethrocystography the prostatic urethra may be normal or narrowed and undulant with mucosal

irregularity, and the urethroprostatic reflux may be normal or greater than normal. On ultrasound, the prostate may appear diffusely hyperechoic with parenchymal cavities (which means that intraprenchymal cysts have developed). The canine prostate is best evaluated in the sagittal and transverse planes using 5.0 or preferably 7.5 MHz scanners. An enema should be administered prior to scanning to eliminate colonic contents which may mimic peripheral prostatic disease. Conditions such as cysts or abscesses are visualized easily. Other less distinct but echogenically complex areas may indicate neoplasia or areas of infection within the gland. Although technically a definitive diagnosis of BPH is only possible by biopsy, such an invasive approach is not necessary to institute a therapy if clinical signs are present, and from a practical standpoint ultrasound assessment of prostatic size and presence of cysts is often the only thing that is necessary to identify the problem and start dealing with it. No alteration of haematological or biochemical parameters are commonly observed in dogs with BPH[7].

Canine BPH can be difficult to differentiate from other most common prostatic disorders (prostatitis, prostatic cysts, carcinoma and adenocarcinoma) because of the similarity of clinical findings. In men with prostatic carcinoma the use of serum markers such as acid phosphatase (AcP) and prostate specific antigen (PSA) has facilitated determination of the extent of disease, evaluation of therapeutic response and detection of relapse after therapy. Information about these markers is still controversial in the dog. Serum and seminal prostatic AcP activities do not differ significantly between normal dogs and those with prostatic diseases, or among dogs with different prostatic disorders[8]; PSA is not detected in canine serum or seminal plasma. The major secretory product of the canine prostate is canine prostate-specific arginine esterase (CPSE) which constitutes more than 90% of seminal proteins in this species. CPSE is a known marker of dog prostatic secretion[9,10]. Screening for CPSE is of potential value in the aging intact male dogs. Its measurement is a useful and accurate method and should be considered as an alternative or complementary tool to conventional methods for the diagnosis of BPH in middle-aged dogs. CPSE is under testosterone control and, therefore, may serve as functional marker of the androgenic state and response to antiandrogenic therapy, either by receptor antagonists or 5-alpha-reductase inhibitors. Although further research is necessary to define the exact role of CPSE, it seems to be a promising diagnostic tool in nonneoplastic canine prostatic disorders.

Treatment for canine BPH

Surgical or pharmacological castration (using GnRH agonists) or the administration of estrogens, steroidal or non-steroidal antiandrogens can be used[11]. Although occasionally reported as an effective treatment for BPH, estrogens carry the potential risk of serious bone marrow

side effects (anemia, leukopenia, thrombocytopenia, pancitopenia) as well as the risk of growth of the fibromuscular stroma of the prostate which may cause metaplasia of the prostatic glandular epithelium and secretory stasis resulting in prostatic enlargement and predisposition to cyst formation, bacterial infection and abscessation[12]. Therefore, we do not currently advice using estrogens to treat canine prostatic hyperplasia. Estrogen receptor blocker may be used to treat BPH as they compete with androgen receptors thereby decreasing prostatic size and weight (although the altered ratio estrogen:testosterone is not modified which means that number and size of prostatic cysts do not change). Recently, tamoxifen (an estrogen receptor blocker with a mixed antagonist-agonist action) has been reported to be efficacious and devoid of side effects in male dogs with BPH (except for a decrease in libido and semen quality)[13]; following 28 days of treatment at the daily dose of 2.5 mg/day, tamoxifen caused a decrease in testicular and prostatic size as well as testosterone and libido[13]. Tamoxifen does not seem to have serious side effects and may be an interesting adjunct treatment for canine BPH, although there is no information on its long-term effect and safety and more studies are probably necessary before it can be prescribed routinely[13].

Castration – The most effective treatment to induce regression of prostatic hyperplasia is castration, after which prostatic size may decrease as much as 50% in 3 weeks and 70% over 9 weeks[14]. Orchiectomy has long been considered the treatment of choice for those dogs whose reproductive function is not important to the owner. As post-castration involution begins within days of surgery, prostatic size should be assessed 3 weeks post-operatively to make sure the involution rate is normal thus ruling out a more serious prostatic disease such as neoplasia or abscessation. Surgical castrations should not be performed in the presence of prostatic infections due to the risk of scirrhous cord development, in which case it would be better to administer a specific antibiotic treatment based on semen culture and sensitivity. With regard to orchiectomy, one important thing to consider is that incidence of prostatic carcinoma in adult/elderly dogs could be higher in castrated rather than in intact dogs; reasons for this are not entirely known yet, but it is speculated that once prostatic atrophy starts, neoplastic cells already present will increase their growth rate perhaps due to lack of suppressing action of testosterone[15,16,17]. For this reason we do not currently advice our clients to castrate their adult to elderly dogs unless it is strictly necessary (i.e. if there is a testicular tumor).

Steroidal antiandrogens - Steroidal antiandrogens compete with androgen receptors and perhaps also with DHT receptors at the cellular level in target organs. Compounds such as megestrol acetate,

medroxyprogesterone acetate, delmadinone acetate, chlormadinone acetate and ciproterone acetate have been successfully used in the dog, although for the majority of them there is only a limited amount of experimental data on their effectiveness in the canine. Their action causes a sort of pharmacologic castration and is rather precociously observed during treatment, as improvement can be observed already after 7-15 days. Medroxyprogesterone acetate has been used as a single SC dose of 4 mg/kg; although prostatic effects were not assessed, the treatment significantly reduced serm testosterone concentrations, which implies that it might be used also for the treatment of BPH[18]. Chlormadinone acetate has been used at the dose of 1-2 mg/kg orally for 1 month, or as a subcutaneous 12-month implant of 5.0 mg/kg[19], but oral doses as low as 0.03 to 0.3 mg/kg/day are effective in dogs with BPH[20]. Delmadinone acetate has also been used at doses of 1.5 to 5.0 mg/kg SC to be repeated every 1-2 weeks[21-23] with the lower dosage being the one prescribed in commercial veterinary formulations. Ciproterone acetate is another progesterone derivative with a very strong antagonistic effect on DHT receptors when used at the daily dose of 0.5-1.0 mg/kg per os for at least 2-4 weeks; the drug is well tolerated in dogs and causes decreased libido and spermatogenesis and reduces prostatic size[24-25]. We currently use it as an adjunct treatment in dogs with acute clinical signs of BPH such as rectal compression, urethral compression or signs of prostatitis. Cyproterone acetate has a very quick action with signs disappearing already at the end of the first week of treatment, therefore it is helpful whenever the dog is suffering from signs related to BPH or when a worsening of the condition is anticipated such as during the first 1-2 weeks following administration of a GnRH agonist implant (see over). Its effects are rather long-lasting, with a 3-4 week course of oral administration being enough to maintain the dog for a few months without clinical signs.

Osaterone acetate (OA) is a more recent progesterone derivative commercially available in several European countries as a veterinary product for use in dogs with BPH[26,27]. As an antiandrogen, OA is an analog of chlormadinone acetate (but it is 5 times more potent in vitro than chlormadinone itself) with a specific inhibitory action on prostatic volume in laboratory animals and dogs[23]. It is marketed as an oral medication with each package containing 7 pills of different dosages depending on body weight: 1.875 mg, 3.75 mg, 7.5 mg and 15 mg for dogs of 3-75 kg, 7.5-15 kg, 15-30 kg and 30-60 kg, respectively. Dosage is 0.25-0.50 mg/kg. Treatment consists of 1 pill/day for 7 consecutive days. OA is slowly metabolized by the liver with a very long half-life of 198±110 hr. For this reason, a 7-day course of treatment allows for a pharmacological concentration of the drug to last for 6 months. OA is very effective in treating canine BPH, and works well also in case of prostatitis[28]. Being a progestogen derivative, OA may cause suppression of the pituitary-hypophysial-adrenal axis with reduced cortisol production and/or low or no response to ACTH stimulation tests lasting for days or weeks after treatment withdrawal. Although this is normally not a problem in healthy dogs, it should be considered when dealing with post-operative cases or in case of trauma, while treatment with OA should be avoided in dogs with hypoadrenocorticism. Semen quality appears not to be affected initially, and may actually improve when poor at treatment onset due to the prostatic condition. However, progestogens are known to cause a progressive deterioration of semen quality with time; in the case of OA, there is little if any data on fertility during the last half of the 6-month treatment.

Non-steroidal antiandrogens – Non-steroidal antiandrogens include finasteride and flutamide. Finasteride inhibits 5-a-reductase (the enzyme responsible for the final transformation of testosterone into di-hydro-testosterone or DHT) thereby lowering the concentration of DHT which is the active metabolite at the level of target tissues, without altering serum testosterone concentrations. This leaves spermatozoa production undisturbed, which makes finasteride a good choice for breeders (although a chronic use may be associated with a decrease in ejaculate volume as well as decrease in semen quality). Finasteride is only approved for use in men, but it is well known to produce a dose-dependent decrease in prostatic size also in dogs. It can be used at the daily dose of 1 mg/kg/day, PO, for up to 4 months resulting in a 50%–70% reduction in prostatic hypertrophy with no negative effect on semen quality[29]. Lower dosages of finasteride (0.1-0.5 mg/kg/day, PO, for 4 months) cause a slightly lower but still curative effect in terms of reduction of prostatic volume by >40%, resolution of clinical signs, reduction of DHT concentration, maintainenance of normal testosterone levels, and have no deleterious effect on semen quality, fertility, or libido[30]. The low dosage (0.1–0.5 mg/kg) of finasteride allows for a convenient dosing of one 5-mg capsule/day for dogs weighing 10–50 kg; however, it is advisable to use 1.5 mg (approximately 1/3 of a 5.0 mg pill) for dogs ≤15 kg body weight, 2.5 mg (approximately half pill) for dogs of 15-30 kg body weight, and 5.0 mg for dogs of >30 kg body weight. Finasteride is well tolerated and can be administered for long periods of time. We currently use finasteride in breeding dogs both to induce remission of clinical signs of BPH as well as to keep the condition under control with 1-2 treatment cycles/year depending on severity of clinical signs. Unlike cyproterone acetate, finasteride is fairly slow to show its efficacy, as clinical signs may take up to 3-4 weeks to disappear, and tend to appear again within a few weeks of treatment withdrawal. Flutamide is a human antiandrogen which can cause a significant decrease

in prostatic size as detected by ultrasonography within 10 days. When administered to research dogs at 5 mg/kg/day PO for 1 year, it did not alter libido or sperm production[31]. Also flutamide is a human drug not approved for use in veterinary medicine, although it appears safe, effective and well tolerated in dogs.

GnRH agonists - Following administration of a GnRH agonist, prostatic size decreases in parallel with the decrease of testosterone. When adult dogs are implanted with 4.7 or 9.4 mg deslorelin their prostatic volume decreases more than 50% from week 6 onwards[32-33], and serum T concentrations decreases 90% from week 3 onwards[34]. We have observed disappearance of conspicuous (>17 mm diameter) prostatic cysts following treatment with a single 4.7 mg deslorelin implant[35] as well as of larger (20x25 mm) prostatic cysts in adult male dogs with clinical signs of benign prostatic hypertrophy undergoing treatment with a 4.7 mg deslorelin acetate administered every 6 months (unpublished observation). In milder or less complicated cases of BPH, an improvement of the clinical situation of treated dogs is observed often without any additional pharmacological treatment already at the first follow-up visit. In cases in which prostatic size is markedly increased and in the presence of rectal or urethral constriction or other signs of discomfort, deslorelin may cause a temporary worsening of the clinical situation because of the initial (first week) rise in testosterone secretion due to temporary hypersecretion of pituitary gonadotrophins (flare effect); in these cases we normally add a 2-week course of a progestogen derivative such as cyproterone acetate. The mechanism of action of GnRH agonists in achieving a decreased prostatic size is probably through the decrease in serum testosterone, which also cause a (reversible) sterility. Therefore, GnRH agonists should not be used for the treatment of BPH in male dogs intended to be used for breeding.

Clinical management of canine BPH

The best way to prevent the development of clinical BPH in the dog is to identify its early pre-clinical signs by performing a regular monitoring of prostatic conditions by ultrasound. If signs of BPH (such as presence of prostatic cysts or increased prostatic size) are observed during a routine check while the dog is asymptomatic, owners should be advised to watch for the development of clinical signs in order to start treatment as soon as possible. There is little information on the value of a preventive treatment for BPH in the dog. In men, preventive treatment is often discouraged because of the many side effects which may be caused by alpha-1 adrenergic antagonists (the most common treatment for human BPH). However, incidence of side effects of such drugs in the dog is unknown, and most importantly alpha-1 adrenergic antagonists are not 1st-choice drugs for canine BPH, while steroidal or non-steroidal antiandrogens or GnRH agonists are more indicated for this purpose. Side effects of long term treatment with steroidal antiandrogen include termporary adrenocortical suppression and a decrease in libido and semen quality. All steroidal antiandrogens should be avoided in breeding animals because of their potential negative impact on fertility. However, the use of medroxyprogesterone acetate (4 mg/kg SC every 3-5 months), chlormadinone acetate (0.1-0.3 mg/kg/day per os for 6 months) as well as osaterone acetate in its well known weekly treatment regimen have been associated with a normal or acceptable fertility in male dogs at least for the first few weeks and therefore might be used for limited amounts of time in these patients.

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AQUATIC PET MEDICINE AND WORLD RABIES DAY

RABIES VACCINATION OF DOGS TO PROTECT PEOPLE: THE GLOBAL AND ASIAN EXPERIENCE FOLLOWED BY BOEHRINGER INGELHEIM/WSAVA WORLD RABIES DAY PANEL DISCUSSION

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Rabies vaccination of dogs to protect people: the Global and Asian experience

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Introduction

The global rabies conference in Geneva in December 2015 set the goal of eliminating canine-mediated human rabies by 2030 through the Global Framework for the Elimination of Dog-Mediated Human Rabies. Five pillars were identified to achieve rabies elimination: S-Socio-cultural; T-Technical; O-Organizational; P-Political; and R-Resources (STOP-R). Two of the key aspects highlighted were the following: importance of strengthening the intersectoral collaboration through the One Health approach (O-Organization); implementation of mass dog vaccination which is the most cost effective intervention to achieve dog-mediated human rabies elimination (T-Technical).Rabies is a neglected tropical disease wherein 59,000 human lives each year are lost mainly in rural areas of Africa and Asia. It has been controlled in Latin America and Caribbean wherein only 10 deaths were reported due to dog –mediated rabies in 2016 (Haiti and Guatemala). Almost 60% of global human rabies deaths (35,172 human rabies deaths) occur in Asia with India accounting to almost 60% of the deaths in the region. Africa, on the other hand, record a high human rabies deaths of 21, 476 annually (36% global deaths).

Importance of Dog Vaccination in Rabies Control

The economic burden of rabies is immense with USD 8.6 billion loss per year, majority of which is productivity loss due to premature deaths (54%). Although dog vaccination is the most effective way to control rabies, only 2% of the cost is allocated to dog vaccination and population control. It has been proven in Latin America that investing in dog vaccination to achieve 70% coverage for mass dog vaccination to maintain herd immunity against rabies is effective the decreasing dog-mediated human rabies.

Awareness about rabies, dog vaccination and prompt access to post-exposure prophylaxis for bite victims are the key in the control and prevention of dog-mediated human rabies. Although dog vaccination seems to be the most cost-effective and simple method to prevent

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rabies, the challenge of ensuring 70% vaccination coverage of the dog population in a country is complex. This presentation will focus on how the global community addresses the challenge on the issue of mass dog vaccination and how individual communities are gaining success in controlling dog-mediated human rabies through dog vaccination.

Challenges and Solutions

Lack of access to safe and efficacious dog vaccines is one of the factors that impact conduct of mass dog vaccination at a country level. Since 2012, the World Organization for Animal Health (OIE) established regional vaccine banks with the aim to support national governments with easier access to high quality, affordable vaccines. By December 2017, 19 million dog rabies vaccines have been supplied to 28 countries in Asia and Africa through the vaccine bank.

Limited focus on strategic planning and allocation of financial resources hinders the implementation of mass dog vaccination in the field. Based on the successful elimination at the regional level of Latin America, a regional rabies elimination approach through the establishment of regional rabies elimination control networks in Africa, Asia and Middle East is underway. These regional meetings provide venue for conduct of workshops such as the Stepwise for Rabies Elimination (SARE) to develop work plans for rabies elimination as well as Global Dog Rabies Elimination Pathway (GDREP) to determine financial requirements to support mass dog vaccination.

At the field level, operational challenges include the lack of manpower to support MDV and proper estimation of dog population as well as dog vaccination coverage. The Global Education Platform (GEP) of Global Alliance for Rabies Control (GARC) provides free online courses to support training of vaccinators such as the Rabies Educators Certificate (REC) and the Animal Handling and Vaccination Course (AHVC). GARC as well as Humane Society International (HSI) and Mission Rabies have also developed computer based tools to support dog population estimation and vaccination coverage. Oral rabies vaccination is now also being seen as a possible support to mass dog vaccination specifically targeted for dogs that are free roaming and difficult to handle for parenteral administration of rabies vaccine.

Lack of knowledge about rabies and misconceptions on its prevention and control hinder the community to support local rabies programs. Innovative methods to mobilize the community have been implemented in different countries through community education and school based intervention.

Success Stories

Countries in Latin America have shown that a regional approach focusing mainly on mass vaccinating dogs together with proper animal bite management and surveillance can decrease rabies human deaths and confirmed dog rabies cases significantly. A total of 50 million dogs are estimated to be vaccinated in the region annually.

In Bangladesh, thousands of dog catchers and vaccinators were trained as result of a snow-ball technique capacity building mechanism. Through this mechanism, 70% mass dog vaccination coverage was achieved in 1 week wherein majority of the dogs are free roaming.

Focusing on mass dog vaccination together with other rabies control components such as information campaign, strengthened surveillance and better access to PEP were also key to reducing human rabies deaths (Sri Lanka) with some countries achieving zero human rabies deaths (Mexico). Strengthening mass dog vaccination campaigns have been also crucial in controlling rabies in Kwazulu Natal (South Africa), Visayas, (Philippines) and Tanzania.

One Health Approach from the Global to the Local Level

One Health approach emphasized on the intersectoral approach to fight rabies. As a global response to the goal of eliminating dog-mediated rabies by 2030, a United Against Rabies collaboration composed FAO, OIE, WHO and GARC has been established to support countries to achieve rabies elimination by sharing existing tools and expertise. Regional networks have also been established in Africa, Asia and Middle East to serve as a venue for national program managers from the animal health and medical sector together with regional partners to plan a regional approach for rabies elimination.

At the country-field level, Integrated Bite Case Management (IBCM) serves as a basis for animal health and medical personnel to collaborate to assess the risk of an animal bite and plan the best course for animal bite management strategy. The impact of IBCM is that it can reduce the cost of treatment of rabies in humans by determining rabies risk in biting animal (low risk if dog is regularly vaccinated; high if dog is free roaming, non-vaccinated and have signs of rabies).

Private practitioners and professional/civic organizations play a huge role in the rabies elimination efforts. This is seen through the cooperation between sectors during World Rabies Day celebration. In the Philippines, the bayanihan (cooperation and community) spirit has been the main driving force in the rabies elimination efforts such as the training of village health workers to support mass dog vaccination. In remote communities in Northern Tanzania, dog vaccination is scheduled in the

community during the deworming of children.

The goal of global elimination of dog-mediated human rabies faces a lot of challenges but inspiring stories of cooperation and sharing of innovations at the global to the local level have shown that reaching the 2030 rabies elimination is feasible.

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SVA EXOTICS

CLINICAL PATHOLOGY OF EXOTIC PETS

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Abstract

Clinical pathology is a commonly used diagnostic tool in exotic pet medicine. In many cases, blood collection techniques are rarely as simple as they are small animal medicine, and the interpretation of the results requires knowledge of species variation in haematologic and biochemical parameters in response to the patient's clinical status. This presentation will discuss blood collection techniques in birds, reptiles and common small mammals. Interpretation of laboratory results will be explored, using case studies to illustrate species variation and response to disease.

Some pearls to help you get the best possible results

Interpretation of avian and biochemistry results is only part of the art of using haematology and biochemistry to assess your patients. To get the most out of a submitted sample it is important to provide your laboratory (external or in-house) with the best possible sample in the best possible condition. The following are some hints for doing that.

1. Submit the largest size sample you can without compromising your patient's wellbeing. Generally we can collect 5% - 10% of the patient's blood volume (equal to 0.5 - 1% of its bodyweight), but the amount we collect in paediatric and anaemic patients is less. It is always worth speaking to your lab to find out what size sample they need, but negotiate with them if they are want volumes larger than 0.5ml of whole blood.

2. Avoid haemolysis by using an appropriate size needle (23-27g) and using gentle negative pressure on the plunger to prevent turbulence during collection. Remove the needle before placing the sample into the collection bottles.

3. Make several blood smears before placing the sample into anti-coagulant. Rather than using another microscope slide to push the blood along the slide, use a cover slip to drag the sample along the slide.

4. Use BD Microtainers® for your sample, but don't under or over fill the tubes. Under-filling the tube mixes excessive anticoagulant with the blood; overfilling may result in a clotted tube.

5. Use lithium heparin tubes (green top) for bio-chemistry and Na EDTA tubes (purple top) for haematology, unless your lab makes a different recommendation.

6. Ensure the blood and anti-coagulant are mixed immediately by rolling the tube along the palm of your hand or gently inverting the tube 10 times. Do not shake the sample, as this may result in haemolysis.

7. If sample processing is likely to be delayed more than a few hours, centrifuge the lithium sample and separate the plasma from the red cells. Decant the plasma and place in another lithium heparin bottle. This prevents artefacts associated by prolonged contact time with the erythrocytes.

8. Avoid over-interpretation of results. Look for significant elevations or decreases, not changes that could be within the range of error or normal variations for the machine or patient. Be aware of artefactual changes affecting some parameters e.g. hyperkalaemia due to haemolysis.

9. Always treat your patient, not your test results!

Blood collection and handling

Birds: Blood can be collected from the right jugular vein (the left jugular vein is accessible, but is much smaller), the basilic vein (on the medial aspect of the elbow) or the medial tibiotarsal vein (in large birds). The right jugular vein is usually preferred in companion birds because of the relatively easy access; it lies under an apteryla and, with practice, a sole operator can both restrain the bird and perform the venepuncture. It must be remembered that avian veins have thin walls and tear easily. Coagulation in birds usually relies on extrinsic clotting pathways requiring tissue thromboplastin, rather than the intrinsic clotting pathways utilized by mammals. Care must therefore be taken to prevent accidentally tearing the vein wall, which can lead to a rapidly fatal haemorrhage. It is advisable to apply digital pressure to the venepuncture site for 30–60 seconds to minimize haematoma formation. Using a 25–29-gauge needle minimizes the iatrogenic trauma to the vein, but the smaller the needle bore the more likely haemolysis is to occur during collection. Once the needle has entered the vein, avoid using excessive pressure to draw back; this will prevent both collapsing the vein and haemolysing the sample. It is sometimes advantageous to use a heparinised syringe for blood collection.

Reptiles: Blood can be collected from most reptiles with varying degrees of difficulty.

1. Snakes – collect from the ventral tail vein or, if unsuc-cessful, from the heart
2. Lizards – collect from the ventral tail vein. Be careful of autonomy in skinks and geckos
3. Turtles – collect from the jugular vein or the dorsal sub-carapacial venous sinus

Small mammals: There are a number of venepuncture sites that can be used in small mammals

Vein	Guinea Pig	Rabbit	Rat and mouse	Ferret
Marginal ear vein		X		
Jugular	X	X		X
Anterior vena cava	X		X	X
Lateral saphenous	X	X	X	
Cephalic	X	X		X
Lateral tail			X	
Orbital venous sinus			X	

The reasons why blood results may vary from reference intervals

Haematologic or biochemical parameters in the blood can be influenced by either physiological or pathological processes. Physiological variations can be due to age, sex, body fat to muscle ratio, nutritional status, reproductive status and species. However, pathological processes, including cellular damage or abnormal function of an organ system (or systems), often produce significant changes in parameters.

There are three major causes of abnormal laboratory results:

• Normal variation between species and individuals
• Artefacts
• Pathology

Normal variation between species and individuals

We are dealing with three classes of exotic pets, with hundreds of species presented for veterinary care. There are major differences in anatomy, physiology, form and function. Some are carnivorous, some are herbivorous, and some are omnivorous. It is unrealistic to expect that they would all conform to a relatively narrow range of haematologic and biochemical values.

Other variations arise between individuals of the same species. These variations occur because of differences in age, sex, diet, husbandry, etc. For this reason some clinicians recommend establishing a set of normal values for individual animals during annual health examinations, and then using these values as a comparison should the animal become ill.

Artefacts

When interpreting a blood result, care must be taken to distinguish between abnormal results due to disease, and abnormal results due to other factors. These other factors, referred to as artefacts, can occur for a variety of reasons, including:

- **Physiological changes**

Stress due to transport and handling of the patient can lead to a release of endogenous corticosteroids, resulting in changes in the haemogram and in blood glucose.

Lipaemia, while occasionally seen in diseases of the liver and reproductive system, can also occur naturally in the reproductively active female. Regardless of the cause, lipaemia can cause false elevations in bile acids, protein, calcium, phosphorus and uric acid.

It may also falsely decrease amylase.

Postprandial lipaemia is uncommon in pet birds and mammals, so fasting will not help; the clinician needs to check with the laboratory if the sample submitted was lipaemic before interpreting these biochemistries.

- **Previous therapy**

Before interpreting biochemistries, the clinician should consider if any treatment given prior to the sample collection could have had an effect on the results. Therapy given by another veterinarian or in an attempt to stabilize a crashing patient can have marked effects.

Parenteral fluids can dilute biochemistries; exogenous corticosteroids can markedly elevate aspartate aminotransferase (AST), creatine kinase (CK) and lactate dehydrogenase (LDH); intramuscular injections, particularly of irritant drugs, can do the same.

- **The clinical condition of the patient**

Trauma, starvation and dehydration can all have marked effects on biochemistries, and need to be considered when interpreting results. Trauma can cause elevations in AST and CK and possibly glucose; starvation can lower glucose and also elevate AST and CK if protein catabolism has begun; dehydration can elevate uric acid.

- **The collection method**

Ideally, sample collection should be performed in such a manner that it has minimal impact on the patient while providing an artefact-free sample suitable for analysis. This usually requires venepuncture to be performed on a minimally stressed patient. Inexperienced clinicians may need to consider gaseous anaesthesia in order to collect a good sample without the bird struggling. Haemolysis can cause elevations in bile acids, LDH, CK, potassium and phosphorus.

Glucose and albumin may be decreased. Calcium may be elevated or decreased, according to the methodology used.

- **Storage and transport of the sample**

Blood collected for biochemistry analysis should be placed immediately into a lithium heparin tube. Ideally, miniature tubes as used in medical paediatrics should be used. The sample should be gently rolled or rocked; clotting must be avoided, but haemolysis must be as well. If the analysis is to be performed in-house, it should be processed immediately. If a delay is likely, or if the sample is to be shipped to an outside laboratory, the sample should be centrifuged and the plasma harvested. Sending whole blood to an outside laboratory can result in decreased glucose (as cell metabolism continues) and haemolysis.

EDTA tubes are unsuitable for biochemistry analysis in most species, but can be used for haematology, lead analysis and fibrinogen determination

Haematology

The complete blood count (CBC) is an important test in determining many disease states. In most cases a CBC involves assessing:

- The erythrocytes, through the determination and assessment of:
- The haematocrit or packed cell volume (PCV).
- Erythrocyte morphology.
- Reticulocytes.
- The leucocytes, through the determination and assessment of:
- The total white cell count.
- The leucocyte differential count.
- The morphology of the leucocytes.
- Thrombocyte numbers.

Erythrocytes

- The PCV of most birds lies between 0.4 l/l and 0.55 l/l. Non-flighted birds, such as chickens, usually have a lower PCV as they do not have the same oxygen demand that flight requires.
- The normal PCV in most reptiles is lower than mammals and birds, and values of 20%- 35% are not unusual. Again, this may reflect their lower metabolic rate and subsequent lower oxygen requirement.
- Small mammals generally lie between these two extremes, with a PCV generally between 35 – 48%.

A low PCV can indicate blood loss, anaemia, shock or haemodilution following fluid therapy.

A high PCV indicates dehydration or polycythaemia (primary or secondary).

Morphological abnormalities seen in erythrocytes include:

- Excessive polychromasia. Polychromasia is an indicator of the patient's erythrocyte regenerative abilities. Although some polychromasia (1–5%) is normal, excessive polychromasia indicates a regenerative response to blood loss or anaemia.

- Reticulocytosis, especially when combined with increased polychromasia, is seen as a regenerative response to blood loss or anaemia.

- Anisocytosis. Variation in the size of erythrocytes is occasionally seen in peripheral blood smears as a normal finding. However, the number increases in response to anaemia.

- Poikilocytosis, or variable cell shapes, may represent artefactual error, but is also seen when severe systemic infections affect the bone marrow. Erythrocytes may appear round, elongated or irregular. The nucleus may vary in appearance, location and number. Erythrocytes that appear round with oval nuclei are indicative of accelerated erythropoiesis. Binucleated erythrocytes may also indicate abnormal erythropoiesis in association with severe, chronic inflammatory processes and neoplasia. Poikilocytes are susceptible to damage, and therefore have a shorter life.

- Erythrocytic ballooning has been reported to be commonly associated with, although not pathognomonic for, lead toxicosis in birds. It is also seen with 'conure bleeding syndrome'. There are bulges in the normal ellipsoid shape of the erythrocyte, often accompanied by areas of hypochromasia.

- Haemopararasites are occasionally seen in the erythrocyte cytoplasm of wild-caught birds and reptiles, or those exposed to biting insects.

Leucocytes

The white blood cell (WBC) count and differential are important tools in assessing a patient's response to disease or injury. The WBC count in birds and reptiles can be determined using three testing methods:

- An automated count, which has recently become available in some laboratories.

- An estimated WBC count determined from a blood smear by counting all leucocytes in 10 high-power (40×) microscopic fields, dividing by the number of fields, and then multiplying this average by 2,000, giving a total WBC/µl.

- The Unopette method using phloxine B stain and a haemocytometer to count eosinophils and heterophils. This count is compared with the percentages of these cells in the differential and the WBC count is calculated using the formula: WBC = (Total Het + Eos) / (% Hets + % Eos) × 100. (Note: In late 2007 the Unopette system was discontinued by the manufacturer. An alternative test in the Avian Leukopet®, Vetlab)

Leucocytosis can be normal in young animalss, but it can also be due to:

- Stress.

- Inflammation, often associated with bacterial and fungal infections.

Leucopaenia can be due to:

- Chronic inflammation or disease, often with an acute decompensatory episode at the time of presentation.

- Overwhelming bacterial and viral infections

- Artefacts resulting from poor sample handling (blood clotting) or technique.

White cell differential counts are best obtained from fresh blood smears, as cellular morphology can be affected by anticoagulants in blood collection tubes. A differential count is typically obtained by examination of stained smears under high magnification. Both the type and morphology of the white cells seen are recorded. In many cases the differential count and cellular morphology give more indication of a bird's health status than the total white cell count.

Heterophils

The heterophil is the avian and reptile equivalent of the mammalian neutrophil. While it has a similar function to the neutrophil, morphologically it appears quite different. The nucleus contains coarsely clumped chromatin and usually has two to three lobes. The cytoplasm contains eosinophilic, spherical, oval or spindle-shaped granules. It is, in most species, the predominant white cell. Heterophils lack lysozyme which is why birds form caseated, rather than liquid, pus. Abnormal changes seen with heterophils include:

- Heterophilia

- Heteropaenia

- Toxic heterophils (increased cytoplasmic basophilia, vacuolization, nuclear degeneration, degranulation or abnormal granules)

- Immature (band) heterophils

Eosinophils

The eosinophil is a round cell with a slightly basophilic cytoplasm (in contrast to the colourless cytoplasm of the heterophil). The granules are usually rounded, although shape and colour may vary greatly between species. The granules are distinctly eosinophilic and brighter in colour when compared with the heterophil granules. The function of eosinophils is still largely unknown; eosinophilia is rare, sometimes associated with parasitic infections but more commonly with marked tissue damage.

Lymphocytes

Lymphocytes are second only to the heterophil in frequency in most species. Size and shape varies, with small, medium and large cells that may be round or moulded around neighbouring cells being seen in the same smear. Occasional reactive lymphocytes are a normal finding, but large numbers indicate marked antigenic stimulation as seen in severe infections (e.g. severe viral infections, chlamydiosis, Aspergillosis, salmonellosis and tuberculosis).

Lymphocytosis is seen in:

- Chronic infectious or inflammatory conditions.

- Lymphoid leukaemia.

- Normal finding in Amazons and canaries.

Lymphopaenia is seen in:

- Viral infections and diseases that cause bursal damage or bone marrow suppression.

- Relative to a marked increase in heterophils.

Monocytes

Monocytes are the largest of the mononuclear leucocytes, but are rarely seen in peripheral blood smears. They spend only a short time in circulation before passing into tissues and becoming macrophages. The eccentric nuclei are either round, elongated or indented, and the cytoplasm typically stains a blue–grey colour with a reticular or finely granular appearance, with occasional vacuoles. Care must be taken not to confuse them with large lymphocytes.

Monocytosis is most commonly associated with chronic granulomatous infections

Azurophils

Mature azurophils are similar in size to heterophils and vary in shape from round to monocytoid in appearance. The nuclei are usually eccentric and the cytoplasm is bluish grey with azurophilic granules. Azurophils occur at relatively low numbers in healthy reptiles, but are increased in bacterial infection and cellular necrosis.

Basophils

Basophils are uncommon in peripheral blood smears of birds. They appear as small cells with clear cytoplasm and spherical basophilic granules. The nucleus stains a light blue colour. Care must be taken not to confuse them with immature heterophils. Basophilia has been reported in respiratory disease (e.g. air sac mite in canaries), chlamydiosis and tissue trauma more than 48 hours old. In birds and reptiles basophils appear to play an important role in early inflammatory and immediate hypersensitivity reactions, but differ from those in mammals by not contributing to delayed hypersensitivity.

Thrombocytes

Thrombocytes are small, oval, nucleated cells that can be differentiated from erythrocytes by their size (they are smaller than erythrocytes) and their nucleus, which is larger, more rounded and darkly basophilic-staining. The cytoplasm is colourless or a faint blue colour with one to two small basophilic inclusions at the poles. Total counts are difficult and not routinely performed as the thrombocytes tend to clump. However, there are typically 1–2 cells seen per high-power field. Their function is unclear; they contain little thromboplastin, so it is unlikely that they initiate clotting. With bacterial infections they tend to increase in numbers and become activated (pseudopodial formation and vacuolation) and tend to aggregate in clumps. They appear to have some phagocytic activity.

Thrombocytosis is rarely reported and may arise as in response to thrombocytopenia. Thrombocytopenia may occur due to bone marrow suppression or disease processes causing an excessive demand (e.g. viral diseases such as circovirus, reovirus or polyomavirus).

Clinical biochemistry

Clinical biochemistry involves the measurement of specific groups of chemicals within the body and the interpretation of the results obtained. These chemicals include:

- Metabolites. Those chemicals that are produced as the end-products of various metabolic processes within the body.
- Tissue enzymes, which catalyse chemical reactions within the body without being altered themselves.
- Electrolytes, including sodium, potassium and chloride.
- Minerals, such as calcium, phosphorus and magnesium.
- Bile acids, produced in the liver from cholesterol and used in the emulsification of dietary fats.
- Lipids

Liver enzymes

The detection of liver disease through biochemistry is complicated by the fact that there are no specific 'liver enzymes' that can be evaluated conclusively in each and every case. Liver disease can be broadly classified into three conditions: hepatocellular rupture, decreased hepatic function and cholestasis. These conditions can occur either separately or concurrently.

Hepatocellular rupture

This releases intracellular enzymes, which then reach elevated levels in the blood. These so-called 'leakage enzymes' include:

- Aspartate aminotransferase. This cytosolic enzyme is found in many tissues in the body, but the highest concentrations are found in skeletal muscle and liver. Significant elevations usually represent either muscu-

lar or hepatocellular damage. AST, therefore, must be interpreted alongside CK (released from damaged muscle) to distinguish between the two. In general, an elevated AST with a normal CK indicates hepatocellular rupture. However, CK has a much shorter half-life than AST; a single-point muscle injury (e.g. an injection) 4–7 hours before sample collection could duplicate this biochemistry pattern. Although AST is considered to be the most useful liver enzyme, it cannot be considered in isolation as an indicator of liver disease.

- Glutamate dehydrogenase (GLDH), a mitochondrial enzyme, is the most specific enzyme for the detection of liver disease, but its sensitivity is low. Because it is bound to mitochondria, extensive and severe liver damage is required before elevations are detectable.

- Lactate dehydrogenase is not specific to any tissue; its main advantage lies with a half-life shorter than CK. Persistent elevation in the presence of normal CK is strongly suggestive of liver disease.

- Alanine aminotransferase (ALT) and alkaline phosphatase are not considered useful in detecting liver disease in birds, reptiles and rabbits. ALT in these species is very nonspecific for the liver, and normal levels have been shown in cases with severe liver damage.

Decreased liver function

Decreased liver function can occur with any number of liver diseases, not all of which involve hepatocellular rupture. Chronic cirrhosis, amyloidosis and hepatic lipidosis can all have an adverse effect on liver function without causing any cellular damage. In these cases a 'liver function test' is necessary to detect the problem. Bile acids serve this purpose well. Produced in the liver, they are excreted in bile into the small intestine where they act to emulsify fat. Most of the bile acids are then resorbed in the small intestine, enter the portal system and are taken up by the liver to be recycled. Elevated levels occur when there is impairment of the liver's ability to remove bile acids from the portal circulation. A two- to fourfold increase in bile acids indicates a significant decrease in liver function. It needs to be noted though that a severely dysfunctional liver (e.g. end-stage cirrhosis) may not be able to produce normal levels of bile acids, leading to low to normal results. Total protein, especially albumin, may also be decreased with decreased liver function.

Cholestasis

Cholestasis occurs when the biliary system is partially or totally obstructed. This can be seen with biliary neoplasia, pancreatic disease or diffuse swelling of the entire liver. Gamma glutamyl transferase (GGT) is an enzyme found in the cell membranes of the bile ducts. Elevations can be seen in cholestatic disease (e.g. bile duct carcinoma), but it is considered to be a relatively insensitive test for liver disease in parrots.

Bilirubin is not produced in birds and reptiles; they utilize biliverdin instead. There are no commercial assays for biliverdin. Bilirubin is useful for evaluating cholestasis in small mammals.

Kidney function

Birds: The end-product of protein metabolism in birds is uric acid. It is produced in the liver, enters the circulation and is then secreted by renal tubules (>90%) or filtered in the glomerulus (<10%). Significant loss of renal tubules will therefore result in elevations of uric acid. Dehydration is less likely to cause hyperuricaemia because glomerular filtration is relatively unimportant.

At first glance it would appear that uric acid offers a sensitive and specific test for renal disease. There are, however, several confounding factors. Firstly, species differences: carnivorous birds have higher normal uric acid levels than granivorous birds. Secondly, age: juvenile birds may have lower levels than adults. Thirdly, although significant elevations usually indicate renal disease, normal levels do not mean the kidneys are normal: mild increases could indicate early renal disease or dehydration (or both). There must be severe renal damage before uric acid levels begin to rise.

Because of this relative insensitivity of uric acid in detecting renal disease, levels are best interpreted alongside a determination of the bird's water intake and loss and a physical examination. To distinguish renal disease from dehydration, the patient's haematocrit, total protein and blood urea nitrogen (BUN) should be evaluated concurrently. Dehydration can lead to decreased glomerular filtration rates (GFRs), in turn leading to elevated levels of BUN; this same decrease in GFR can lead to elevations of uric acid without primary renal disease being present. It is therefore prudent, in cases of an elevated uric acid level, to rehydrate the patient over 2–3 days before definitively diagnosing renal disease. Persistent hyperuricaemia after fluid therapy, and with haematocrit, total protein and BUN returning to normal, confirms a diagnosis of renal disease.

Creatinine is generally accepted as being of little or no value in evaluating renal function in birds. Phosphorus elevations are usually not seen in birds with renal disease.

Reptiles: Hyperuricaemia in reptiles is commonly associated with high protein diets, a recent meal, dehydration, and gout. It is not commonly observed in reptiles with renal disease. This is because it is excreted in the proximal tubules and so it is usually widespread renal disease that affects blood concentrations. Elevations in urea nitrogen may be associated with pre-renal, renal, or post-renal disease. Hypocalcaemia and hyperphosphataemia are common in reptiles with renal disease, with a Ca:P ratio less than 1.0 been strongly

suggestive of renal dysfunction.

Rabbits: Renal disease will increase both BUN and creatinine concentrations. In chronic renal disease, an increase in the phosphorus concentration can be observed. Interpreting high calcium concentrations can be difficult as clinically normal rabbits can have high calcium concentrations. Rabbits appear to be more efficient than other mammals in absorbing calcium from their gastrointestinal tract. It is important to realize that high serum calcium concentrations in rabbits are not always associated with disease and may be an indication that the diet is too high in calcium.

Reproductive activity

Birds: Clinical biochemistries can tell the clinician little about the male reproductive tract; they can, however, reveal something about the activity of the female reproductive tract. Oestrogen, produced by developing follicles, induces the production of calcium-binding protein and vitellogenesis in the liver. The net result of this activity is an increase in circulating total protein, calcium, triglycerides and cholesterol. The serum may appear lipaemic.

Radiographic evidence of hepatomegaly and increased long bone density can confirm reproductive activity. It should be noted, though, that normal calcium and protein do not reflect a lack of reproductive activity. Elevated triglyceride levels can be due to dietary factors, liver disease, ovarian activity, pancreatic disease, or causes not yet understood in parrots.

Reptiles: Elevated albumin concentrations in female reptiles during the breeding season are indicative of impending egg-laying; concurrent elevations in calcium and phosphorus are common in these individuals.

Gastrointestinal tract

Gastrointestinal disease typically only gives nonspecific results with clinical biochemistry. Elevations of CK, AST and LDH are not uncommon, and are not specific to the intestinal tract. Electrolytes may give more information and should be evaluated with an understanding of the patient's appetite and thirst, hydration status, previous or current therapy and pathologic processes (i.e., gastrointestinal or renal disease) which may alter electrolyte concentrations.

- Sodium must be interpreted with the knowledge of the patient's hydration status. It may be elevated with decreased water intake or dehydration through renal disease, vomiting or diarrhoea. Sodium may also be lost through the gastrointestinal tract or the kidneys. Other causes of hyponatraemia include over-hydration, end-stage liver disease and congestive heart failure.

- Chloride is interpreted alongside sodium. It may be elevated with vomiting or regurgitation, although this is uncommon; low levels are usually associated with regurgitation or vomiting, renal disease congestive heart failure and other conditions which cause water retention.

- Potassium may be decreased with vomiting/diarrhoea and elevated with dehydration, haemolysis, tissue damage, or poor sample handling.

There are many other possible causes of electrolyte disturbance, and our understanding of avian electrolyte balance is still in the very early stages.

Amylase and lipase have been proposed as useful parameters in the detection of pancreatic disease in birds. It is of little value in rabbits and its significance in reptiles is unknown. There is still considerable discussion of the incidence of pancreatic disease and the specificity of these enzymes. Significant elevations of these enzymes, when accompanied by clinical signs of gastrointestinal dysfunction (vomiting, ileus, diarrhoea, coelomic pain) should lead the clinician to consider pancreatic disease as a differential diagnosis. However, normal levels do not preclude a diagnosis of pancreatic disease, nor do abnormal levels confirm such a diagnosis.

Blood glucose

Glucose is an essential energy source for nearly every cell in the body. Blood levels are governed by its intake, absorption, the interactions of hormones controlling carbohydrate metabolism (insulin, glucagon and somatostatin), the body's metabolism, its ability to store glucose and its excretion. As disorders of glucose metabolism involve so many organ systems, it is treated here as a separate entity.

Hyperglycaemia may be a normal physiological process, (e.g. in juvenile birds). However, elevated levels are usually related to increased production or release (e.g. stress) or failure of tissues to take it up out of the blood (diabetes mellitus). Iatrogenic hyperglycaemia occurs when corticosteroids are administered or intravenous dextrose is given. Female reproductive disease may also elevate blood glucose, but this may be an indirect result due to inflammation affecting the endocrine pancreas.

Hypoglycaemia may result from poor handling of blood samples (i.e. artefactual rather than factual), or with decreased food intake (starvation, anorexia), increased glucose usage (septicaemias, neoplasia and multi-organ failure) or decreased production (liver disease). Reptiles normally have lower blood glucose levels than birds and mammals, reflecting their low metabolic rate. Glucose concentrations in reptiles are highly variable, but generally range between 2.0-10.0 mmol/L. Lower concentrations are more commonly noted in large snakes, while higher concentrations are common in animals under stress. Diabetes mellitus is rare in reptiles.

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INTEGRATIVE MEDICINE (LECTURES GIVEN IN MANDARIN CHINESE)

NUTRACEUTICALS IN ARTHRITIS: WHAT DO WE KNOW?

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Introduction

Degenerative joint disease (DJD) also known as osteoarthritis (OA), is a chronic, debilitating disorder affecting a wide range of animal species and humans. It has been reported that OA may affect up to 20% of the canine population over one year of age, and likely a similar percentage of cats (and has increased in incidence 38% in dogs and 67% in cats since 2007 according to data from Banfield Pet Hospitals). It is estimated that more than 11 million dogs, or 1 of out 5 dogs, in the US alone, was affected by OA. It is regarded as the number 1 cause of chronic pain in dogs. Unlike in humans where the DJD is primarily as a result of aging and "wear and tear", DJD in dogs and cats is typically secondary, initiated by a previous insult to the joint or joint instability. It is a common cause of joint failure with stiffness, loss of mobility, and varying degrees of inflammation and pain. It is a slowly progressive condition for which no specific treatment can restore the articular cartilage to normal. Therefore, early identification of DJD is critical to provide patients with the most effective treatment strategies and relief of clinical signs.

What is cartilage?

Cartilage is made up of three main components:

1) Chondrocytes, which by weight comprise the smallest proportion of the cartilage;

2) Extracellular matrix (ECM), made up of type II collagen fibers and proteoglycans (PG), which serve to trap water within the matrix;

3) Water, which makes up 70% of the cartilage by weight and gives cartilage it's unique biomechanical properties.

Chondrocytes function to synthesize the components of the ECM and also produce the enzymes that degrade the ECM, thus regulating cartilage anabolism and catabolism. Various cytokines and growth factors balance these anabolic and catabolic activities in healthy cartilage. In a joint affected by DJD, catabolic processes outweigh the anabolic processes. This imbalance is driven by cytokine cascades and inflammatory mediators, such as interleukin-1ß (IL-1ß) and tumor necrosis factor-α (TNF-α). It results in reduced proteoglycan concentration, altered aggregation of the PGs and ultimately, reduced ability of the ECM to retain water. Collagen fibers also become disrupted. These cytokines and other cytokines (IL-8, IL-6, etc.) also upregulate the expression of proinflammatory enzymes cyclo-oxygenase-2 (COX-2) and inducible nitric oxide synthase (iNOS) that lead to increased production of prostaglandin E2 (PGE2) and nitric oxide (NO).

Although we tend to focus on the articular cartilage when discussing OA, it is important to remember that OA is a global process within the joint, affecting not only the hyaline cartilage, but the synovial membrane, synovial fluid, and subchondral bone as well. Clinical effects in patients suffering from OA include joint instability, joint mal-alignment, joint pain and diminished range of motion, joint effusion, and varying degrees of lameness and immobility. The patient as whole begins to lose muscle strength and cardiovascular fitness while gaining weight. This catabolic process takes on a "snowball effect" that affects the family interactions with the pet and becomes difficult to reverse. It is important to remember that once patients begin showing these signs, the disease process is typically quite advanced.

Recent years have seen a dramatic shift toward multimodal management strategies for OA management, including anti-inflammatory medications, analgesics, therapeutic modalities, therapeutic exercise, and nutraceuticals or so called "disease modifying osteoarthritis agents" (DMOAs). The potential benefits of the majority of these recommendations are readily recognized.

Where's The Evidence For Nutraceuticals?

A. Adequan (Novartis)

Adequan is probably the most well-known and frequently used DMOA and is chemically referred to as a polysulfated glycosaminoglycan (PSGAG). It is typically given intramuscularly (IM) and studies have shown that it does reach therapeutic levels in serum, synovial fluid, cartilage and tendons. It modifies the progression of OA by maintaining a more normal cartilage histology through stimulation of increased glycosaminoglycan (GAG) synthesis by chondrocytes and inhibition of destructive enzymes such as MMPs. Though experimental evidence of Adequan's positive anabolic effect on cartilage is conflicting, its ability to decrease cartilage catabolism has been shown in numerous studies on both horses and dogs. Adequan also has a poorly understood anti-inflammatory effect. Ultimately, the PSGAG allows the cartilage to hold more water and makes it more resistant to degradation. For optimal results, Adequan should probably be used earlier in the disease process than it has typically been employed in the past. In dogs, a dosage of 5 mg/kg intramuscularly (IM) twice weekly is commonly recommended.

B. Glucosamine−Chondroitin Combination:

Glucosamine is an amino monosaccharide unit of glycosaminoglycan, which is the building block of the cartilage matrix seen within joints. Its mechanism of action may involve inhibition of inflammatory enzyme activity and stimulation of glycosaminoglycans (GAG) synthesis. Chondroitin sulfate is a long-chain polymer of repeating disaccharide units containing galactosamine sulfate and glucuronic acid and constitutes the majority of glycosaminoglycans in cartilage found on the moving surfaces of joints, or articular cartilage. Its bioavailability is well documented with up to 70% absorption after oral administration in animals and humans. Its mechanism of action including contributing to a pool of substrate available for cartilage matrix deposition, inhibition of proteases, stimulation of glycosaminoglycan, and collagen synthesis. When given in combination, glucosamine and chondroitin sulfate reportedly support cartilage production and protect existing cartilage by inhibiting enzymes in the joints that break down cartilage. In one study treated canine OA with a combination of glucosamine and chondroitin sulfate showed a significant anti-inflammatory effect against chemically induced inflammatory synovitis in dogs. [canapp]

CANAPP SO, MCLAUGHLIN RM, HOSKINSON JJ, ET AL: SCINTIGRAPHIC EVALUATION OF DOGS WITH ACUTE SYNOVITIS AFTER TREATMENT WITH GLUCOSAMINE HYDROCHOLORIDE AND CHONDROITIN SULFATE. J VET RES. 60:1552-1557, 1999.

C. Avocado Soybean Unsaponifiables (ASU)

The mechanism of action of ASU include suppressing TNF-α, IL-1β, COX-2, iNOS gene expression, and prostaglandin E2 and nitric oxide production in articular chondrocytes and monocyte/macrophages.3 In a canine study, ASU caused an increase in both TGF-ß isoforms as compared with the control group. TGF-ß is stimulator of extracellular matrix production (type II collagen, proteoglycan) in chondrocytes.4 In another canine study, ASU supplemented group showed reduced development of early OA cartilage and subchondral bone lesions (macroscopic and microscopic lesion severity, subchondral bone loss) as compared to placebo treated controls.5

A recent study evaluated the effect ASU, CS and ASU + CS combination upon proinflammatory cytokine (IL-1ß, TNF-α) expression and PGE2production from synovial lining surrogate cells.7 The ASU + CS combination inhibited IL-1ß and TNF-α expression and PGE2 production better than either agent alone.7

A similar effect was noted in activated feline chondrocytes when ASU+CS+Glu pre-treated cells were compared to positive (untreated activated cells) and negative (non-activated cells) controls.9

3. AU RY, AL-TALIB TK, AU AY, ET AL. AVOCADO SOYBEAN UNSUPONIFIABLES (ASU) SUPPRESS TNF-A, IL-1SS, COX-2, INOS GENE EXPRESSION, AND PROSTAGLANDIN E2 AND NITRIC OXIDE PRODUCTION IN ARTICULAR CHONDROCYTES AND MONOCYTE/ MACROPHAGES. OSTEARTHRITIS & CARTILAGE. 2007,15:1249-1255

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7. GRZANNA MW, OWNBY SL, HEINECKE LF, ET AL. INHIBITION OF CYTOKINE EXPRESSION AND PROSTAGLANDIN E2 PRODUCTION IN MONOCYTE/ MACROPHAGE-LIKE CELLS BY AVOCADO/SOYBEAN UNSAPONIFIABLES AND CHONDROITIN SULFATE. J COMPLEMENT INTEGR MED. 2010,7(1):ART10. (DOI:10.2202/1553-3840.1338).

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Epigallocatechin Gallate

Recently, a study was conducted to evaluate the anti-inflammatory effect of ASU+epigallocatechin gallate (EGCG) on activated equine chondrocyte cell culture.10 EGCG is a major antioxidant component of green tea and has been reported to inhibit the onset and severity of induced arthritis in mice. Chondrocyte activation caused upregulated gene expression of COX-2 and increased PGE2 production and NF-kB nuclear translocation. Individually, ASU and EGCG marginally inhibited COX-2 expression and PGE2 production in activated chondrocytes. In contrast, ASU+EGCG combination reduced COX-2 expression close to that of non-activated control levels, significantly inhibited PGE2 production and NF-κB translocation. NF-κB is an essential transcription factor for COX-2 induction. Inhibition of the NF-κB pathway is known to attenuate COX-2 expression. This study demonstrates that the anti-inflammatory activity of ASU and EGCG is potentiated when used in combination.10 Collectively, a large body of evidence supports the conclusion that ASU+CS+Glu inhibits the expression and production of proinflammatory mediators

in multiple species (canine, feline, equine and human) and in multiple joint cell lines (chondrocytes and synovial lining cells). Millis et al have evaluated 8 hound dogs with chronic induced stifle OA. A recent study arm on this dog colony evaluated Dasuquin (Nutramax Labs), a nutraceutical containing Glu + CS + ASU + EGCG. Dogs treated with Dasuquin showed increased peak vertical force similar to that seen with various nonsteroidal anti-inflammatory drugs (NSAIDs) in a previous study arm.11 Although the small study population does not permit meaningful statistical analysis, these data suggest the need for larger scale and longer term evaluation of Dasuquin in the restoration of function and pain relief of dogs with chronic OA.

10. HEINECKE LF, GRZANNA MW. AU AY, ET AL. INHIBITION OF CYCLOOXYGENASE-2 EXPRESSION AND PROSTAGLANDIN E2 PRODUCTION IN CHONDROCYTES BY AVOCADO SOYBEAN UNSAPONIFIABLES AND EPIGALLOCATECHIN GALLATE. OSTEOARTHRITIS & CARTILAGE. 2010,18:220-227.

11. MILLIS DL. UNPUBLISHED DATA, 2011.

D. Omega 3 Fatty Acids (docosahexaenoic acid, or DHA, and eicosapentaenoic acid, or EPA)

The mechanism of action of omega-3 fatty acid is to inhibit the conversion of Arachidonic acid (AA). AA is an omega-6 fatty acid producing prostaglandins, leukotrienes, and thromboxanes. These eicosanoids have predominantly vasoactive and proinflammatory effects. Inhibiting the conversion of AA to these eicosanoids is the principal action of most NSAIDs used to treat OA. Eicosanoids produced from EPA and DHA are not potent inflammatory mediators, in contrast to those produced from AA. In a study of dietary supplementation with fish oil omega-3 fatty acids in osteoarthritic dogs showed a significant improvement in weight-bearing (peak vertical force- PVF, improved 82%) as compared with the control-group (38%).12 The magnitude of improved weight-bearing (+ 5.6%) was of similar magnitude reported in many NSAID trials. A similar study alos showed that food supplemented with fish oil omega-3 fatty acids improved the owner-assessed ability for the ability to rise from a resting position, play, and walk compared with control dogs (whom showed no improvement).13 Another study showed that a diet supplemented with omega-3 fatty acid allowed for more rapid reduction in carprofen dosage in osteoarthritic dogs as compared to dogs fed a diet with a low omega-3 fatty acid content.14

12. ROUSH JK, CROSS AR, RENBERG WC, ET AL. EVALUATION OF THE EFFECTS OF DIETARY SUPPLEMENTATION WITH FISH OIL OMEGA-3 FATTY ACIDS ON WEIGHT BEARING IN DOGS WITH OSTEOARTHRITIS. JAVMA. 2010,236:67-73.

13. ROUSH JK, DODD CE, FRITSCH DA, ET AL. MULTICENTER VETERINARY PRACTICE ASSESSMENT OF THE EFFECTS OF OMEGA-3 FATTY ACIDS ON OSTEOARTHRITIS IN DOGS. JAVMA. 2010,236:59-66.

14. FRITSCH DA, ALLEN TA, DODD CE, ET AL. A MULTICENTER STUDY OF THE EFFECT OF DIETARY SUPPLEMENTATION WITH FISH OIL OMEGA-3 FATTY ACIDS ON CARPROFEN DOSAGE IN DOGS WITH OSTEOARTHRITIS. JAVMA. 2010,236:535-539.

E. Green-Lipped Mussel (Perna Canaliculus)

Green-Lipped Mussel (GLM) is a shellfish from New Zealand that obtains nutrients directly from the phyto-plankton and minerals in sea water. In all, there have been identified at least 50 nutrients in the GLM, including complex proteins, amino acids, nucleic acids, naturally chelated minerals, enzymes, vitamins, glycosaminoglycans, and fatty acids. GLM has been shown to contain a unique omega-3 fatty acid, eicosatetraenoic acid (ETA), which appears to act as dual inhibitor of arachidonic acid oxygenation by both the cyclo-oxygenase (COX) and lipoxygenase pathways. In a study, the GLM-enriched diet modified gait in dogs with OA in that the peak vertical force significantly increased over the 60-d period when GLM was introduced into a standardized control diet. [Rialland] Another study showed that GLM alleviated chronic orthopedic pain (veterinary-assessed mobility, owner-evaluated chronic pain index and pain VAS) in dogs compared to control group. [Hielm] Another study showed that GLM alleviated chronic orthopedic pain (veterinary-assessed mobility, owner-evaluated chronic pain index and pain VAS) in dogs compared to control group. [Hielm]

Rialland P, Bichot S, Lussier B, et al. Effect of a diet enriched with green-lipped mussel on pain behavior and functioning in dogs with clinical osteoarthritis. Canadian Journal of Veterinary Research. 2013;77(1):66-74.

Hielm-Björkman A, Tulamo R-M, Salonen H, Raekallio M. Evaluating Complementary Therapies for Canine Osteoarthritis Part I: Green-lipped Mussel (Perna canaliculus). Evidence-based Complementary and Alternative Medicine: eCAM. 2009;6(3):365-373. doi:10.1093/ecam/nem136.

Curcumin

Curcumin is a polyphenol extract from the yellow spice, turmeric. Curcumin has been used for thousands of years in traditional Chinese and Ayurvedic medicine. Curcumin and various curcuminoid extracts have been studied extensively both in vitro and in vivo. In vitro studies have shown anti-inflammatory and anti-neoplastic effects including decreases in inflammatory cytokines (IL-6, IL-8, COX-2, PGE2, iNOS, MMP-3, MMP-9) and inhibition of NF-kB and TNF-α signaling. The major challenge with curcumin as an oral supplement is its extremely poor

bioavailability (<1%). A recent in vivo study in dogs with OA showed decreased gene expression for inflammatory mediators in dogs receiving curcumin compared to NSAIDs. In this study, the bioavailability of curcumin was enhanced with a phytosome delivery (curcumin combined with phospholipids).

Boswellia serrata

Boswellia serrata is a large tree that grows in India, North Africa, and the Middle East. The resin of this tree, and other members of the Boswellia species, also known as Indian frankincense and has been used for centuries to support joint health. One of the resin extracts, AKBA (3-O-acetyl-11-keto-beta boswellic acid), appears to have the most potent anti-inflammatory effects and has been shown to support structural integrity of joints and connective tissues. In 2003, a randomized, double-blind, placebo-controlled study assessed the efficacy, safety, and tolerability of Boswellia serrata in human osteoarthritis. All patients in the group receiving the active drug reported increased knee flexion, increased walking distance, improvement in capacity to climb stairs and better kneeling, crossed-legged sitting, and squatting ability. In vitro studies have shown significant immunomodulatory and inflammation-modulating effects of Boswellia serrata. Possible modes of action for AKBA include inhibition of the inflammatory mediator 5-lipoxygen-ase, cytokines (interleukins and TNF-α) and the complement system, as well as inhibition of NF-kB. In a prospective open multicenter study in dogs, statistically significant improvement was noted in lameness and pain in 71% of dogs. In a randomized, placebo controlled trial in naturally occurring OA in dogs, peak vertical force and daily activity (measured via accelerometers) was in improved compared to baseline in groups receiving a supplement containing Boswellia.

Astaxanthin

This ingredient is naturally found in reddish-colored marine algae (Haematococcus pluvialis). It belongs to a group of compounds known as oxygenated carotenoids. Astaxanthin has potent antioxidant and anti-inflammatory properties. It scavenges free radicals and decreases the development of nitric oxide. It is one of the most powerful anti-oxidants in nature. As comparison it is 6000 times stronger than Vitamin C, 800X stronger than CoQ10, 550X stronger than Vitamin E, 75X stronger than α- Lipoic Acid, and 36X stronger than ß-Carotene. Astaxanthin reduces and prevents damage from free radicals by donating an electron. This will neutralize the free radical without developing another. In a study, dogs that were fed astaxanthin showed enhanced immune response. Another prospective study in dogs showed astaxanthin alleviates age-related oxidative and inflammatory damage and enhances mitochondrial function. This effect was greater in geriatric than young dogs. Also, in a study were exercise-conditioned dogs

were fed supplemental astaxanthin, they demonstrated increased plasma triglyceride levels pre-exercise and prevention of exercise-induced decreases in plasma glucose concentrations compared to controls. This shows astaxanthin may mitigate exercise-induced fatigue and improve exercise performance.

F. Velvet Antler

The underlying mechanisms of velvet antler (VA) remain poorly understood. Molecules identified as having potentially important local roles in antlers include parathyroid hormone–related peptide and retinoic acid (RA). Both are present in the blastema and in the rapidly growing antler where they regulate the differentiation of fibroblasts, chondrocytes, osteoblasts, and osteoclasts in vitro. VA powder was evaluated on client-owned dogs with osteoarthrosis in a clinical double-blind, and placebo-controlled study. Gait analysis measured with a force plate, clinical signs assessed by an orthopedic surgeon, performances in daily life activities and vitality assessed by the owners, and complete blood analyses were obtained. Gait, performance in daily life activities, and vitality were significantly improved on VA. No clinical changes were revealed on blood analyses.

Moreau M, Dupuis J, Bonneau NH, Lécuyer M. Clinical evaluation of a powder of quality elk velvet antler for the treatment of osteoarthrosis in dogs. The Canadian Veterinary Journal. 2004;45(2):133-139.

G. Zeel

Zeel inhibit the metalloproteinases (MMPs), which are enzymes such as hyaluronidase. It also has inhibitor effects on the production of Leukotriene B4 by 5-lipoxygenase (5-LOX) and on the synthesis of prostaglandin PGE2 by COX-1 and 2 enzymes. In a study, dogs with moderate to severe OA receiving the HCP Zeel for 8 weeks had significantly less pain than their placebo peers.

H. Eggshell Membrane

Eggshell membrane (EM) is the thin, proteinaceous layer between the raw eggshell and the egg white is made primarily of proteins (>88%) and naturally rich in elastin, collagen and glycosaminoglycans (GAGs). In vitro study has shown that eggshell membrane suppresses the release of TNF-α from stimulated peripheral blood mononuclear cells. A recent prospective, randomized, double-blind, placebo-controlled study showed supplementation with EM, ~13.5 mg/kg (6 mg/lb) taken once daily, significantly reduced joint pain and improved joint function rapidly (CBPI 1 week) and demonstrated a lasting improvement in joint pain (VCSA 6 weeks) leading to an improved quality of life (CBPI 6 weeks). Moreover, serum CTX-II (type II collagen) levels in EM-supplemented dogs was significantly improved versus

placebo at 6 weeks.

Ruff, Kevin & Kopp, Kenneth & Von Behrens, Pamela & Lux, Mark & Mahn, Matthew & Back, Matthew. (2016). Effectiveness of NEM® brand eggshell membrane in the treatment of suboptimal joint function in dogs: a multicenter, randomized, double-blind, placebo-controlled study. Veterinary Medicine: Research and Reports. Volume 7. 113-121.

Vitamin D3

Vitamin D3 is important for supporting healthy bone structure. Unlike humans, VitD3 in dogs is primarily absorbed from dietary sources with negligible absorption from the sun. After oral ingestion, VitD3 is converted in the liver to 25- hydroxyvitamin D and then further hydroxylated in the kidney to its biologically active form 1,25 dihydroxyvitamin D (or calcitriol). In recent studies, a multitude of chronic illnesses have been associated with VitD3 deficiencies in people.

In addition, an observational study in people showed that participants with low intake of dietary vitamin D and lower serum levels of vitamin D were approximately 3 times more likely to exhibit progression of established knee osteoarthritis than those with higher levels. Another study noted that vitamin D may be related to the processes that impede or give rise to locomotor conditions and could play a major role in modulating oxidative stress, participating in immune responses, and contributing to cell differentiation.

Summary

There is a strong and compelling body of evidence supporting the role of nutraceuticals in joint health in veterinary practice. The final question is "how confident are you that the product you are recommending contains what the label says that it does?" A study of randomly selected nutraceuticals showed that 84% of the products tested did not meet their label claims with contents ranging as low as 0% of the claimed content in some products. Contamination with unwanted ingredients is also a problem within this relatively unregulated industry. For a small fee, Consumer Labs (consumerlab.com) provides independent laboratory testing of various products contents and purity.

The use of joint nutraceuticals in dogs prior to the development of OA is controversial. No controlled studies have been reported that document the efficacy of nutraceuticals in preventing the development of OA. However, because of their reported effects on improving cartilage matrix and reducing levels of inflammatory mediators within the joint, many clinicians have advocated the prophylactic use of joint nutraceuticals, particularly in athletic dogs that might be susceptible to joint injury. Additional research is needed to confirm the value of prophylactic use of joint nutraceuticals.

SUMMARY:

Joint nutraceuticals have been shown, through in vitro studies and controlled clinical trials, to be useful in the treatment of OA. They can be used for long-term management of patients with all stages of OA and carry minimal risk. The use of joint nutraceuticals can significantly reduce the need for NSAIDS and other medications in patients with OA.

Amongst all the appealing supplements that are purported to have a 'protective' effect on the development of OA, nothing has been proven to have the degree of protection as calorie restriction has. Although glucosamine and chondroitin sulfate have been found to have a mild analgesic effect in moderate-to-severe pain patients,[10,11] the same study found no significant effect of these supplements on disease progression over a 2-year period,[12] whereas other studies have found a small beneficial effect of glucosamine sulfate on disease progression.[13] Indeed, there seems to be some evidence of a small structure modifying effect of glucosamine sulfate (particularly in the mild OA cases) in a few human clinical studies.[14,15]

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43RD WORLD SMALL ANIMAL VETERINARY ASSOCIATION CONGRESS AND 9TH FASAVA CONGRESS

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NURADSE (HILLS)

NURSES (HILLS)

THE INS AND OUTS OF ANESTHETIC MONITORING FOR OPTIMUM PATIENT OUTCOMES

J. Hatcher[1]

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CARDIOPULMONARY RESSUCITATION PRACTICAL FOR NURSES

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When to Resuscitate

Many factors need to be considered when deciding if to proceed with resuscitation of the patient depending on the patient's condition, financial concerns, post CPR care etc.

- What is the potential outcome of CPR? – does the patient have such extensive injuries that chance of full recovery from them is already compromised?
- What have you agreed with the owner – we must honour their decisions, whether we feel they are correct or not.
- Does the patient have a treatable disease? – if we resusitate the patient – can we treat / cure the underlying disease / problem?
- Is the patient in the terminal stages of an incurable disease? – e.g. cancer, renal failure etc
- We must consider if we will be able to restore a near-normal mentation.
- When did the arrest occur – have we suffered brain damage due to prolonged hypoxia etc. we can get a heart back but we cannot resolve brain damage from hypoxia.

Your clinic may have a classification system on when or not to resuscitate. As part of the Recover guidelines the following system was derived:

Class	Risk/Benefit Ratio	Recommendation
1	High Benefit, low risk	Should be performed
2	Medium benefit, medium risk	Reasonable to perform
3	Benefit and risk equal	May be considered
4	High risk, low benefit	Should not be performed

Vital Signs

Vital signs are, as they are called "vital" in assessing a patient's condition and status. All staff must know the normal ranges for the species they are treating.

	Dog	Cat
Heart rate:	80 – 140 bpm	110 – 180 bpm
Pulse:	Strong & rhythmic	Strong & rythmic
Respiration rate:	10 – 30 bpm	15 – 40 bpm
SpO2:	98 – 100%	98 – 100%
ETCO2:	35 – 45 mm Hg	35 – 45 mm H
Temperature:	37.5 – 38.5C	38 – 38.5C
Mucous Membranes:	Pink & Moist	Pink & Moist
CRT:	1 – 2 seconds	1 – 2 seconds

Order of Priority & Team Roles

The patient has arrived, a quick visual assessment has been completed and the patient is in a critical condition. What do you do now and in what order?

Firstly establish if the patient is in just pulmonary arrest (not breathing) or cardiopulmonary arrest (not breathing and no heartbeat).

Use the acronym CAB:

C – Circulation – No heartbeat, audible heartbeat or pulse then start compressions

A – Airway – Check for a patent airway and provide oxygen supplementation or intubate

B – Breathing – Start Intermittent Positive Pressure Ventilation

Circulation is the most important priority when starting CPR. It has been shown that there will be potentially enough residual oxygen in the body for up to 3 minutes post arrest but in a cardiac arrest that oxygen is not going anywhere so we need to start compressions immediately to help with circulation. Then ensure a patent airway and intubate to provide oxygen supplementation or start Intermittent Positive Pressure Ventilation (IPPV). Compressions should NEVER be stopped for intubation.

If you are in the unfortunate circumstance of being the first responder to a patient in cardiac arrest and do not have any assistance the first priority is to start chest compressions. If after 3 minutes you are still the only responder then stop compressions to intubate and breathe for the patient in between compressions. 2 breaths to every 30 compressions.

All staff available in the hospital should assist with CPR. Often I have seen Vets or Nurses come in to the room and see ventilation and compressions occurring and someone getting drugs and they think there is nothing for them to do. Even if you aren't taking an active role in performing CPR, recording all the actions taken such as start time of CPR and timing the 2 minute cycles along with all observations and drugs administered is very important. Roles that each team member can perform are:

Compressions – This is performed in 2 minute cycles. It is important that this person swaps out this role to another team member after each cycle as performing compressions is tiring and compressions will not be effective after this time.

Airway and ventilation – This person could swap with the team member performing compressions and vice versa.

Circulation - Placing an IV Catheter and collecting and administering drugs and fluids

Monitoring - Attaching monitoring equipment and

performing observations and monitoring vital signs. Communicate what you are observing and how effective is the CPR being performed.

Recording - Recording all actions, drugs administered with doses and time, time CPR was started and time the 2 minute cycles of CPR.

Compressions

Cardiac arrest will shortly follow respiratory arrest often if not occurring at the same time. Cardiopulmonary arrest occurs when cardiopulmonary function fails. The aim of cardiopulmonary resuscitation is to restore cardiac function and circulation. This is done by creating adequate pressure in the thoracic cavity by compressing the chest wall to stimulate cardiac output and create adequate venous return. There are two ways of performing cardiopulmonary resuscitation, external cardiac massage and internal cardiac massage.

External cardiac massage

There are two methods of external cardiac massage depending on the size of the animal.

Patient Positioning – Experimental evidence suggests higher left ventricular pressures and aortic blood flow in dogs in lateral recumbency compared to dorsal recumbency and higher rates of return of spontaneous circulation in compressions performed in lateral recumbency suggest that this is the ideal position. Either left or right is now considered acceptable although right lateral is preferred. However there are great variations in chest conformation among dogs and cats so a single identical approach to compressions is unlikely to work.

Cardiac Pump (Patients under 20kg)

The cardiac ventricles are directly compressed between the sternum and spine in patients in dorsal recumbency and between the ribs in lateral recumbency. This method is most suitable for patients weighing less than 20 kg. The patient is placed in lateral recumbency and the chest wall is compressed by placing both hands on the upper side of the chest wall at a rate of 100 - 120 compressions per minute. The chest should be compressed to 30 – 50% of its circumference. In very small patients such as cats the chest can be compressed between the thumb and forefinger with the thumb being on the upper side of the chest wall. Ventilation is given simultaneously with compression at 1 breath every 5 seconds. Continue the cycle for 2 minutes without interruption.

Thoracic Pump (Over 20 kg)

Chest compressions increase the overall intrathoracic pressure, secondly compressing the aorta and collapsing the vena cava leading to blood flow out of the thorax. During the elastic recoil of the chest, sub atmospheric intrathoracic pressure provides a pressure gradient that favours the flow of blood form the periphery back into the thorax and into the lungs where oxygen and carbon dioxide exchange occurs.

This method is most suitable for patents weighing over 20 kg or medium, large or giant breeds with round chests. The patient can be in lateral or dorsal recumbency. The chest is compressed to 30 – 50% of its circumference by placing both hands on the widest point of the chest. Ventilation is simultaneously supplied to increase the thoracic pressure also at 1 breath every 5 seconds. Continue the cycle for 2 minutes without interruption.

Abdominal counter compressions

Abdominal counter compressions can help with venous return and improve cardiac output. By applying abdominal compressions alternate to chest compressions the blood is forced to the chest cavity for more effective cardiopulmonary resuscitation. If abdominal compressions are not possible then binding the hind limbs and abdomen with bandages (Vetwrap) helps to create the same effect. There have been some reports of injury to abdominal organs in some cases when counter compressions are applied. Therefore this is often used as a last resort method.

Internal Cardiac Massage

This method is not common as it involves opening the chest and hence a surgical approach. Clip the hair on the chest between the sternum and chostochondral junction and apply a quick surgical scrub. The veterinarian will make an incision between the 4th and 5th ribs and spread them apart with retractors while delicately moving the lungs. The pericardium is then grasped and the heart compressed with a thumb and forefinger. It is important that IPPV is maintained throughout this process.

Airway

LOOK AT THE ANIMAL! If the respiration rate is slow and the mucous membrane colour is pale to grey then respiratory arrest is often not too far off. Agonal gasps are generally a clear indicator of cardiac arrest or imminent arrest. Ensure a patent airway. Wipe or suction away any debris in the mouth and airway such as blood, vomitus or foreign objects.

Breathing

If the animal is in actual respiratory arrest (not breathing at all) then an appropriate sized endotracheal tube must be placed down the trachea and the cuff inflated. Attach the tube to an oxygen source which ideally would be an Ambu bag but the anaesthetic machine is adequate however ensure the vaporiser is turned OFF and the circuit closed. Commence Intermittent Positive Pressure Ventilation (IPPV). One breath every 5 seconds at a tidal volume of 10ml/kg. If you can monitor ETCO2 with a capnograph a measurement of at least 15 mm Hg will

give an indication that ventilation provided is adequate.

CPR Cycle

An uninterrupted cycle of Basic Life Support lasting 2 minutes in intubated patients is recommended before checking for vital signs. If mouth to snout 2 minute cycle however 30 chest compressions with brief interruption to allow 2 quick breaths. Rotate personal doing chest compressions after each 2 minute cycle as it is very tiring and your compressions will not be effective after this time. If you feel you are tiring before the two minutes is up speak up and swap with another team member immediately.

ECG

An ECG being placed is often classed as Advanced life support but it will tell us about the electrical activity of the heart in particular if we have Ventricular Fibrillation occurring.

PQRST Complex

The PQRST complex can tell us what is happening within the heart.

P wave indicates atrial depolarisation (atrial contraction)

QRS wave indicates ventricular depolarisation (ventricles contracting)

T wave indicates repolarisation (heart relaxed)

Defibrillation

Ventricular Fibrillation is an irregular quivering motion of the ventricles caused by continuous disorganised electrical activity in the heart. An ECG trace will show no QRS complexes. Without co-ordinated contractions the blood is not propelled forward. The idea of a defibrillator is to depolarize the myocardial cells in the ventricles or to shock them into their refractory period and allow the pacemaker of the heart to start a normal sinus rhythm. If a defibrillator is not available a pre-cordial thump on the chest over the heart can be used although this is likely to not be very effective.

Conclusion

We only have 3 – 5 minutes to restore cerebral and coronary perfusion so we must act quickly but not panic. If you understand your role, work well as part of a team who practices CPR regularly and perform the necessary steps of CPR the patient has a greater chance at a positive outcome.

To access the Recover CPR Guidelines go to http://onlinelibrary.wiley.com/doi/10.1111/vec.2012.22.issue-s1/issuetoc

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WSV18-0235

ROLE AND USE OF KETAMINE IN PERIOPERATIVE PAIN CONTROL

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NMDA Antagonists: Ketamine and Tiletamine (telazol: Dissociative plus benzodiazapine)

NMDA antagonism- receptor associated with learning, memory and neuroplasticity.

As an anesthetic drug, some areas of the CNS are depressed (neocorticothalamic) while others are stimulated (limbic). This gives it variable effects in disease states of the brain.

Increased muscle tone

Spontaneous movement

Open eyelids with a brisk palpebral

Excessive salivation

Seizurogenic (in some types of epilepsy- protective in others)

- Clinical signs of anesthesia are much different than anesthetics that act as depressants.
- The typical gradations of surgical anesthesia are non-existent with ketamine as a single anesthetic (plane 1-4). If the patient is overtly moving during surgery, it is too light, if apneic or convulsing, it is too deep, otherwise, anesthesia is probably adequate (thus it is not used alone, much). It is also not directly analgesic (anti-hyperalgesic), so is not recommended as a sole anesthetic drug.

Metabolism: Liver biotransformation, forming partially active metabolite nor-ketamine, which undergoes conjugation prior to renal excretion. Conjugation is limited in cats which are therefore more dependent on renal excretion of nor-ketamine for recovery.

Systemic effects

CNS: Seizurogenic: commonly use adjunctive drugs, do not use in patients susceptible to seizures (epileptics, myelograms, cranial trauma) Increases cerebral blood flow (CBF), oxygen requirement, and intracranial pressure. Caution with use as an induction drug with CNS disease. HOWEVER, as knowledge about the neuro-protective effects of NMDA antagonists has grown, it has become commonplace to use ketamine as a CRI (at analgesic and mildly supra-analgesic doses) for craniotomy, TBI, spinal cord injuries, and other injuries to nervous system tissues.

Revising a Dogma: Ketamine for Patients with Neurological Injury? Sabine Himmelseher, MD*, and Marcel E. Durieux, MD, PhD (Anesth Analg 2005;101:524–34)

Analgesia: ketamine is a potent anti-hyperalgesic at sub-anesthetic dosages. It also acts as an inhibitor of amplification of pain via the NMDA receptor, and inhibitor of neuro-inflammation and glial. Ketamine is a poor direct analgesic, especially for visceral sources of pain.

Cardiovascular: Increased heart rate, blood pressure and cardiac output: centrally mediated (sympathetic tone). The DIRECT effect of ketamine on the myocardium is depression. Thus, these increases are NOT seen with pre-existing tachycardia, sympathetic overload, severe sickness, or denervated heart. When stimulation is seen, there will be increased left ventricular work, with increased oxygen requirement. Therefore, beware if oxygenation or myocardial bloodflow are compromised, or in heart conditions where myocardial hypoxia is likely (HCM)

Respiratory: mild respiratory depressant at clinical doses. Apneustic pattern is common. Laryngeal functionis maintained to a degree: patients may still swallow, so there may be some retention of function, but the coordination is impaired and aspiration is usually not prevented.

Temperature: ketamine alone raises body temperature, due to mm rigidity (but ketamine is seldom used alone). As a component of general anesthesia, ketamine will not guard against environmental hypothermia.

Regulatory Update: Human abuse potential has created strong international pressure (China) to increase regulatory control. The commission on narcotic drugs elected NOT to upgrade the regulation of ketamine to schedule 1 in 2015, but there is still international debate. For more information see the fact sheet that was endorsed by a large number of human and veterinary organizations (in which the WSAVA played an extremely active role) regarding this issue: https://www.asahq.org/~/media/sites/gho/ketamine-fact-sheet-2015

Important consideration, however, is the human abuse potential of opioids, and the opioid sparing, addiction-mitigating effects of NMDA antagonist drugs.

Le Cornec, Lariby Brenckmann, et al. Is intravenously administered, subdissociative-dose KETAmine non-inferior to MORPHine for prehospital analgesia (the KETAMORPH study): study protocol for a randomized controlled trial Trials. 2018 May 2;19(1):260. doi: 10.1186/s13063-018-2634-3.

Usage update: Ketamine has uses that bridge: anesthetic (inexpensive and widely available in first through third-world countries), amnesiac, cardiovascular sparing in intensive care, cerebro-protective, anti-glial activation, neuropathic pain and depression suppressing.

The Global Pain Council of the WSAVA utilizes ketamine extensively in the recommendations for countries where opioid analgesics, and safe non-steroidal analgesics are limited.

www.wsava.org/guidelines/global-pain-council-guidelines

Recent human papers evaluating safety in:
- Intensive care and cerebral perfusion in the ICU (not increasing ICP)
- Reduced neurotoxicity and improved perfusion in neuro-anesthesia
- Improved hemodynamics for sedations in ICU
- Modification of neuropathic pain and anxiety disorders
- Replacement for opioid drugs in acute pain in opioid-free and reduced opioid procedures

Veterinary uses: Two primary considerations: Anesthesia and sedation:

Acute Analgesia: NMDA antagonism reduces requirement for opioids, reduced post-operative pain, reduced wind-up at the level of the spinal cord and glia.

The Global Pain Council of the WSAVA utilizes ketamine extensively in the recommendations for countries where opioid analgesics, and safe non-steroidal analgesics are limited.

www.wsava.org/guidelines/global-pain-council-guidelines

Chronic Analgesia: NMDA antagonism mitigates glial hyper-activity and neuropathic pai
- Spinal cord injury, brain injury peripheral nerve injury
- Chronic OA

Low cost and high margin of safety at sub-anesthetic doses

Systemic and peripheral applications

Other (oral) NMDA antagonists:
- Amantadine 2-5 mg/kg q 12-24 hours
- Memantine 0.3-0.5 mg/kg q 12-24 hours

WSV18-0200

CARE FOR A CHRONIC ENTEROPATHY CASE: MEDICINE AND NUTRITION THERAPIES

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Care for chronic enteropathy cases: medicine and nutritional therapies

Drs Minna Rinkinen and Marge Chandler

Diet and Chronic enteropathy

The gastrointestinal tract (GIT) is impacted directly by the diet more than any other part of the body. It is affected by the nutrients, the frequency and timing of meals, and the effect on the microbiome. The diet may contain toxins, allergens, nutritional excesses or deficiencies. The diet also has a direct effect on GI physiology, affecting motility, cell renewal rate, enzyme production, immune functions, ammonia production, and volatile fatty acid content. Nutrition plays a key role in the management of many GI diseases, and many cases may be managed by dietary therapy alone.

Nutritional Assessment

Nutritional assessment is part of the routine history taking and physical examination. Nutritional assessment has two parts: a screening evaluation and an extended evaluation if areas of concern are found. The screening evaluation should be performed at every veterinary visit and includes a diet history, body weight, body condition score (BCS), muscle condition, and evaluation of the coat and teeth.

 A complete dietary history is especially crucial for patients with GI disease. Often the pet has access to treats, foods provided to give medications, or outside food sources (e.g., scavenging or hunting) which the owner may not consider part of the "diet", so the questioning must be done carefully.

Chronic diarrhoea

Chronic diarrhoea is diarrhoea which has lasted longer than 2 weeks. It has many potential aetiologies, including adverse reactions to food, inflammatory bowel disease/chronic enteropathy, parasites, infectious agents, neoplasia, and systemic disorders such as pancreatitis, pancreatic insufficiency, kidney or liver disease, and hypoadrenocorticism.

Large vs small intestinal diarrhoea

One of the first diagnostic steps is to determine if the diarrhoea is large or small intestinal in origin or both. Many patients exhibit signs which fall into "both"

categories, which may be due to small bowel disorders affecting the large bowel function or disease which involves both the small and large bowel. Up to 30% of dogs with chronic diarrhoea have diffuse disease of the gastrointestinal tract. The classic signs of large vs small intestinal diarrhoea are presented here.

SIGN	SMALL BOWEL	LARGE BOWEL
Faecal volume	Large	Small
Faecal frequency	Increased 4x	Increased 8x
Haematochezia	None or digested blood (melaena)	None or fresh blood.
Faecal mucus	None	Often present
Steatorrhoea	Possible	No.
Tenesmus	None	Frequent
Dyschezia	No	Frequent
Flatus/borborygmic	Present	Present
Vomiting	Possible	Possible
Weight loss	Common	Relatively rare

Inflammatory bowel disease (IBD) or Chronic Enteropathy (CE)

Canine and feline IBD or CE is a heterogeneous group of disorders characterized by persistent or recurrent gastrointestinal signs and in the case of IBD also an inflammatory infiltrate within the GIT. It may affect the stomach, small intestine, colon or any combination of these organs. The infiltration is most often lymphoplasmacytic but may include eosinophilic and neutrophilic infiltrates and may be associated with crypt abscessation and/or lacteal dilation with protein-losing enteropathy (PLE). The underlying cause is not fully understood and is likely to be multifactorial. " When the disorder affects the small intestine "chronic enteropathy" is probably a better term than "inflammatory bowel disease" in dogs and cats, because the treatment and outcome of the disease is very different from that of inflammatory bowel disease in humans[1].

About two-thirds of canine CE cases respond to an elimination diet trial with a hydrolysed protein diet or novel ingredient diet [2,3] (see below). Hydrolysed diets may improve nutrient absorption and decrease antigenic exposure. In dogs which don't respond to dietary therapy, about 16% will respond to antibiotics (e.g. metronidazole or tylosin) and about another fifth may require immunosuppressive medications (prednisolone, cyclosporine). Food responsive dogs have a better outcome than the other groups[2]. In a study comparing a highly digestible "intestinal" diet and a hydrolysed diet in dogs with chronic small intestinal disease, both diets improved clinical signs initially; however, longterm remission at one year was better using the hydrolysed diet[4]. Thirty-one of 39 dogs with food responsive chronic diarrhoea did not have a recurrence of clinical signs when switched back to their original diet after 14 weeks of elimination diet trial[5]. A diet with high protein, low carbohydrate, and moderate fat was effective in resolving diarrhoea in 7/15 cats[6]. A highly digestible diet appears to be effective regardless of the fat content[7].

Anorexia and poor appetite as a GI sign

There are many reasons for decreased appetite or anorexia. Food responsive enteropathy can cause nausea and vomiting and, subsequently, lead to learned food aversion (LFA). Especially in young dogs that show intermittent GI signs and reduced appetite, or are "finicky eaters", LFA secondary to CE should be suspected. An important differential diagnosis for poor appetite is hypoadrenocorticism, which should be ruled out either by basal cortisol measurement or ACTH stimulation test[8](Bovens et al 2014).

Diet trial

Adverse reactions to food are diagnosed using elimination-challenge trials. Dietary trials confirm or rule out adverse reactions to food but do not establish an immune mediated basis for the reaction, although that does not affect the case management. Ingredients previously fed should be avoided or a hydrolyzed protein diet can be fed. Hydrolysed proteins are generally less antigenic than whole proteins. Absolutely no other foods or ingredients should be fed during the diet trial as this makes it impossible to confirm that diet is part of the problem. Counselling the owner on feeding management, including the feeding of treats or snacks, is key to the success. Dogs with antibiotic responsive enteropathy often respond better if the antimicrobial therapy is combined with an elimination diet. Animals that respond to elimination diets usually do so within 2 weeks, although rare patients may require 4-6 weeks.

To confirm an adverse reaction to food a rechallenge or provocation is necessary. The initial food in reintroduced or individual ingredients from the initial diet are added to the elimination diet one by one. Cases with gastrointestinal disease usually react within several days. Many clients do not want to rechallenge and the pet can be kept on the test diet if it is complete and balanced or another novel protein complete diet can be tried [10].

If an elimination diet trial does not resolve the CE signs, additional diagnostics and therapies are warranted. Dogs with idiopathic antibiotic-responsive diarrhoea respond well to antibacterials (e.g. metronidazole or tylosin). Some chronic inflammatory enteropathy (CIE) patients fail to respond to diet or antibiotics. Definitive diagnosis of CIE is based on histology; therefore, endoscopy and histopathological evaluation of mucosal biopsies are needed before initiating the immunosuppressive medication (e.g corticosteroids, cyclosporine or chlorambucil).

Prebiotics and probiotics

Prebiotics are complex carbohydrates which are fermentable, promote the growth of beneficial intestinal bacterial and decrease the growth of pathogenic bacteria, e.g. fructo-oligosaccharide and mannos-oligosaccharide's. Probiotics containing non-pathogenic bacteria such as **Bifidobacterum**, or **Enterococcus faecium**, are used to increase the ratio of normal to pathogenic GI microbes, which have a variety of effects on the intestine.

Cobalamin (Vitamin B12) and folate

Many animals with CE are cobalamin deficient[11,12]. Cobalamin is needed for GI epithelial cell turnover and repair, and in many feline GI cases signs won't resolve until cobalamin has been repleted. Serum cobalamin concentrations are usually measured simultaneously with serum folate concentrations. Folate can become deficient when cobalamin is replaced and may also need to be supplemented. Cobalamin has previously been administered parenterally, although a recent study showed that oral cobalamin supplementation was effective in normalizing serum cobalamin concentrations[12,13].

References available upon request

WSV18-0258

SVA SOFT TISSUE SURGERY

SURGERY OF THE EAR

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Recurrent unresponsive otitis is a frequent indication for aural surgery in the dog. Certain breeds are predisposed such as the spaniels due to their pendulous ears, shar peis with their narrow ear canals, and poodles due to excessive hair growth. Cavalier King Charles can develop secretory otitis media and cocker spaniels may have increased cerumen production.

Inflammation from otitis may be a precursor to tumour development and increased glandular dysplasia. Many aural tumours are linked to chronic inflammation. Benign lesions include cholesteatomas, inflammatory polyps, papilloma, basal cell tumour, and ceruminous gland adenoma. However, malignancies such as ceruminous gland adenocarcinoma and squamous cell carcinoma can also be seen. Cats will commonly be evaluated for inflammatory polyps or squamous cell carcinoma of the ear tip.

In addition to the otoscopic examination it is important to perform a complete neurological assessment and thorough external palpation of the external ear canal. Local extension of infection or neoplasia can cause facial nerve deficits, Horner syndrome, and/or peripheral vestibular signs. Intracranial extension of the process can cause forebrain, brainstem, cerebellar or central vestibular signs. Identification of neurological deficits warrants pre-operative CT or MRI. Differential diagnoses such as Chiari like malformation should be considered for at risk breeds.

In most cases of severe persistent otitis a **total ear canal ablation** is the most practical management option. Medical therapy should be based on culture and sensitivity and administered prior to surgery to optimize surgical conditions – however in most cases extensive delay is not prudent as this can result in development of multi drug resistant organisms while not addressing the underlying cause. A lateral ear canal resection is often reserved for disease processes limited to the lateral vertical canal (such as a focal neoplasia). Lateral ear canal resections for severe otitis typically will result in stricture due to the profound inflammation and inability to maintain an adequate aural opening.

Adequate analgesia should be provided prior to, during and following a total ear canal ablation and can include full agonist opioids, local blocks or infusions and anti-inflammatories. The surgical approach is well described, attention should be given to avoiding damage to the retroglenoid/retroarticular vein (rostral), facial nerve and carotid artery and maxillary vein. Pre-

operative CT will assist in determining the extent of middle ear involvement however a bulla osteotomy is almost invariably required in cases of severe otitis. The placement of a drain following surgery remains controversial and is not considered mandatory. In instances of subsequent abscessation this is more likely associated with inadequate debridement of the epithelial lining of the bulla rather than the lack of drain placement.

Post-operative care is based around providing adequate analgesia. Management of facial nerve paralysis in hospital is required in some cases with reports describing up to 36% of dogs developing transient facial nerve paralysis and up to 13% permanent paralysis (these numbers are expected to be higher in cats).

Pinnectomy is performed most often for neoplastic lesions, commonly squamous cell carcinoma in the cat. Margins should be 1-2cm and skin is brought over the cut edge of the cartilage (usually from the convex surface) and then sutured.

Ventral bulla osteotomy is a common management option for cats with inflammatory polyps and those with disease limited to the tympanic bulla. The true middle ear is separated from the larger hypotympanic cavity and must be concurrently accessed. Cats are positioned in dorsal recumbency and incision centred over the tympanic bulla. Relevant anatomical structures encountered during the approach include the salivary gland, bifurcation of the linguofacial and maxillary veins, hypoglossal nerve, and lingual artery.

The incidence of neurological deficits following otic surgery is higher in cats than in dogs however attempts to avoid excessive curettage of the promontory can help reduce the risk of Horner Syndrome when performing the ventral bulla osteotomy.

WSV18-0287

INTEGRATIVE MEDICINE (LECTURES GIVEN IN MANDARIN CHINESE)

INTEGRATIVE MEDICINE FOR CANCER PATIENTS

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Introduction

Cancer is a common disease in small animal practice; it has been estimated that 1 in 4 dogs and 1 in 6 cats will develop a form of neoplasia at some point during their life.[1,2] It has been reported that almost 50% of dogs over 10 years old die of cancer-related problems, and cancer is the most common cause of natural death in dogs and cats in the United States.[1] However, treatment of cancer remains a challenge for both veterinarians and caretakers.

Conventional medicine understands neoplasia to arise due to a decrease in anti-tumor defenses, increased mutations and silencing of tumor suppressor genes such as p53 through methylation and deacetylation, along with exposure to carcinogens and a decrease in immune defenses and therefore DNA repair mechanisms. While we are beginning to unravel the complex biochemistry of cancer development and have begun to understand how DNA is damaged and repaired, we still have a long way to go before the cure for cancer will be found.

Traditional Chinese Medicine (TCM) has been used successfully in the treatment of cancer for thousands of years, long before we understood the basic pathobiology of tumors in Western medicine. Traditional Chinese Veterinary Medicine (TCVM) shares the similar philosophy and treatment of cancer in TCM. TCVM is not a replacement for Western diagnosis and therapy, but may be used with Western approaches to help heal patients. When the option for Western therapy is lacking, there are TCVM therapies which can be employed to help the patient live a quality life, reducing the rate to cancer expansion or, in some cases, leading to remission of the cancer. TCVM may be best suited to prevention of the development of cancer through healthy living. On the other hand, Chinese herbal medications have been shown to lead to spontaneous remissions of cancer. In some cases, these herbal products can be used in conjunction with traditional Western therapies, improving the outcome and reducing the side-effects from Western therapy alone. An integrative approach combining the best of both Western medicine and TCVM seems to be the only sensible course of action, providing the best overall care for the patient.

TCVM Etiology And Pathology

From a TCVM perspective, cancer is the result of Stagnation and lack of free movement of Qi and/or Blood. Tumors are the end result of a prolonged process of accumulation and densification of tissue due to the Stagnation and eventual Stasis of Qi and Blood. Many factors can cause this blockage in Qi or Blood flow and therefore lead to development of cancer. The common underlying (root) factors are: Zheng Qi (vital Qi) deficiency, Qi & Blood deficiency and Qi & Yin deficiency. Zheng Qi composed of Ying Qi (Nutritive Qi) and Wei Qi (Defensive Qi); When Qi is deficient, there will be Deficiency of the Wei Qi. If the Wei Qi is deficient, external pathogenic factors (Cold, Wind, Heat, Summer Heat, Dry and Damp) cannot be expelled from the body, blocking flow of Qi and Blood and allowing cancer to develop. It can therefore be appreciated that Qi deficiency is the root of cancer, and Phlegm accumulation, Qi/Blood Stagnation and Blood Stasis are the branches.

1. **Qi & Blood Deficiency:** Patients with the pattern of Qi & Blood deficiency have a lower cell immunity response than normal. Symptoms include: hair loss; dizziness; fatigue; a thin or obese body; shortness of breath; poor appetite; insomnia; abdominal pain; chronic digestive issues; a pale tongue with a white tongue coating; and a deep, thin, and weak pulse.

2. **Qi & Yin Deficiency:** Patients with Lung Qi deficiency may have a lower lymphocyte transformation rate and lower levels of serum immunoglobulins such as IgM and IgG. Symptoms include: panting; shortness of breath; insomnia; chronic respiratory or skin issues; cough without phlegm; lassitude; dry mouth; a thin tongue coating; and a thin pulse.

Treatment Strategy:

1. **Acupoints:**
   - LI-4, LI-11, ST-36, SP-6, SP-10, HT-7, BL- 17, BL-20, BL-21, BL-24, BL-26, TH-5, CV-6, Shen-shu
     - LI-4 is the Yuan-Source point that clears all Channels and calms the mind (especially when used with LIV-3), moves masses, tonifies and moves Qi and Blood, clears Heat and is a general immune stimulant
     - LI-11 tonifies Qi and clears Heat
     - ST-36 is a major tonification point; it tonifies the Spleen, Stomach, Qi and Blood and stimulates the appetite

- SP-6 (the crossing point of the three Yin Channels of the pelvic limb) tonifies the Spleen, Yin, Qi and Blood
- SP-10, the Sea of Blood point, tonifies, moves and cools the Blood
- HT-7 is a Shu-Stream point and relieves pain, calms Shen and tonifies Heart Blood and Qi
- BL-17 is the Influential point for Blood, which invigorates and nourishes Blood
- BL-20 is the Back-Shu Association point of the Spleen, which is used for Deficient Spleen Qi and Deficient Blood
- BL-21 is the Back-Shu Association point of the Stomach, and tonifies Spleen and Stomach Qi, Yuan (Source) Qi and Kidney Jing
- BL-24 is the Sea of Qi point, which tonifies Qi
- BL-26 is the Gate of Yuan (Source) Qi, which tonifies Qi
- TH-5 tonifies Wei Qi
- CV-6 is the Sea of Qi point; it tonifies Yang, Qi and Blood, and moves Stagnant Qi in the abdomen
- Shen-shu is a classical point that tonifies Yuan (Source) Qi

1. **Chinese Herbal Formulas:**
   - Qi Blood Deficiency:
     - Shi Quan Da Bu Tang: Tonifies Qi, nourishes Blood and tonifies Yang; indicated for Qi deficiency accompanied by Blood and Yang deficiencies.
     - Gui Pi Tang (Restore the Spleen Decoction): Nourishes Blood and tonifies Spleen Qi; indicated for combined Qi and Blood Deficiency
     - Xiang Sha Liu Jun Zi Tang (Eight Gentlemen): Tonifies Qi, strengthens the Spleen, moves Qi and eliminates Damp; indicated for Qi deficiency with Dampness and anorexia.
     - Bu Zhong Yi Qi Tang (Tonify the Middle and Augment the Qi Decoction): Tonifies Qi and raises sunken Yang; indicated for severe Spleen Qi deficiency causing low fever, chronic

diarrhea and organ prolapse.

- Wei Qi Booster (JT): Tonifies Qi and Blood, boosts Wei Qi and inhibits mutation

- Anticancer formulas:
  - Max's Formula (JT): Softens hardness, transforms Phlegm and clears enlargement. The classical antecedent of this formula is Nei Xiao Luo Li San.
  - Stasis Breaker (JT): Breaks down Stasis, softens hardness and clears enlargement. The classical antecedent of this formula is Nei Xiao Wan.
  - Xue Fu Zhu Yu Tang (Drive Out Stasis in the Mansion of Blood Decoction): Invigorates Qi/Blood, breaks down Blood Stasis and relieves pain; indicated for Blood Stasis with concurrent Blood Deficiency
  - Stasis in the Mansion of the Mind (JT): Breaks down Blood Stasis, transforms Phlegm, resolves nodules and relieves pain; used for tumors in the brain, particularly when Phlegm is accompanied by Blood Stasis and pain

- Transporter formulas:
  - Bone: Bone Stasis Formula (JT)
  - Nose and sinuses: Xin Yi San
  - Neck: Cervical Formula (JT), or Ge Gen Tang
  - Thyroid: Hai Zao Yu Hu Tang (Sargassum Decoction for the Jade Flask)
  - Spine: Da Huo Luo Dan
  - Thorax: Ge Xia Zhu Yu Tang
  - Lung: Qing Fei San
  - Abdomen: shao Fu Zhu Yu Tang
  - Vessels or heart: Xue Fu Zhu Yu Tang
  - Mammary glands: Breast Stasis Formula (JT), or Chai Hu Shu Gan Wan
  - Prostate: Prostate Invigorator (JT), or Qian Lie Xian Fang
  - Bladder: Wu Ling San

- Herbal Formulas for Treating Clinical Signs in the Cancer Patient
  - Anemia: Si **Wu Tong** or **Gui Pi Tang**
  - Anorexia: Xiang Sha Liu Jun Zi Tang (Eight Gentlemen)
  - Anxiety or Depression: Shen Calmer or

Tian Wang Bu Xin Dan

- Bleeding/Hemorrhage: Yunnan Baiyao
- Bowel Incontinence: Bu Zhong Ti Qi Tang
- Constipation: Ma Zi Ren
- < >: Xiang Lian San or Shen Ling Bai Zhu
  Coughing: Zhi Sou San
- Cystitis: Ba Zheng San
- Edema: Shi Pi Yin
- Fatigue: Bu Zhong Ti Qi Tang, Si Jun Zi Wan
- Immunosupression: Gui Pi Tang
- Indigestion: Bao He Wan, Si Jun Zi Wan
- Nausea/Vomiting: Happy Earth (JT) or Ping Wei **San**

1. **Diet Therapy:**
   - Although eating healthy is the best tool in the fight against cancer, once cancer takes hold certain dietary changes may be help the patient fight against the effects of the cancer. Tumor cells rely heavily upon carbohydrates for their energy and rob the body of amino acids. On the other hand, tumor cells cannot utilize lipids (fats) for energy while the rest of the body can. As such, diets with increased fat content may slow tumor growth, allowing the patient to fight against the tumor. Protein content must be maintained a level sufficient for tissue repair, but carbohydrates should be held to a minimum. For those who cannot cook for their dog, a commercial food should be of good quality, moderate protein, moderate fat, and low carbohydrate (<10%) content.

1. **Dietary Supplements:**
   - The rationale for each of these products is sound, but more than I wish to explain at the moment. Antioxidants (such as vitamin E, vitamin C, selenium, beta-carotene, ginkgo bilboa, green tea and grape seed extract) do protect and help stabilize the immune system. Collagen support may help inhibit angiogenesis by the tumor. Mushrooms and astragalus help boost the immune system (activate NK Killer cells which attack tumor cells and to prevent destruction of T-Helper cells). n-3 polyunsaturated fatty acids (PUFAs, or Omega 3 Fatty Acidss), eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), can exert anti-neoplastic activity by inducing apoptotic cell death in cancer cells

either alone or in combination with conventional therapies. COX-2 inhibitor drugs double life expectancy with carcinomas while melatonin appears to improve survival times in all solid tissue tumors including gliomas. None of these measures will necessarily treat or cure cancer, but they will not do any harm and may provide quality of life. That is probably what is important in cancer which cannot be surgically removed.

Summary

Combining Integrative Medicine with mainstream oncology care can reduce clinical signs burden and improve the quality of cancer care and quality of life, and the well-being of patients and their owners.

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WSV18-0302

SVA SOFT TISSUE SURGERY

FELINE PERINEAL URETHROSTOMY, A NOVEL APPROACH

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Key Points

- Patients with cystic and urethral calculi present with stranguria
- Retropulsion of urethral calculi into the urinary bladder simplifies management of urethral calculi
- Aggressive lavage of the urethra and bladder should be performed during cystotomy
- Permanent urethrostomy is an acceptable method of managing chronic stone formers

Definition: Cystic and urethral calculi have various compositions (i.e., oxalate, struvite, urate) and may be present in the urinary bladder or lodged in the urethra, respectively. They may be multiple or single, may cause partial or complete obstruction (i.e., urethral), and may require surgical manipulation for removal.

DIAGNOSIS

Clinical presentation:

Signalment: There is no age, sex or breed predisposition.

History: Patients generally present with a history of urinary obstruction and/or signs of urinary tract infection. Common complaints include difficulty urinating, straining to urinate, hematuria, blood tinged urine in the litter pan, and/or a distended abdomen. Patients that present several days after complete obstruction may have a distended and painful abdomen and a history of anuria. These patients may be so compromised that they present in shock.

Clinical signs: The most frequently reported clinical signs in patients with cystic and urethral calculi include unproductive straining to urinate, blood tinged urine seen in the litter pan, hematuria, and/or polakiuria. Severity of clinical signs may vary with the degree of urethral obstruction and duration of obstruction prior to presentation. Patients with complete obstruction for several days may show signs of post-renal azotemia (i.e., severe depression, recumbant, shocky).

Physical examination: Abdominal palpation may reveal a full urinary bladder; occasionally, calculi within the bladder may be palpable. Patients with severe clinical signs (i.e., presented several days after complete obstruction) may show azotemia, shock, and/or severe depression. Abdominal palpation generally reveals a large, turgid urinary bladder and may result in discomfort to the patient.

Laboratory findings: Results of a complete blood count and serum chemistry profile are generally normal in patients presenting acutely; urinalysis may show evidence of urinary tract infection and and/or crystalluria. Patients presenting after several days of complete obstruction may have significant changes in their biochemical profile including increased BUN, increased creatinine, metabolic acidosis, and severe electrolyte abnormalities. Urine is generally grossly hemorrhagic and urinalysis may show signs of urinary tract infection and crystaluria.

Radiography: Survey radiographs may show presence of radiodense calculi in the urethra and/or urinary bladder as well as a distended urinary bladder. Occasionally, radiolucent calculi occur and can only be visualized using retrograde contrast cystourethrography. Careful radiographic evaluation of the kidneys and ureters should be done to rule out renal and ureteral calculi.

Ultrasonographic examination of the bladder, ureters, and kidneys may be helpful in diagnosis of cystic, ureteral, or renal calculi.

Differential diagnosis: Any disorder causing urinary obstruction, including urethral neoplasia, granulomatous urethritis, urethral stricture, and urethral trauma. Definitive diagnosis is based on clinical signs, inability to pass a catheter, and evidence of calculi on survey or contrast radiographs.

MEDICAL MANAGEMENT:

Immediate care: In animals with complete obstruction long enough to cause azotemia, temporary urinary diversion is provided by performing a prepubic cystostomy (see technique described below) or frequent cystocentesis (i.e, tid to qid). Azotemia is treated with crystalloid IV therapy prior to calculus removal.

Urethral catheterization of a female cat:

Female urethral catheterization is easier than male

Use a closed ended tom cat catheter

Ventral recumbancy is recommended

Pass the catheter with no evidence of resistance

RETROGRADE HYDROPULSION OF LODGED URETHRAL CALCULI

Calculus removal: Retrograde hydropulsion: This technique should result in an 80-85% success rate for retropulsing urethral calculi into the urinary bladder!

Thoroughly mix 20 cc of sterile saline and 5 cc of Surgilube or K-Y Jelly in a 35 cc syringe and attach the syringe to a 3.5 - 5.0 French soft rubber catheter/feeding tube.

Anesthetize the patient, extrude the penis and pass the lubricated urinary catheter in the urethra up to and against the calculus. Place a dry gauze sponge around the extruded tip of the penis and occlude the penis around the catheter by squeezing it with thumb and finger.

Using a back and forth action on the catheter, simultaneously inject the saline/lubricant mix under extreme pressure.

a) During injection, the calculi and urethra are lubricated by the saline/lubricant mix while the viscosity of the mixture (i.e., KY jelly and saline) encourages the calculus to dislodge and become retropulsed into the urinary bladder.

b) This technique is attempted, and generally successful, regardless of how many stones are in the urethra and no matter where they are lodged.

If the above technique fails, use a stiffer catheter (i.e., open or closed ended tomcat catheter) and repeat the above maneuvers. Use care when manipulating these stiffer catheters against the calculus.

SURGICAL TREATMENT:

The objective of surgical treatment is to remove all retropulsed calculi from the urinary bladder and any remaining urethral calculi that were unable to be retropulsed. Bladder calculi are removed via cystotomy, urethral calculi are removed via urethrotomy, and patients that are frequent stone formers may benefit form a permanent urethrostomy to allow continual passage of small urethral calculi.

Preoperative management: Patients that present acutely can be anesthetized immediately and retropulsion attempted (see above described technique). If urinary tract infection is suspected, preoperative treatment with antibiotics may be instituted.

Patients that present after several days of complete obstruction should be treated medically until the azotemia resolves, blood gas abnormalities resolve, and electrolytes return to normal. The patients' electrocardiogram should be monitored if hyperkalemia is present preoperatively. Medical treatment may consist of intravenous fluids, systemic antibiotics, continuous ECG monitoring, and bladder decompression. Bladder decompression may be accomplished via multiple cystocentesis (i.e., tid or qid), or placement of an antepubic cystostomy tube (described in detail below).

Anesthesia: Routine general anesthesia is performed in patients that present acutely without signs of azotemia. Azotemic, shocky patients with moderate to severe biochemical abnormalities should be treated as described above until these abnormalities return to normal.

Surgical anatomy: The male feline penile urethra consists of urethral mucosa (i.e., urothelium) surrounded

by corpus cavernosum urethra, which is in turn surrounded by tunica albuginea. Because of the blood filled corpus cavernosum urethra and the tough fibrous connective tissue tunica albuginea, the urethra can withstand tremendous pressure (e.g., as with aggressive retropulsion) without the fear of urethral rupture.

The urinary bladder consists of the following layers; serosa, muscular, submucosa and mucosa. The bladder is lined with transitional epithelium.

Positioning: Patients are positioned in dorsal recumbancy for retropulsion, cystostomy tube placement and routine cystotomy.

Urethrostomy: Urethrostomy is generally performed in patients that are recurrent stone formers. It provides a permanent opening that is large enough to accommodate passage of most urethral calculi, crystals and mucoid debris.

Perineal urethrostomy; perineal approach: The perineal urethra is the location of choice for urethrostomy in cats. It is a convenient location for surgical manipulation, the urethral diameter will accommodate passage of most urethral calculi and there is less urine scald postoperatively.

Prior to surgery a urethral catheter is passed, if possible. After a routine castration, an elliptical incision is made around the scrotum and penis. Then the subcutaneous tissues are dissected to expose penile urethra. The penile urethra is dissected free from surrounding connective tissue. The ventral attachment of the pelvic urethral to the pubis (i.e., ishiocavernosus m.) is identified and transected. The penile urethra is freed from its connective tissue attachments to the pelvic floor using blunt digital dissection. The retractor penis muscle is identified on the dorsal aspect of the penis and is dissected from its attachment on the penis. The dissected retractor penis muscle is then used to develop the dorsal plane of dissection to separate the pelvic urethra from its dorsal connective tissue attachments. Once the urethra is dissected enough to visualize the dorsolaterally located bulbourethral glands penile dissection can stop. The penis is catheterized and the urethral orifice identified. An incision is made from the penile urethra to the pelvic urethral to the level of the bulbourethral glands using a Stevens tenotomy scissor or Iris scissor. The urethral orifice at the level of the bulbourethral glands is generally of large enough diameter to accept the flange of a tomcat catheter.

After incision of the urethra, the glistening urethral mucosa is identified. 5-0 nonabsorbable monofilament suture with a swaged on cutting or taper-cut needle is recommended by the author. The first urethrostomy suture is placed at the dorsal aspect of the urethrotomy incision on the right or left side at a 45o angle to include urethral mucosa and skin (suture split thickness of skin).

The suture is tied and cut leaving the ends 3-4 cm long to act as a stay suture. A mosquito hemostat is placed on this suture to provide traction and countertraction to enhance visualization of the urethral mucosa. The second suture is placed opposite the first suture and tied as described for the first. A stay suture is also placed here. A third urethrostomy suture is placed directly on the dorsal midline to hold the dorsal margin of urethral mucosa to the dorsal margin of the skin incision. Alternating sutures from dorsal to ventral are placed until approximately one half of the penile urethra has been sutured to skin. The remainder of the penis is amputated and the subcutaneous tissue and skin are closed routinely. Fine ophthalmic instruments make tissue handling and suturing easier. Use of a 2X magnifying loupe and headlamp light source enhances visualization of the urethral mucosa and facilitates accurate suturing. It is critical for the surgeon to recognize the glistening urethral mucosa and carefully suture it to skin. This will decrease (or eliminate) the chance of urethral stricture.

Perineal urethrostomy; dorsal approach: Perineal urethrostomy can be performed with the patient placed in dorsal recumbency. This positioning is more ergonomic for the surgeon and allows easy access of the urinary bladder for concurrent cystotomy. When positioning the cat tie the hind limbs cranially until the pelvis is slightly elevated off the surgery table. Place a folded towel under the pelvis to support this slightly elevated position. The surgical technique is as described above for the perineal urethrostomy performed using a perineal approach.

POSTOPERATIVE CARE AND ASSESSMENT:

Perineal Urethrostomy: An Elizabethan collar should be considered, especially in patients that may be prone to self-mutilation. Patients should be kept quiet and away from other animals. An indwelling urinary catheter placed routinely postoperatively is NOT necessary following an uncomplicated urethrostomy.

PROGNOSIS

The prognosis for surgical management of urethral and cystic calculi is dependant upon preoperative management of azotemic patients prior to anesthesia, success of retropulsion of urethral stones into the urinary bladder, care in removing all stones via cystotomy, and care of ensuring urethral mucosa to skin apposition during urethrostomy.

Patients that have successful retropulsion of urethral calculi and do not require urethrostomy have an excellent prognosis. If careful attention is paid during cystotomy to ensure that no calculi are left behind (see discussion on cystotomy technique), the prognosis for cure is excellent. Long term prognosis is dependant on evaluaiton of calculus composition, dietary management, management of urinary tract infection, and attention to

urine pH.

Patients that have an elective perineal urethrostomy have a favorable prognosis if attention is paid to proper surgical technique (i.e., urethral mucosa is sutured to skin). Occasionally, chronic stone forming patients will form a calculus that is too large to pass through the urethrostomy stoma.

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REPRODUCTION

UPDATE ON CLINICAL USE OF AGLEPRISTONE IN BITCHES AND QUEENS

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Introduction

The progesterone (P4) antagonist Aglepristone has been available in Europe since early in this century. It is an interesting and unique molecule with high affinity for progesterone (P4) receptors in vitro (three times greater than that of P4 in the canine species and nine times in the feline species). Aglepristone binds to uterine progesterone receptors without producing the biological effects of progesterone. Thus it can be used to block the action of P4 during pregnancy (fœtal resorption, abortion, induction of parturition) or non-pregnancy (uterine and mammary conditions) in the bitch, and also in the queen. It is an oily injectable solution for subcutaneous administration at the dose of 10 mg/kg repeated on two consecutive days, 24 hours apart. Aglepristone is currently marketed with the indication to terminate pregnancy in the bitch. However, because of its novel mechanism of action, it has been the object of numerous studies over the last 2 decades and several indications have been proposed both in pregnant as well as non pregnant females as well as for endocrine, mammary and perineal conditions.

INDICATIONS FOR PREGNANT FEMALES

Early and late pregnancy termination - Aglepristone (AGL) is currently marketed with an indication for pregnancy termination in dogs at any time between day 0 (the day of mis/mating) and day 45. Efficacy is very high, 95-100% and side effects are extremely rare and characterized by a short-lasting pain at the injection site [1, 2, 3]. Treatment causes prevention of implantation if performed prior to day 24 post-ovulation, embryonic resorption if performed between day 25 and 35 post-ovulation, and expulsion of well developed foetuses after day 35 post-ovulation[4]. Clinical signs vary from no sign when prevention of implantation or resorption occurs to vulvar discharge, behavioural signs of parturition and occasionally fever, loss of appetite and metritis following fetal expulsion. The leaflet's indication to be used between day 0 and 45 is somewhat controversial due to the fact that a) bitches may ovulate up to 5-7 days following mating and still conceive, and b) treatment in late pregnancy may take up to 9-10 days to achieve an effect and therefore 45-day old foetuses may occasionally be still alive following a late treatment and either be expelled live around day 53-55 or die and not be expelled from the uterus. Therefore it is advisable

to a) **make sure that in early pregnancy corpora lutea are present prior to starting treatment** by assaying serum P4, and b) **avoid using aglepristone as the only abortifacient drug in bitches who are pregnant beyond day 40**. Treatment failures are occasionally observed, therefore it is always advisable to recheck treated females at least twice at weekly intervals following the end of treatment. Treatments with aglepristone can be started after day 40 provided that the issue is thoroughly discussed with the owner and a prostaglandin or antiprolactinic treatment is associated to help evacuating the uterus. There is no effect on subsequent fertility although the first post-treatment estrus may be delayed. AGL is effective to induce abortion in cats. The suggested protocol is the same although most authors use a higher dosage for cats, 15 mg/kg; however the 10 mg/kg dose seems equally effective at least during the first half of pregnancy[5,6]. The success rate is similar to the bitch during the first half of pregnancy, while effectiveness may drop to 66% when causing late term abortion[7]. Hemorrhagic vulvar discharge following abortion has been reported to occur in pregnant queens treated with aglepristone: this is a consequence of damage to maternal uterine venules with endometrial and cervical extravasation and blood loss, a feature which has not been observed in bitches[4].

Planning parturition or C-Section – When used near term, AGL will induce labor. Parturition should be induced as close to spontaneous parturition as possible, therefore ovulation should have been staged previously. Whelping has been successfully induced in bitches treated on day 58 and 59 day of gestation using AGL alone at the dose of 15 mg/kg or combined with oxytocin or prostaglandin F2alpha. Gestation length is shorter in treated vs control bitches (59 vs 62 days)[8]. Fontbonne and coworkers[9] used a combined AGL+oxytocin treatment: one injection of aglepristone at the dose of 15 mg/kg was administered on day 59-61, and then starting 24 hrs later oxytocin was administered at the dose of 0.15 IU/kg every 2 hr. Parturition began approximately 30 hours after the Aglepristone injection (from 9:00 to 12:00-18:00 of the following day) and resulted in the birth of puppies which were alive and viable at 1 month [9]. Length of parturition, expulsion time and incidence of neonatal mortality are comparable to what happens during normal parturition. However, 2 small size treated bitches delivered some of their pups before the first administration of oxytocin[9]; furthermore, 4 Yorkshire terrier pups (treated group) were born premature and died at 19-29 hr after birth[9].

AGL can also be used in the planning of an elective caesarean section, particularly if surgery needs to be done prior to physiological termination of pregnancy because of fetal death, or in case of prolonged singleton pregnancy. Levy et al.[10] administered one injection of 15 mg/kg AGL 59-60 days post-ovulation to 37 bitches of 15 breeds. C-section was performed 20-24 hours after treatment. There were no post-operative complications and no signs of prematurity in all pups. 5/188 pups died during the first 2 weeks of life (2.6%). Serum P4 remained > 2.0 ng/ml at time of surgery, which would justify the use of AGL as a high serum P4 concentration following a C-section would delay uterine involution[10].

INDICATIONS FOR NON PREGNANT FEMALES

Open and closed-cervix pyometra - Pyometra is a uterine condition characterized by accumulation of pus within the uterus and very often fever, luekocytosis, depression[11]. It is potentially a life threatening disease which has been treated for a long time mostly through surgical removal of the reproductive tract[12]. Medical treatment used to be possible only for open cervix cases while closed cervix ones could only be treated with (sometimes elaborate and dangerous) surgery[13], as the only way to open the cervix was using prostaglandins with the inevitable risk of causing uterine rupture[14,15]. AGL has solved this problem as cervical opening occurs without any appreciable uterine contraction within the first 48 hrs post-treatment[16,17]. AGL treatment of pyometra requires often a longer protocol than what is used for pregnancy termination; the usual dosage of 10 mg/kg aglepristone is administered on days 1, 2, and then at weekly intervals counting from day 1 for as long as necessary. Therefore, injections are typically given on days 1, 2, 8 and then also 15 and 28 or even longer depending on the clinical situation[18,19]. The use of aglepristone should be associated with antibiotics if necessary, and may also be associated with PGF/ PGE1 provided that cervical opening has occurred. Aglepristone is as effective for the treatment of pyometra also in the queen[6,20].

MAMMARY INDICATIONS

Feline mammary hyperplasia - Benign mammary hyperplasia is a benign fibroglandular proliferation of one or more mammary glands which typically occurs in young queens at their first luteal phase. The proliferation of the mammary gland is due to an excessive response to the action of progesterone which is present in presumably normal concentrations in affected animals. Mammary glands will start swelling rapidly and within 2-3 days all glands become very swollen, firm and nodular. If left untreated, the problem may disappear on its own without any complication in most cases. Treatment with prostaglandins or antiprolactinic is not effective, while removal of ovaries or administration of aglepristone is often (but not in 100% of cases) curative. When mammary hyperplasia occurs following progestogen administration, signs typically do not subside immediately following neutering or withdrawal of progestin therapy[21]. In such cases, surgical removal of persisting nodules should be considered in order to perform histology and rule out presence of neoplasia. Feline mammary hyperplasia may

be treated successfully with aglepristone[21,22,23], which may be an option also for cats treated with long-acting progestogens. Dosages for mammary hypertrophy may need to be prolonged in time depending on whether it is a spontaneous disease or if it is due to progestogen administration[4]. Muphung et al [24] studied the effect of AGL in a group of queens treated with a high dose of medroxyprogesterone acetate (MPA - 50 mg) followed by 2 injections of 10 mg/kg AGL 3 weeks later. Based on histology and immunohistochemistry, no evidence of an effect of AGL on the mammary gland of treated queens was present [24]. Lack of effect might be due to the very high dose of MPA used, to the rather low dose of aglepristone (10 mg/kg instead of 15 mg/kg), to the short treatment with AGL (only 2 injections) or to the long interval between MPA and AGL treatments.

ENDOCRINE INDICATIONS

Diabetes - Diabetes mellitus is a condition characterized by an altered carbohydrate metabolism due to an absolute deficiency of insulin, which is fairly common in the canine, particularly in dogs of > 5 years of age [25]. Neutered male and female dogs as well as intact (also pregnant) bitches are at a higher risk of developing this condition. In diabetic dogs, insulin deficiency is typically paralleled by an increase in glucagon which, coupled with the decrease in peripheral use, causes hyperglycemia[25]. The action of insulin is modulated by a number of so called counter regulatory hormones (glucagon, glucocorticoids, catecholamine and growth hormone) responsible for reducing the effect of insulin at the cellular level[26]. Whenever a higher than normal concentration of these hormones is present in the general circulation for a prolonged period of time a normal amount of insulin will produce a lower biologic response [26]. Such decreased response to insulin is referred to as insulin resistance, and it is generally suspected whenever hyperglycemia is present despite administration of single insulin doses higher than 1.0-1.5 IU/kg.

An increase in the concentration of the above counter regulatory (or diabetogenic) hormones occurs whenever there is a) an inflammatory condition, b) an endocrine disorder or c) following exogenous administration of one of the counter-regulatory hormones (i.e. glucocorticoids) or of other compounds which may raise their concentration, such as P4 [26, 27]. In the bitch, endogenous or exogenous P4 can cause insulin resistance by stimulating the release of growth hormone (GH) from the mammary gland thus raising GH concentrations in the general circulation [27,28,29]. While pituitary GH secretion is normally pulsatile, mammary GH secretion is not characterized by a pulse pattern and is not sensitive to stimulation and inhibition tests [30], the only exception being the capacity of being inhibited by the P4 antagonist AGL[31]. AGL binds to P4

receptors displacing P4 as well as progestogens from their binding sites, therefore it can be used to decrease clinical effects of high P4 concentrations directly as well as indirectly by causing a decrease in serum GH levels. A decrease in serum GH levels can be very important in an intact diabetic bitch as soon as diestrus starts, because of the high production of ovarian progesterone characterizing the first 2-3 weeks of the canine luteal phase. The effectiveness of AGL in helping in the clinical management of diabetes in intact bitches was assessed in a group of 8 diabetic intact bitches of 9-15 yrs of age diagnosed as having a P4-induced insulin resistance complicating the management of their glycemia, with blood glucose levels remaining persistently higher than 200 mg/dl over 12 hour periods regardless of being treated with doses higher than 1.5 IU/kg BID of insulin[32]. These animals, whose serum P4 was higher than 5.0 ng/ml at the beginning of the study, were treated with a 10 mg/kg dose of AGL on days 1, 2, 9 and 17, and compared to a control group of 6 diabetic intact bitches in which an equivalent amount of saline solution was administered on the same days of the treated bitches [32]. Both groups of dogs received a porcine insulin zinc suspension treatment as appropriate in relation to their serum glucose concentrations. Glycemia was controlled on the day of treatment (day 0) and on days 5, 12 and 20 post-treatment. While no significant variation was observed in serum glucose levels at day 5, glycemia was significantly reduced at day 12 (when the average dose of insulin could be reduced to 0.8 IU/kg) and further on at day 20. Progesterone did not change between day 0 and 20, while GH showed a significant decrease in AGL treated bitches[32]. The use of AGL should always be considered when dealing with diabetic intact bitches in diestrus.

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AQUATIC PET MEDICINE AND WORLD RABIES DAY

RABIES VACCINATION BACKGROUNDS AND FACTORS AFFECTING RABIES CONTROL IN AN ENDEMIC AREA FOLLOWED BY BOEHRINGER INGELHEIM/WSAVA WORLD RABIES DAY PANEL DISCUSSION

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Introduction

Rabies is one of the deadliest zoonoses, caused by the virus of the **Lyssavirus** genus (family **Rhabdoviridae**). Spreading in almost every continents of the world, with more than 150 endemic countries, rabies claims nearly 60,000 human lives annually and it is estimated that every 10 minutes, a person dies from rabies infection. Most of the human rabies victims were children, <15 years of age, in the Africa and Asia. Every year, more than 15 million people worldwide receive a post-bite vaccination. Recent increases in human rabies deaths in parts of Africa and Asia suggest that rabies is re-emerging as a serious public health issue. More than 95% of human rabies deaths resulted from exposure to infected dogs (http://www.oie.int/). Despite the almost 100% mortality, rabies is a vaccine preventable diseases. Effective vaccines, available commercially for both humans and dogs, can be used for both pre- and post-exposure prophylaxis.

Immunology of rabies vaccination

Currently available rabies vaccines can protect against all strains of the rabies viruses and provide good cross-protective immunity against the viruses in phylogroup I, but not II and III, of the **Lyssavirus**. Currently, there are inactivated vaccine, recombinant canarypox virus expressing the rabies glycoprotein available for the use in domestic animals. In addition, live vaccinia vector expressing the rabies glycoprotein (oral vaccine) is also available for certain wildlife species including raccoon and coyotes. The goal of rabies vaccination is to induce viral neutralizing antibodies (VNA) against the glycoprotein before the virus gain access to the host nervous system. Although a level of protective VNA cannot be truly established, a minimum level of 0.5 IU/ml is used as a correlate of protection. In general, protective immunity should be achieved by day 14 of a post-exposure immunization regimen, regardless of concurrent administration of rabies immunoglobulin (RIG) and irrespective of age. In vaccinated animals, an antibody titer gradually increases and peaks approximately at 28 days following initial vaccination. Unlike other vaccines, anti-rabies VNA rapidly declined within 60 days and vaccinated animals turned

seronegative (<0.5 IU/ml) by 180 days post vaccination. Therefore, veterinarians should be aware of this timeframe, when planning for booster vaccination and serological determination for pets traveling from rabies endemic zone to other countries.

In contrast to serum neutralizing antibodies, immunological memory in vaccinated pet persisted longer, possibly lifelong, and can be reactivated upon vaccination booster or natural exposure. In healthy dogs, the duration of immunity (DOI) for rabies vaccine lasted longer than 7 years and revaccination was not necessary for maintaining the immunological memory or protective immunity in animals. This information supports an extended booster interval in owned adult pets, as recommended by several veterinary professional organizations, including WSAVA. Rabies vaccine is considered a core vaccine for rabies endemic countries. The 2016 WSAVA vaccination guidelines recommended to give one dose of rabies vaccine to puppies at 12 weeks of age. In high-risk areas, a second dose may be given at 2-4 weeks later. For initial adult vaccination, a single dose is recommended. Revaccination (booster) should be given at 1 year of age. Subsequently, the time of following booster is determined by the vaccine licensed DOI, and country's statute. In supporting the biological relevance of immunological memory, dogs and cats with an out-of-date rabies vaccination exhibited similar anamnestic responses to the animals with regular vaccine status, regardless of the time since last vaccination or numbers of previously received rabies vaccines. Thus, post-exposure management of any previously vaccinated dogs or cats, regardless of vaccination status (regular vs overdue), should be similar. The updated protocol for post-exposure management in rabies-exposed animals is available from the provided reference.

Factor affecting the effectiveness of rabies vaccination and control in the endemic area

Several factors, including animal size, age, breed, blood sampling time and vaccine (company, batch), significantly affect the levels of antibody responses in vaccinated dogs. Young (<1yr) and old (>7yr) dogs usually had lower antibody responses to rabies vaccination. Although maternal-derived immunity (MDA) has been one of the major interfering factors for vaccination in young animals, the interfering effect of MDA on rabies vaccination is not prominent. By 3 months of age, the level of MDA should have decreased, and active immunization will succeed in most puppies. In addition, host health and immunological status, proper vaccine handling, and delivery techniques are all critical for effective rabies immunization. Successful rabies vaccination and control program depend on both vaccine efficacy and coverage. Neither of these will reach 100%. In the endemic areas, achieving 70% herd immunity against rabies in the dog population will be crucial for controlling disease transmission and elimination of dog-mediated human rabies. Continuing education of health and veterinary professionals in rabies prevention and control, strengthening of public awareness and community engagement are essential for successful rabies control.

Rabies control and eradication require multi-sectoral coordination and collaboration. In 2015, the WHO-FAO-OIE have set the tripartite collaboration with a goal for elimination of the dog-mediated human rabies by 2030. The global frameworks have been agreed and now being implemented. Success will depend on implementation of the five pillars of rabies elimination (STOP-R): socio-cultural, technical, organizational and political approaches.

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SVA EXOTICS

TIPS FOR DESEXING RABBITS AND GUINEA PIGS

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Desexing of rabbits and guinea pigs is a commonly requested procedure in exotic animal practice. Both the anaesthesia and the surgical procedures in these species are complicated by differences in anatomy and response to anaesthesia and surgery. This paper reviews the surgical anatomy of these species, anaesthetic considerations, surgical procedures and common complications.

Reasons and age for desexing

Desexing in rabbits in guinea pigs is performed for three main reasons:

- To control reproduction

    - Rabbits reach sexual maturity at 4-8 months of age (depending on their size: small breeds mature at 4-5 months, medium breeds at 5-6 months and large breeds at 5-8 months. Each doe can have up to 10 litters a year, each litter with 4-10 kits.

    - Guinea pigs reach sexual maturity at 2–3 months of age. Each sow can have numerous litters each year, each with anywhere between 1-13 offspring

- To prevent disease

    - Entire rabbit does have a high incidence of uterine adenocarcinoma, the most common neoplasm in rabbits. The study by Greene (1959) showed that 4% of does had uterine cancer at age of 2-3 years, rising to 60-80% at 5-6 years. Whether the rabbit has had a litter does not appear to affect this incidence; the incidence increases with age.

    - Entire guinea pig sows have a high incidence of cystic ovaries. Although the majority of these cysts are non-functional rete ovarii cysts, they can have a spacy-occupying mass effect. Functional cysts frequently cause hormonal alopecia and uterine changes

    - Male rabbits and guinea pigs do not appear to have any significant sex-related medical problems

- To prevent or manage behavioural reasons

    - Entire rabbits of both sexes often become territorial as they mature and dis-play aggression towards other rabbits, other animals and even their owners

    - Entire male rabbits housed alone may have marked sexual behaviours, mating with other animals and even inanimate objects

    - Entire guinea pigs of either sex may develop similar behavioural issues, although not as marked as rabbits

    - Desexed rabbits and guinea pigs of both sexes are usually more docile, less destructive, and are better 'pet quality' animals

Rabbits are generally desexed at 3-6 months of age. It can be difficult to determine gender if the testicles have not descended, which occurs around 12 weeks of age – it is therefore worth waiting until the animal can be accurately sexed. Guinea pigs are easier to sex at an earlier age, and are often desexed at around 6-8 weeks of age.

Surgical Anatomy

The ovaries of rabbits are not located in a true ovarian bursa but are usually surrounded by fat within the mesovarium and the mesosalpinx. The uterus of young rabbits is found just dorsal to the bladder, coiled in the caudal abdomen. In older rabbits, the cervix is dorsal to the bladder, but the horns extend laterally. The uterus and ovaries are generally easy to exteriorize; however, they are more fragile than those of dogs and cats. The uterus is bicornuate and each horn has its own cervix. There is no distinct uterine body. The mesometrium of rabbits is a site of fat storage. In obese rabbits this can make surgery more challenging as it is often difficult to definitively identify the ovarian and uterine vessels for ligation. The vaginal body is a long, large, and flaccid unpaired organ, and the urethra opens into the ventral aspect of the vaginal body. This marks the division between the vestibulum, which is caudal to the urethral opening, and the larger true vaginal body, which is cranial to the urethral opening.

The testicles of rabbits are elongated, not round, and they move freely between the abdomen and the scrotum through the function of a well-developed cremaster muscle. The epididymis is located at the caudal pole of the testicle, but it is not as developed as in guinea pigs. The inguinal canal is open in rabbits; however, the intestine does not usually herniate because of the large epididymal fat pad which fills the inguinal canal when the testes are within the scrotum and the inguinal fat pads within the abdomen[2]. The proper ligament of the testis which attaches the tunica vaginalis to the scrotum is quite strong. The position of the testicles at any given time depends on many factors including body position, body temperature, breeding activity, gastrointestinal tract

filling, and the amount of abdominal fat. The position of the penis caudal to testicles makes a pre-scrotal approach with a single incision on the midline possible.[3]

Guinea pig sows possess paired ovaries and a bicornuate uterus, consisting of paired uterine horns, a short uterine body, and a single cervical canal which opens into the vagina[2, 3]. The ovaries are located in a craniodorsal position, immediately caudal to the kidneys.

Mature male guinea pigs possess paired, large, ovoid testes with a well-developed epididymis, located on either side of the perineum in distinct, shallow scrotal sacs, often with a large amount of surrounding fat. The testicles are usually fully descended into the scrotum by 3 months. If the testes have not descended by 4 months of age, the guinea pig should be considered cryptorchid. The wide inguinal canals remain open for life. Reproductive glands, located within the abdomen, include the seminal vesicles (vesicular glands), coagulating glands, bulbourethral glands, and the prostate.

Analgesia and anaesthesia

Both rabbits and guinea pigs have low pain tolerance thresholds, often responding to pain by inactivity and inappetence. Both of these may, if untreated, result in intestinal ileus which may in turn lead to the death of the patient. It is therefore important that a sound analgesic plan is developed to manage both surgical and post-operative pain. The use of opioids, non-steroidal anti-inflammatory drugs and local anaesthesia can be combined to produce multimodal analgesia. It is important that analgesia be continued for several days to ensure the patient is comfortable and eating well.

There are several factors to consider when formulating an anaesthetic plan

- Their small body size

    - Large surface area to body mass ratio can lead to rapid temperature drops

    - Rapid metabolic rate means that there is a high demand for oxygen and energy

    - This metabolic rate affects the dose rate and frequency of many drugs

- They are obligate nasal breathers

- Intubation can be difficult

- A rapid recovery with minimal 'hangover' effects is needed to ensure a rapid return to eating, minimising the risk of ileus

- Rabbits, in particular, can have a catastrophic re-lease of catecholamines when stressed, leading to cardiac arrhythmias and arrest

- They cannot vomit

Anaesthetic plans should therefore include the following:

1. Minimal / no fasting

2. Washing the mouth out before anaesthetic induction

3. Pre-medication should include analgesia and, if possible, be reversible

4. Induction can be given IM, IV or via mask. In rabbits, mask induction should only be used in a well-sedated and calm rabbit

5. Ventilation support can be given via a well-sealed face mask (guinea pigs, small rabbits), endotracheal tube (rabbits), or laryngeal mask (rabbits)

6. Pre- and post-oxygenation of the patient is essential

7. Patients should be encouraged to eat as soon as possible after recovery

Orchidectomy

Male rabbits and guinea pigs can be castrated via either an open or closed technique. The initial incision may either be scrotal (a 1-1.5 cm incision through the scrotum longitudinally on each side of the midline about midway along the length of the scrotum) or a single pre-scrotal incision

Closed: The tunic is grasped and the testicle is removed from the scrotum with the tunic intact. The tunic is tightly adhered to the end of the scrotum by the proper ligament of the testis. This ligament must be broken down to allow exteriorization of the testicle. Caudal traction is applied to the testicle and dry gauze is used to strip the facial attachments allowing the narrow portion of the cord to be exteriorized. Once the testicle has been exteriorized adequately the cord is ligated using a 2 or 3 clamp technique.[2]

Open: The vaginal tunic is incised to allow exteriorization of the testicle, spermatic cord, and vascular supply. The tail of the epididymis will still be attached to the tunic. This attachment must be broken down freeing the testicle for removal. The spermatic cord is double ligated and the testicle is removed. The vascular pedicle is traced cranially and the inguinal canal is identified. A single interrupted suture is placed across the inguinal canal being careful not to compress the blood vessels passing through the canal. The vascular pedicle is ligated prior to transection. (It is not necessary to pull the testicle out far from the body, risking accidentally tearing the vessels. The surgeon only needs the entire testicle exposed and the vessels can be ligated close to the testicle.) Once transected (or torn) the vascular pedicle retracts

into the retroperitoneal space as the testicular vessels are branches off the renal vessels. Haemorrhage from these vessels, therefore, occurs in the retroperitoneal space and does not cause haemoabdomen. Inadequate control of subcutaneous vessels and vessels within the tunics are the likely causes of scrotal hematomas, not haemorrhage from the testicular vessels.[2]

A modification of this technique in rabbits involves an open castration being careful to remove only the testicle and leaving the epididymal fat pad intact. The fat pad will then prevent herniation of intestine through the inguinal ring.[2]

With any of these techniques the scrotal incision may be left open to heal by second intention or it may be sutured closed using either an intradermal pattern, tissue adhesive, or skin staples.

Ovariohysterectomy

Rabbits: A 2-3 cm incision is made starting midway between the umbilicus and pubis extending caudally. The cecum and bladder may be directly under the linea alba and it is recommended that the body wall be elevated from the abdominal structures prior to making the initial incision in the linea alba. Once the peritoneal cavity is opened the viscera will drop away as air enters the peritoneal cavity. The uterus is usually visible dorsal to (under) the cranial pole of the bladder. The uterine horn is usually redder in colour than surrounding viscera, making it easily identifiable. One uterine horn may be lifted through the incision using atraumatic forceps. (It is best to avoid using a spay hook as such an instrument may perforate the cecum leading to disastrous consequences.) Once the uterine horn has been elevated through the incision it is traced to the ovary which is loosely attached to the dorsal body wall by a long, fat-filled mesovarium. The oviduct is usually visualized as a fine tubular structure which literally encircles the ovary. A clamp may be placed between the ovary and the uterine horn to allow traction to be applied to the ovary. The ovarian ligament does not usually need to be broken down. There are many vessels that supply the ovary within the fat of the mesovarium. An opening is created by blunt dissection through the fat of the mesovarium and a ligature is passed around the portion of the mesovarium containing the vessels supplying the ovary. As the suture is tightened it will cut through the fat, but will ligate the blood vessels. This procedure is repeated on the contralateral side and the fat-filled broad ligament of the uterus may be broken down by gentle blunt dissection. Any large vessels or any haemorrhage from vessels within the broken ligament may be controlled by ligation or haemostatic clips. Following dissection of both uterine horns the uterus may be ligated on either the cranial or the caudal side of the cervix. The uterine vessels lay on each side of the uterus several millimetres lateral to the uterus. It is best to

ligate these vessels individually and place a transfixation ligature around the uterus prior to transection. Closure is routine with body wall, subcutaneous tissue, and skin being closed as separate layers.[2]

Guinea pigs: Two approaches can be used: a ventral midline approach as described above, or a bilateral flank approach. The latter approach is particularly useful in young sows as it minimises the risk of handling the intestinal tract. The 1cm skin incision starts where the last rib passes under the lumbar muscles, and is directed at a 45° angle caudoventrally (the author directs it towards the stifle). The subcutaneous fat is separated (or removed) and the muscle wall is incised along the same line as the skin incision. Peritoneal fat comes into the incision, and when exteriorised gently with atraumatic forceps, it contains the ovary and/or fallopian tube. The ovarian blood vessels, between the ovary and the kidney, are ligated and transected and the fallopian tube followed caudally to the uterine horn. As much horn as possible is exteriorised, ligated and transected. The muscle, fat and skin are closed in separate layers and the procedure repeated on the other flank. If needed, the remnants of the uterine horn and the uterine body can be removed via a small ventral midline incision.

Post-operative care

Post-operative complications include:

- Pain (see the earlier discussion on analgesia)

- Ileus occurs when the animal is inappetant, dehydrated, in pain, or all of these. Animals should be well hydrated before and after the surgery, fasting should be minimised, analgesia provided and, if the patient in not eating, it should be assist fed with a suitable diet (e.g. Critical Care®, Oxbow), Animals should not be discharged after surgery until they have been seen to eat and defecate.

- Infection is most common in guinea pig castrations, with abscess formation under the skin incision occurring several days after the procedure. Post-operative antibiotic therapy should be considered, and the owner advised to watch for swelling at the surgery site

- Scrotal herniation can occur in both rabbit and guinea pig castrations, and the owners should be advised to monitor the site for swelling.

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IMPROVE SEIZURE CONTROL WITH INTEGRATIVE MEDICINE

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Introduction

Integrative Medicine (IM) has become one of the most frequently requested medicine to be used by itself or in conjunction with conventional medicine or other treatments to treat a variety of challenging illnesses, including pain, seizures, Cushing's disease, skin problems, and cancer. Practitioners of IM use many different modalities, including acupuncture, Chinese herbal medicine, supplements, massage, and dietary therapy to treat or prevent health problems, improve outcomes, and enhance quality of life.[1]

Seizure disorder or epilepsy is one of the most challenging neurological conditions affecting pets and represents a significant number of referrals to veterinary neurologists. It is estimated that 1% of the canine population has some form of seizure disorder.[2] The incidence of idiopathic (inherited) epilepsy in certain breeds of dog can be as high as 15% to 20%.[2]

To date, there is no cure nor ideal treatment for epilepsy. While antiepileptic drugs (AEDs), such as diazepam, midazolam, phenobarbital and potassium bromide (KBr), can be very helpful in the control of seizure activity, they reduce the clinical signs but do not treat the cause[2], and not all treatments provide absolute control. Approximately 20% to 40% of epileptic dogs may become refractory to phenobarbital and KBr.[3] In addition, some animals are less tolerant of their side effects, which include lethargy, polyuria/polydipsia, polyphagia, vomiting, sedation, and weight gain (phenobarbital).[4] Although these newer drugs, such as levetiracetam, zonisamide, felbamate, topiramate, gabapentin, and pregabalin, have gained considerable popularity in the management of epilepsy, scientific data on their safety and efficacy are very limited and cost is often prohibitive.

Traditional Chinese Veterinary Medicine (TCVM)

Regardless of the causes of epilepsy, TCVM is an effective treatment to help complement current medications and improve seizure management. Indications for TCVM therapies include side effects caused by AEDs, refractory seizures, and quality of life of the patient.

- A number of published studies demonstrate the anti-epileptic effects of **acupuncture** as an adjunctive treatment for seizures in animal models and humans.[5-13] Different modalities of acupuncture have been used to treat seizures, including needle insertion,[5,10] electrostimulation,[14] scalp acupuncture,[12] auricular acupuncture,[6,7,15], and gold bead/wire implants on acupuncture points.[8,11] Accumulating data have showed that acupuncture may have an effect on epilepsy by increasing the release of inhibitory neurotrans-mitters, such as serotonin, GABA, nitric oxide, or opioid peptides.[15]

- **Herbal medicine** is another major component of TCVM and has been advocated as an adjunctive therapy in seizure control, usually in conjunction with acupuncture.[16] TCVM practitioners usually prescribe combinations of herbal medicines. The most frequently used Chinese herbal medicine in the management of seizures is **Di Tan Tang** (Chinese herbal equivalent of phenobarbital). The author uses 0.5g per 10-20 pounds q12h. It contains **Uncaria**, **Arisaemi**, **Acorus**, **Poria** and **Glycyrrhiza**, which have been shown to possess anti-epileptic activity in animal models.[17] **Nux vomica**, **Illicium henryi**, betelnut and mulberry are only a few herbals that should be avoided as they have been found to induce seizures.[18]

- Form the TCVM standpoint, pattern differenti-ation (Diagnosis) is important for the treatment strategy for seizures. Selections of acupoints and herbal formulas are based on the pattern differentiation of the patient.

The TCVM philosophy of seizures

The philosophy of disease treatment in TCVM differs from that of Western medicine. TCVM treats the individual, not the disease. From the TCVM standpoint, seizure is caused by "internal Wind" invading the channels of Liver due to Heat generated by the Liver (known as Liver Yang rising). The metaphor of "Wind" implies the shaking of tree leaves in a strong breeze, which resembles seizure activity. The Heart and Kidneys are also involved in seizures. The Kidney, in TCVM, is Water. Water nourishes Wood (Liver) and hinders Fire (Heart), so if the Kidneys are out of balance, it could influence the Liver or Heart imbalances that trigger seizures.

TCVM treatment for seizures involves calming the Liver, eliminating Wind, calming the Mind, clearing Phlegm, and restoring consciousness (see table).[1] It is also important to balance the Qi, Blood, Yin and Yang if they are involved. Acupuncture can be given once every two to four weeks for five to eight sessions initially, along with Chinese herbal medicine. After that, the treatments can be spaced out to once every three to six months for maintenance. Once the seizures are under control, you can gradually reduce the dosage of phenobarbital, potassium bromide, or other AEDs to the lowest effective dose (one at a time). Gold bead or wire implant can be

considered for refractory epilepsy as a last resort. In one study, nine of 15 dogs had at least a 50% reduction in seizure frequency after gold wire implantation in acupoints.[8] Acupuncture and herbal medicine should be tried before permanent materials are implanted, since metal implants may interfere with future MRI testing.

Dietary Supplements

1. **Omega 3 Fatty acids** had reported a significant positive association between omega-3 fatty acids (EPA and DHA) and epileptic seizures in reducing the frequency of seizures in human patients.[19] The author doses 1,000-1,500 mg daily per 1,000 kcal of food intake daily.

2. **Thiamine (vitamin B1)** can be considered as an add-on treatment in deficient and non-deficient thiamine epileptic patients, and might improve attention and other mental abilities in people with epilepsy.[20]

3. **Vitamin E:** Co-administration of Vitamin E 400 IU/day with antiepileptics for 6 months has shown to improve seizure control and reduces oxidative stress in a double-blind, placebo-controlled trial.[21]

4. **S-adenosyl methionine (SAMe)** and **milk thistle (Silybum marianum)** provide hepatocellular protection by stabilizing hepatic cell membranes. SAMe (15 to 20 mg/kg q24h) and/or milk thistle extract (5 to10 mg/kg q24h) or silybin (1-2 mg/kg q24h) to prevent liver damage from AED.

5. < > has been shown to be effective in both experimental models and patients suffering from epilepsy. It is also potent antioxidant enzymes scavenging oxygen free radicals.[22] The author commonly supplements 3-5 mg orally before bed time.

   Antioxidant levels like catalase, glutathione peroxidase (GPx), vitamin E, glutathione (GSH), thiol group (SH), uric acid, and total antioxidant capacity (TAC), were found significantly low levels of antioxidant in epileptic patients as compared to controls. AED did not influence the antioxidant status suggesting that seizures induce oxidative stress.[23]

6. **Hemp-based cannabidiol (CBD) oil** is an extract from industrial hemp plants that contains mainly non-psychoactive CBD with minimal to no psychoactive THC.. Emerging data support its use as a therapeutic option for refractory epilepsy in humans.[24]

7. **Huperzine A** is a compound isolated from Chinese club moss **Huperzia serrata**, and is available as an over-the-counter supplement to enhance memory. It has been shown to have anti-seizure action in animal models.[25] Huperzine A is given orally (1 µg/kg q8-12h).

Nutrition Therapy

A ketogenic diet, a diet that is high in fat and low in protein and carbohydrates (typically with ratios of up to 4:1 fats to proteins and carbohydrates), has showed some promising results in controlling the frequency of seizures in children. In two canine epilepsy studies, ketogenic diet (5.5 % MCT. MCT content was about 10 % of the total formula calories), when added to standard AED treatment, was associated with a lower seizure frequency and reduced some ADHD-like behaviors, compared to the placebo diet.[27,28]

Summary

Integrative Medicine may prove to be an excellent adjuvant to conventional therapy in the treatment of seizures in animals, especially those with poorly controlled seizures. In mild cases, Integrative therapies, especially TCVM can be used on its own to help prevent and minimize the occurrence of further seizures. It may reduce the requirement for anti-epileptic medication. Nevertheless, there is a need for evidence-based research in the study of integrative therapies for managing seizures in animals. A pet owner looking at integrative medicine for epilepsy should ensure their pet is treated by a veterinarian specialized in integrative medicine, in addition to having the animal evaluated by a veterinarian or a veterinary neurologist.

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Common acupuncture points for seizures

Action	Acupoint	Description
Soothe Liver and calm Liver Yang	BL-18	Back-shu association point for the liver
	BL-19	Back-shu association point for the gallbladder
	LIV-3	Shu-stream (earth) of the LIV channel; subdues Liver Yang
	TH-5	Subdues Liver Yang
	Er-jian	Clear Heat and subdues Yang; hemoacupuncture this point

Extinguish Wind	GV-14	Extinguishes interior Wind
	GV-16	Extinguishes both interior and exterior Wind
	GB-20	Extinguishes Wind and subdues Yang
	BL-17	Influential point for Blood; activates Blood to dispel Wind
	SP-10	Sea of Blood; activates Blood to dispel Wind
	Da-feng-men	Classical point for Wind pattern
Calm the Shen	GV-20	Calms Mind, extinguishes interior Wind, subdues Liver Yang
	HT-7	Calms Mind and opens the Mind's orifices
	An-shen	Classical point for calming the Mind
	Nao-shu	Brain association point for calming the brain
Transform Phlegm	ST-40	Resolves Phlegm and opens the Mind's orifices
Special point	GV-1	Opens Mind's orifices and extinguishes interior Wind
During seizure	GV-26	Opens Mind's orifices, extinguishes interior Wind, promotes consciousness

Notes: Additional acupoints based on pattern diagnoses as follows:

1. Wind-Phlegm: BL-20, BL-21, ST-36

2. Phlegm-Fire: LIV-2, ST-36, Wei-jian

3. Blood Stagnation: LI-4, GB-41

4. Yin-Blood Deficiency: BL-23, LIV-8, SP-6, KID-3

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INJECTABLE AND ORAL TRAMADOL FOR PAIN CONTROL

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Tramadol, a synthetic racemic mixture of the 4-phenyl-piperidine analogue of codeine, has received widespread acceptance in human medicine since it was first introduced in 1977 in Germany. Its efficacy is attributed to a dual mechanism of action, namely, the interaction with µ opioid receptors and the monoaminergic effect on spinal pain modulation through inhibition of the reuptake of norepinephrine and serotonin. Tramadol does not have δ or κ opioid receptor affinity but its affinity for µ receptors is approximately 10 times less than codeine and 6000 times less than morphine. The (+) enantiomer of tramadol has a low affinity for µ receptors, inhibits the cellular reuptake of serotonin (5-hydroxytryptamine, 5-HT) and increases its extracellular release. The (-) enantiomer more effectively inhibits norepinephrine reuptake and increases its cellular release. Inhibition of norepinephrine reuptake leads to activation of the descending pain inhibitory system, causing inhibition of the transmission of painful stimuli through the dorsal horn of the spinal cord via endogenous opioids. The opioid receptor-associated analgesic efficacy of tramadol depends on the ability of the individual to convert the drug by P450 enzymes in the liver to the main active M1 metabolite, O-desmethyltramadol (ODM), with 86% to 100% of the drug and its derivatives being excreted through the renal system. In humans, some individuals are unable to convert tramadol to its metabolites efficiently due to a P450 enzyme deficiency. The inability to produce tramadol metabolites results in decreased serum levels concentrations of ODM and consequently a significantly reduced analgesic effect of the medication. Dogs have a low capacity to produce this active metabolite, which might be breed or individual dependent.[1-3] Therefore dogs are not expected to have substantial opioid effects after tramadol administration. Repeated doses of tramadol either decreased drug absorption or enhanced presystemic metabolism of tramadol in dogs, in which a 60% to 70% decrease in tramadol plasma concentrations resulted after just eight days of treatment (20 mg/kg by mouth).[4] The effects of multiple-day administration on the tramadol metabolites has not been reported. In contrast with dogs, cats produce high concentrations of ODM after tramadol administration and as a result have prominent opioid effects.[5] The concentrations of ODM after 5.2 mg/kg tramadol by mouth were 10 times higher than ODM concentrations in humans after 100 mg by mouth. The terminal half-life of ODM after

oral tramadol in cats was 4.5 hours, suggesting that administration every 12 hours may be appropriate in cats. The plasma concentrations of tramadol and ODM were dose proportional from 0.5 to 4 mg/kg by mouth.[6] The pharmacokinetics of repeated doses of tramadol have not been reported in cats.

ANALGESIC EFFICACY

Acute perioperative pain

In several clinical trials, tramadol has been shown to provide good perioperative analgesic efficacy in dogs. [7-10] However, there is limited published information evaluating the analgesic effect of tramadol using standardized nociceptive stimulation methods in this species. Intravenous tramadol has been shown not to evoke effective acute cutaneous antinociception in laboratory Beagle dogs, therefore its use in acute nociceptive pain has been questioned.[11] Oral tramadol administration yielded antinociceptive effects in Greyhounds, but plasma concentrations of tramadol and ODM were lower than expected. Compared with the approved dose (100 mg, PO) in humans, a mean dose of 9.9 mg/kg, PO resulted in similar tramadol but lower ODM plasma concentrations in Greyhounds.[3] Therefore, more studies of tramadol administration, including studies with larger numbers of dogs and multiple doses, are needed. More comparisons among dog breeds are recommended using the same analytic technique to fully characterize the metabolic pattern of tramadol.

The effects of tramadol on thermal thresholds in cats has been reported. The thermal thresholds exceeded the 95% confidence interval at 0.75, 3, and 6 hours after 1 mg/kg tramadol, but not at 1, 2, 4, 8, and 24 hours.[12] A dose titration study evaluated the effects of tramadol dosed 0.5 to 4 mg/kg by mouth in cats using a thermal threshold model. Thermal thresholds increased proportionally with increased doses.[6] The duration of increased thresholds were also related to the dose, with 2 mg/kg producing significant effects from less than 6 hours to up to 13 hours after administration, 3 mg/kg producing significant effects from 9 to 12 hours after administration, and 4 mg/kg producing significant effects from 10 to 16 hours after administration.

There are few clinical studies assessing the effects of tramadol administration on cats in controlled clinical trials. A blinded study with negative controls by Brondani et al. 2009 reported the effects of tramadol in patients after ovariohysterectomy.[13] Treatment groups included placebo, the NSAID vedaprofen, tramadol (2 mg/kg SC), and the combination of tramadol and vedaprofen. Patients were evaluated with a composite pain scale. All of the patients receiving placebo and vedaprofen received rescue analgesia, 50% of the tramadol patients received rescue analgesia, and none of the vedaprofen and tramadol group received rescue analgesia. The

composite pain scale was significantly lower for the combination of vedaprofen and tramadol from 1 to 56 hours after surgery, but not significantly lower in any of the other treatment groups for more than 1 time point compared with placebo. Another study by Cagnardie et al. (2011) found that preoperative administration of tramadol (2 mg/kg IV) to cats undergoing gonadectomy decreased the isoflurane requirement and, according to the pain scoring system used, produced sufficient postoperative analgesia.[14] These findings, together with the positive kinetic behaviour, suggest that 2 mg/kg of tramadol IV might be useful as intra and postoperative analgesic in cats undergoing gonadectomy. A study by Evangelista et al. (2014) compared the analgesic efficacy of preoperative administration of tramadol at two doses (2 mg/kg IM or 4 mg/kg IM) with pethidine (6 mg/kg IM) in cats undergoing ovariohysterectomy.[15] Tramadol provided adequate analgesia and it was more effective than pethidine to at least six hours for the studied animals. At the higher dose (4 mg/kg IM) tramadol thought to be more effective, as no rescue analgesia was required. Finding from this study were confirmed by Bayldon and Bauquier (2017), who found preoperative administration of oral tramadol (6 mg/kg) or intramuscular tramadol (4 mg/kg) provided effective analgesia for 6 hours following ovariohysterectomy surgery in cats.[16]

Chronic pain

Tramadol is used worldwide for its effects on improved physical function and good tolerability in humans with chronic osteoarthritis pain. Nevertheless, evidence of its efficacy in canine and feline osteoarthritis are scarce. In human pain medicine, tramadol and other serotonin and noradrenalin re-uptake inhibitors (SNRIs) are mainly used for the treatment of chronic pain. It seems probable that the analgesic effect of tramadol in dogs and cats with chronic pain is also likely to be mediated through a non-opioid-based mechanism. This is particularly likely to be the case with any positive analgesic findings in dogs as repetitive tramadol administration in dogs has shown that plasma ODM concentrations decrease by 60% to 70% within just one week.[3] Further investigations on long-term administration of tramadol in chronic pain models in dogs and cats could help to assess the clinical utility of tramadol in a growing population of geriatric patients with chronic pain.

There are few studies assessing the effects of tramadol administration to clinical canine patients with chronic pain in controlled clinical trials. A blinded study by Malek et al. (2012) investigated the analgesic efficacy or oral tramadol using positive and negative controls, in canine patients with osteoarthritis.[17] Significant improvement was noted in the positive control group (carprofen, 2.2 mg/kg twice a day) and tramadol (4 mg/kg 3 times a day) group compared with the placebo (administered 3 times a day). Plasma concentrations of carprofen and tramadol

were measured 3 hours after the first dose and last dose (14 day). The plasma concentrations of carprofen were within the expected plasma concentrations. The plasma concentrations of tramadol were low (39.3 _ 35.3 ng/mL) 3 hours after the first dose and were significantly decreased 3 hours after the last dose (7.1 _ 8.8 ng/mL), and not even detected in 4 of 11 dogs, again suggesting decreased bioavailability with multiple doses. In contrast, a recent randomized, blinded, placebo-controlled crossover study investigating the effectiveness of tramadol for the treatment of pain due to osteoarthritis of the elbow or stifle joint in dogs concluded that following 10 days of treatment with tramadol (administered 5 mg/kg, PO, q8h) provided no clinical benefit.[18] The most likely reason for this disparity is the method of Canine Brief Pain Inventory (CBPI) data evaluation and the definition of treatment response in each study. In the study by Malek et al. (2012), absolute change in overall CBPI score was used as the outcome measurement; however, in the study by Budsberg et al. (2018), a positive treatment response was defined as a score reduction ≥ 1 for pain severity score and ≥ 2 for pain interference score. In the study by Malek et al. (2012), overall CBPI score was the only outcome variable measured to evaluate response to tramadol administration. The data evaluation methodology utilized by Budsberg et al. (2012) is regarded by many to be the more accurate approach. [19]

Although more long-term controlled clinical studies are still needed in cats, the available evidence would suggest that tramadol maybe more efficacious for the treatment of chronic pain in this species than in dogs. A prospective, randomised, blinded, placebo-controlled, crossover study by Monteiro et al. (2017) found that treatment with tramadol increased weight-bearing, mobility and decreased central sensitisation in cats with naturally occurring osteoarthritis.[20] This study reported that tramadol therapy of up to 19 days (3 mg/kg orally) seems safe in cats, with the most common adverse side-effects being mydriasis, sedation and euphoria. These results are encouraging for promoting tramadol as a treatment for pain in osteoarthritic cats and are further supported by a 2018 published randomised controlled crossover trial by Guedes and colleagues which reported that that twice-daily oral administration of tramadol at a dosage of 2 mg/kg for five days produced detectable improvements in measures of mobility in geriatric cats, with a positive impact on cats' quality of life according to owners.[21] Clinically, because there were dose-dependent adverse events, most frequently manifested as behavioural changes, decreased appetite, and diarrhoea, additional dosage refinement may be necessary in individual cats, aiming to balance efficacy and tolerability.

Summary:
- There is growing evidence to support the use of tramadol for the treatment of pain in cats in both the perioperative period and cats with chronic osteoarthritis.
- A high degree of intersubject variability and inter-study variability has been demonstrated in the dog with respect to the pharmacokinetics and analgesic efficacy of tramadol. Due to the high degree of breed and individual variation in analgesic efficacy of tramadol in dogs it should not be utilised as a first-line or sole analgesic drug in this species.

DOSAGE

Cats: Most current recommendations for dosing in cats are 1 – 2 mg/kg PO q12h. Some suggest that some cats may only need once daily doses; others suggest going as high as 4 mg/kg. Perioperative doses of 2 mg/kg IV and 4 mg/kg IM in clinical studies are documented in cats, while doses of 3 to 4 mg/kg IV have been administered during thermal threshold studies.

Dogs: Dogs may benefit from tramadol administered 4 – 10 mg/kg PO q 6-12 h. Maximum analgesic effects may not occur immediately and may be delayed up to 14 days for chronic pain conditions such as cancer and degenerative joint disease. Long-term efficacy of tramadol (particularly opioid actions) may decrease with time. Perioperative doses of 2 mg/kg IV, are documented in dogs.

SIDE EFFECTS AND DRUG INTERACTIONS

Side effects are considered rare but may include: Transient signs of nausea, emesis, salivation, pupil constriction and panting (dogs) may occur. Cough suppression, decreased heart rate and constipation may result but should not be clinically significant. Overdose may manifest as seizures, pinpoint pupils, and mental alterations.

Potential drug interaction that should be considered: Tramadol is not compatible (or may require dose reduction) with other psychoactive drugs such as serotonin reuptake inhibitors, tricyclic antidepressants, or monoamine oxidase inhibitors. Tramadol can induce sedation when combined with amitraz, the active ingredient in many tick control products. Concurrent use of tramadol and cyproheptadine, an appetite stimulant, can reduce the effect of the tramadol. A human product called Ultracet® is available. It contains acetaminophen (paracetamol) in addition to tramadol. This product is NOT safe for cats. If discontinuing tramadol after long-term use, dose tapering is recommended.

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WSAVA GLOBAL NUTRITION

PROBIOTICS: NOT ALL CREATED EQUAL

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Probiotics – Not All Created Equal?

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Interest in the use of probiotics has continued to grow over the past 10 years. A PubMed search on "probiotics" brings up over 18,000 articles, with nearly 650 published in early 2018. With increasing knowledge of the gastrointestinal (GI) and other microbiomes there is increasing interest in probiotic use

When practitioners consider the use of probiotics, it is often for pets with chronic or acute diarrhoea or possibly as adjunct therapy for pets on antibiotics. Most commercial probiotic products are marketed for gastrointestinal disorders. Ideally the choice should be made based on scientific evidence.

Definition

A panel of the International Scientific Association for Probiotics and Prebiotic slightly modified the FAO/WHO definition of probiotics to: "live microorganisms that, when administered in adequate amounts, confer a health benefit on the host". While fermented foods have benefits, the benefits of the food could not be separated from the those of the microorganisms, so these foods are not considered probiotics. Similarly, while faecal microbiota transplantation has shown benefits, the lack of identification of the microorganisms used excluded this from the definition of probiotic other than when a defined mixture of microbes is used. The term probiotic has also been mis-used on products as diverse as mattresses, shampoos, face creams and aftershave and shampoos containing yoghurt.

Prebiotics are nutrient sources for beneficial bacteria included in a food or supplement, e.g. fructooligosaccharide or inulin. When a prebiotic is included with probiotics it is termed a synbiotic.

Core benefits

While the decision to use a probiotic should be made on evidence that the microorganisms and the amount have a benefit for the disorder treated, there is now evidence of effects which can be ascribed to some probiotic strains in general. These are strains used at a functional dose as a food or supplement and for humans include **Bifidobacterium (adolescentis, animalis, bifidum, brese and longum)** and **Lactobacillus (acidophilus, casei,**

fermentum, gasseri, johnsonni, paracasei, plantarum, rhamnosus and **salivarius).**

In humans the beneficial dose is 1 x 109 colony forming units/serving or per day. The intended benefit is supporting a healthy gut microbiota, although this benefit is not well defined. Human disorders where core effects may help include infectious diarrhoea, antibiotic-associated diarrhoea, irritable bowel syndrome, and ulcerative colitis. Mechanisms of action which may be wide spread among probiotics include resistance of pathogen colonization, competitive exclusion of pathogens, production of short chain fatty acids, regulation of intestinal transit, normalization of microbiota, and increased turnover of enterocytes. Core benefits from non-strain-specific microorganisms have not been described in veterinary patients, although there may be general benefits for the gastrointestinal tract (GIT) and the microbiome.

The microbiome

The microbiome is composed of microbes in and on the body on a mucosal or skin surface and their environment and has been termed the second genome. It is a source of genetic diversity, a modifier of disease, an essential component of immunity, and influences metabolism and modulates drug interactions.

The GIT contains tens of trillions of microbes, outnumbering host cells by ten fold. Each individual has its own unique intestinal microbiome, with variation along the GIT. The density and diversity of species increase exponentially from the stomach to highest numbers in the colon. The phyla **Actinobacteria, Bacteroides, Bififobacteria, Firmicultes, Fusobacteria, and Proteobacteria** comprise most of the organisms.

By fermenting fibre (e.g. in prebiotics), microbes produce short chain fatty acids (SCFA), including butyrate. Butyrate provides energy for colonocytes, affects the GIT barrier function, has anti-inflammatory and anti-oxidative potential, plays a regulatory role on transepithelial fluid transport, reinforces the epithelial defence barrier, modulates visceral sensitivity, intestinal motility, effects gene regulation, and has a role in the prevention and inhibition of human colorectal cancer.

Probiotics can induce microbiota changes in the large intestine, but these changes are usually minor, transient and dose dependent. High doses over prolonged periods of time are usually required to maintain viable counts of probiotic species. **Lactobacillus** spp increased from 1% to 2.5% of the total bacteria after administration of a multi-species probiotic containing several **Lactobacillus** spp. A mucosa-adherent probiotic may affect the microbiota more significantly; administration of VSL#3 to mice resulted major changes in ileal microbiota.

Acute, antibiotic and stress related associated gastroenteritis

Several studies in dogs and cats with acute gastroenteritis or idiopathic diarrhoea with multi-species probiotic, **Bifidobacterium animalis**, **Enterococcus (E) faceium** SF68, or **Lactobacillus acidophilus**, sometimes used with a prebiotic or metronidazole, have shortened duration of diarrhea or decreased incidence of signs in at risk (rescue shelter) dogs or cats.

In cats given clindamycin, those also given a multi-strain synbiotic had better appetites and were more likely to have completed the treatment due to less vomiting. The yeast **Saccharomyes boulardii** shortened the duration of diarrhoea in dogs given lincomyin and prevented diarrhoea when given concurrently.

Enterococcus faecium SF68 may also have a benefit in puppies with parvovirus enteritis, and in stress related diarrhoea in sled dogs and in kennelled dogs

Chronic diarrhea

Dogs

Dogs with chronic enteropathy (CE) or inflammatory bowel disease (IBD) may have decreased intestinal microbial diversity (dysbiosis). **Enterococcus faecium** significantly increased the faecal bacterial richness and diversity of dogs with IBD, which became more similar to healthy dogs. Supplementation with L**actobacillus** spp to dogs with food responsive diarrhoea showed beneficial effects on intestinal cytokines and microbiota, although the changes were not associated with the clinical response as the dogs had responded to a hydrolysed diet.

A probiotic containing multiple bacteria (VSL#3, now Vivomixx) given to 10 dogs with chronic IBD significantly decreased clinical scores, histological scores and CD3+ T-cell infiltration, and normalized dysbiosis.

Cats

There are fewer probiotic studies in cats with chronic diarrhoea. A synbiotic with seven microbial strains improved stool quality in cats with chronic diarrhea. **Lactobacillus and E. faecium** given to juvenile cheetahs increased body weight and improved faecal quality compared to a control group.

Other Potential Uses

Dental Disease

Dental plaque, a microbial biofilm on the tooth surface, is a main cause of dental pathology. A probiotic with **Lactobacillus acidophilus** LA-5 and **Bifidobacteria bifidum** BB-12 had **in vitro** bacteriocidal effects on pathogenic species from supragingival sites of dogs with dental disease. Topical **L brevis** CD2 in dogs reduced gingival inflammatory infiltrates.

Weight management

The gut microbiota differs between obese and lean individuals and is a potential determinant of obesity. Probiotics may affect the gut microbiota to modulate obesity. A meta-analysis of human studies on probiotics as a treatment for weight loss indicated limited efficacy for decreasing body weight and body mass index; however, a more recent meta-analysis concluded that probiotics or prebiotics (but oddly, not synbiotics) compared to placebo were associated with significant decreases in human weight and fat mass. Short-term use of **E faecium** SF68 dietary supplementation in eight cats had no effect on food intake, bodyweight, body composition or metabolic parameters in overweight and obese cats; however, longer, larger studies would be useful.

Chronic Kidney Disease (CKD)

In humans with CKD, decreased azotaemia was seen with 6-month probiotic treatment. The probiotic VSL#3 plus a renal diet in 60 dogs with CKD increased the glomerular filtration after 2 months compared to controls on just the renal diet. Similarly, in azotaemic cats, serum urea nitrogen and creatinine concentrations decreased after 60 days probiotic supplementation; although concurrent treatments varied and the relationship to quality of life or survival time was not clear. In 10 cats with naturally occurring azotaemia, synbiotic supplementation had no effect on azotaemia compared to a control with prebiotic alone, although GFR was not measured. This same synbiotic supplementation decreased serum creatinine but not serum urea of 15 azotaemic large cats (e.g. tigers, lions, etc.) after 6 months.

Calcium Oxalate Uroliths

Oxalate is eliminated through urinary excretion, forming insoluble calcium oxalate in the GIT and faecal elimination, or by oxalate degradation by gastrointestinal microorganisms. Some probiotics containing **Lactobacillus spp or Oxalobacter formigenes** degrade GI oxalate, resulting in decreased absorption and urinary excretion. The prevalence of O formigenes in faeces from dog with calcium oxalate uroliths was 25%, 50% in healthy dogs and 75% in healthy dogs of breeds not at risk for oxalate uroliths. The faeces of 86% of healthy cats had the genes for **O formigenes**, although the association with oxalate urolithiasis has not yet been explored. Further investigations need to determine whether there is a direct link between the lack of oxalate-degrading bacteria and hyperoxaluria, if absence is a risk factor for urolithiasis, and if supplementation decreases the risk.

Canine atopic dermatitis (AD)

Human and dogs with AD have a skin microbiota dysbiosis, with lower diversity of microbial populations, than healthy individuals. Whether altered microbial populations are the cause or the effect of inflammatory skin conditions is not yet known; however, the microbiome has an important role in skin health. Studies on the prevention or treatment of canine AD have had mixed results on clinical signs. One study showed a decrease in prednisolone use in dogs with AD given **Lactobacillus paracasei** K 71 compared to those on cetirizine hydrochloride.

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SVA SOFT TISSUE SURGERY

NON-HEALING WOUNDS

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Wound healing is divided into the following overlapping phases:

- Inflammation and Debridement, days 0-5

- Proliferation, days 4-12

- Remodelling and maturation, up to 12-18 months

Identification of the stage of the wound can help to guide management. The type of support that is required for the wound will vary during wound healing and therapy should be tailored appropriately. For example, in the early inflammatory phase of wound healing management practices should revolve around supporting the wound without allowing excessive maceration from wound discharges rather than attempting to reconstruction. Inflamed tissues have poorer suture holding strength and sutures is more susceptible to pull through – primary closure at this time is not always a good idea. Attempts at premature wound closure while inflammation still predominates may result in failure of the closure and wastage of the skin utilized for reconstruction. In the proliferation phase of wound healing the wound should be adequately protected, and aggressive debridement avoided (ie wet to dry bandaging or other non-selective debridement techniques should not be used). Moist wound management in these circumstances will allow wound healing without constant removal of tissue healing factors and mechanical damage to the wound bed. For the remodelling period it is important to protect the wound from excessive mechanical strain or abrasion that would overcome healing.

If a wound remains in the inflammatory phase despite appropriate wound management practices the question that should be asked is – what is perpetuating the inflammation? Biopsy of the wound should take place and bacterial or fungal culture should be performed. Less common organisms such as mycobacteria or inflammatory conditions such as canine eosinophilic granuloma complex or pythiosis should remain differentials as appropriate.

If inflammation of a wound has subsided but is not adequately progressing through the remaining steps, patient factors should be considered and investigated if not already performed. Conditions that can affect wound healing include; uraemia, hepatic disease, diabetes mellitus, hyperadrenocorticism, FIV FeLV, anaemia, hypothermia, hypotension, malnutrition, local tissue hypoxia and ischemia, bacterial colonization, repetitive trauma, presence of necrotic tissue and/or tension.

The golden wound period is defined as 6 hours from injury, theoretically within this timeframe bacterial load has not proliferated to the level of 10^5 organisms per gram of tissue. On presentation even contaminated degloving wounds can be partially closed following surgical debridement. However extensive flaps or reconstruction should not be attempted at this time as the risk of failure will be higher while inflammation is still severe.

If a wound is **necrotic** the aim of treatment will be to debride and remove as this tissue can be a host for infection and impair healing. If a wound demonstrates significant **sloughing** there is often a mixture of fibrin, serous discharge, leucocytes and bacteria. The aim in this case should be to remove the sloughing tissue and infectious load to provide a clean base for granulation. If a wound is **granulating**, there are intact capillary loops, collagen, protiens and polysaccharides. This wound should be protected such that it can serve as a base for epithelialization. If a wound is already **epithelializing** the bandaging material should aim to protect and promote maturation.

How aggressively the area is debrided is based on how accessible the region will be subsequently and the importance of the tissue. For example, if extensive muscle damage of the quadriceps is noted with in a wound that is designated for closure, aggressive debridement should take place as the muscle group is big providing some degree of redundancy. Once the wound is closed it will no longer be accessible limiting further opportunity for debridement. However, if the wound is associated with the medial aspect of the stifle a more conservative debridement could be performed to protect structures like the medial collateral ligament and joint.

The type of dressing applied to the wound and the times for bandage change remains contentious. While moist wound healing has gained significant popularity for its ability to provide selective debridement and foster a more conducive wound healing environment some still favour wet to dry or dry to dry dressings for initial management.

Wound dressing management largely revolves around the amount of wound moisture. What is the objective of the dressing? Is it t rehydrate or to absorb exudate? If a wound is too dry the bandaging should act to add moisture. If the wound is too wet moisture should be removed by absorption, retention or sequestration, or perhaps the wound should be further debrided, or an infection more comprehensively treated. Moisture retentive dressings include foams, alginates, hydrogels, hydrocolloids. Each dressing should be utilized in response to the level of moisture within the wound. Each bandage change should serve as a means of re-assessing wound progress as the requirements will

change over time as the wound progresses. The wound should also be constantly re-evaluated for options for definitive closure.

An exception to the rule of adding moisture can be in instances where there is no blood supply (in the case of extensive eschars) it may be prudent to keep the external aspect of a wound dry. If there is suspected to be viable cutaneous tissues selective debridement and use of a moist healing technique can be employed. However, in the complete absence of viable cutaneous tissues this may not speed wound healing.

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REPRODUCTION

USE OF DESLORELIN TO CONTROL REPRODUCTION IN CATS

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Introduction

Deslorelin is a long acting synthetic agonist of gonadotropin-releasing hormone (GnRH) marketed for veterinary use in most western countries as a 2.1 mg, 4.7 mg and 9.4 mg implant; the 2.1 mg implant is marketed for use in horses (Ovuplant™), but its extra-label use in dogs is rather common, while the 4.7 (Suprelorin™) and 9.4 (Suprelorin 12™) mg implants are currently marketed in Europe and Oceania for the control of fertility and aggressiveness in male dogs but their extra-label use in cats is becoming inceasingly common. This paper will review current and potential clinical applications of GnRH agonists in the control of feline reproduction and reproductive-related conditions.

Delaying puberty

Long acting GnRH agonists act by initially over-stimulating and subsequently down-regulating GnRH receptors at the pituitary gonadotropes, thereby suppressing the function of the Hypothalamic Pituitary Gonadal (HPG) axis and causing an arrest of secretion of gonadal steroids as well as their by-products. When HPG suppression occurs prior to the onset of puberty, puberal estrus is delayed for as long as the action of the GnRH agonist persists. The postponement of puberty following prepuberal administration of deslorelin has been reported in cats[1-5]. We administered a 4.7 mg deslorelin implant to 9 prepuberal domestic shorthair European queens[2:] 3/9 queens showed signs of estrus (vaginal cheratinization) one week following implantation, but estrus signs gradually subdued and did not appear again for the following 14-25 months (at an age of 21-33 months). Similar results in prepuberal queens were obtained when comparing the effect of the 4.7 mg deslorelin implant in 15 treated and 15 control 4-month old queens followed for a maximum of 18 months with physical exams and vaginal cytology[3]. Average age at puberty was 281 ± 21 and 178 ± 11 days in treated and control queens, respectively, while there was no difference in weight at the end of the study[3]. A delayed puberty may be obtained also by implanting post-natal kittens with a1.6 mg deslorelin implant, although the reported delay was only 63 ± 3 weeks[4,5], which is shorter than what was obtained by implanting queens right prior to puberty onset[1,2]. We have implanted 3 tomcats prior to puberty: although their post-GnRH serum testosterone

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increased sample to adult levels prior to implantation, penile spikes and masculine behavior were not present at study onset and never appeared until puberty which occurred in the 3 toms at an age of 19-22 months[1]. Although detailed information on onset of susceptibility to exogenous GnRH around the time of puberty is not available for dogs and cats, use of GnRH agonists can probably be regarded as a reasonably safe method to postpone puberty in cats; more data are necessary in tomcats to draw the same conclusion (although a similar effect is likely to occur).

Suppressing cyclicity in adult queens

In adult queens administration of a 4.7 mg deslorelin implant initially stimulates follicular growth and oestradiol secretion, after which no further evidence of estrus is observed for periods of 4-14 months [6] or up to 16-37 months[7,8]. The initial stimulation of follicular growth may lead to a true heat in a small percentage of implanted queens. Small estrogen increases have been observed in 50% of treated queens 5-14 months after treatment[6,8]. Deslorelin-treated queens may ovulate if bred early in the follicular phase [8]; progesterone increased significantly in all queens treated in the follicular phase until day 14 after treatment, then slowly decreased reaching basal level on day 56 post-treatment: duration of this luteal phase could be regarded as comparable to the normal, non-pregnant feline luteal phase[8]. Incidence of spontaneous ovulation at the induced heat and incidence of premature luteal failure at the induced pregnancy are unknown. More studies are needed on post-treatment luteal function in queens administered a deslorelin implant. General health and social behaviour have never shown any deviation from normality, and introduction of a male is not capable of reversing the deslorelin-induced cycle suppression[6].

The 9.4 deslorelin implant is characterized by a longer action in queens. We looked at duration of effect of the 9.4 deslorelin implant in 15 adult (6 months- 5yrs of age) queens. Based on vaginal cytology and serum P4 data induction of heat was observed in 40% of queens during the first month post-implantation. Subsequently, the first post-tretment heat was observed in 15% of queens after day 600, and by post-treatment day 720, 800 and 1090 percentages of queens in heat were 50%, 70% and 100%, respectively (unpublished data).

Inducing oestrus in adult queens

Because of the high fertility of cats, estrus induction in queens is an unusual request to veterinarians except for wild felids in captivity which may be object of germ cell conservation programs. Historically, estrus induction in cats has been performed for decades using variable protocols with Pregnant Mare Serum Gonadotropins which is however characterized by excessive ovarian stimulation and sometimes equivocal results. The occurrence of signs of heat has been repeatedly observed when treating adult queens with 4.7 or 9.4 mg deslorelin implants, albeit not in all queens and not with the same intensity. We observed signs of heat (vaginal epithelial keratinization, and in some females also vocalization or crouching) in 19/20 treated queens 3-4 days after treatment with the 4.7 mg deslorelin implant (queens were implanted irrespective of oestrous cycle stage)[7]. In another study oestrus induction was observed in 2/20 queens when a 4.7 mg deslorlein implant was administered during the follicular phase or immediately post-estrus[8]. Recently, Zambelli et al. 11 treated 13 adult queens with a 4.7 mg deslorelin implant: all queens showed vaginal epithelial keratinization, and 7/13 queens showed behavioural signs of heat[11]; three of these 7 queens were artificially inseminated and gave birth to normal litters[9]. In our 9.4 mg deslorelin study 40% of queens showed vaginal epithelial or behavioural signs of heat during the first month post-treatment (unpublished data). Deslorelin may be used to induce oestrus in adult queens. The induced oestrus is normal and fertile. As the cat is a seasonal breeder, time of the year may play a role in oestrus induction response to deslorelin.

Controlling fertility and reproductive behaviour in males

In adult tomcats, a 4.7 mg deslorelin implant is able to suppress the HPG axis leading to disappearance of serum testosterone and male urine odour. Following treatment, serum testosterone drops significantly to undetectable levels already during the second week post-treatment and does not start rising again often for one year or longer; penile spikes start disappearing at 60 days and are absent by 90 days; testicular volume decreases reaching ≤2/3 of normal volume by 7-8 months; also, food intake and body weight tend to increase in implanted cats, who often gain 10-20% of their initial body weight in about 6 months[10]. From a reproductive behaviour standpoint, roaming and mating behaviour decrease within 2 months and there is an improvement in friendliness towards humans in a high percentage of treated cats. Similarly to queens, the 9.4 deslorelin implant has a longer action in tomcats. When16 adult tomcats were implanted with the 9.4 formulation the average interval to resumption was 805 (range 750 to 850) days[12].

Deslorelin can be considered as a safe alternative to surgical castration in tomcats as it prevents them from displaying all unwanted effects of gonadal steroid secretion. However it should be underlined that – unlike in dogs – not all cats respond to deslorelin. Out of approximately 45 tomcats implanted with either the 4.7 mg or 9.4 mg deslorelin we have observed 4 cases of tomcats becoming friendlier and more affectionate with humans and other cats as well but continuing to impregnate queens for the entire duration of the

treatment period (unpublished observation). Owners and particularly breeders should be warned about this possibility. Likewise, in cats implanted with deslorelin the onset of sterility may be delayed beyond the second month post-implantation. In a group of 7 tomcats implanted with the 9.4 mg deslorelin implant semen quality actually improved during the first month and then decreased gradually during the second month; complete sterility was achieved from day 40 post-treatment onwards with one cat actually still being potentially fertile at 70 days post-implantation (capable of ejaculating fertile semen when penile spikes had disappeared and serum testosterone had already reached basal levels)[11].

Treatment of urinary incontinence in queens

Urinary incontinence is a relatively common condition in neutered bitches, with an incidence of 5-10% depending on breed and age at neutering. It is due to a deficiency of the urethral sphincter mechanism developing after gonadectomy. Treatment includes the use of alpha-adrenergic compounds (i.e. phenylpropanolamine) or short acting estrogens such as estriol[13,14]. Deslorelin also may be used as it has been shown to be effective in about 50% of cases. Urinary incontinence is anecdotally thought to be very rare or absent in cats. However, the condition was recently reported in a Norwegian Forest queen neutered 6 months prior to the onset of the condition[15]. A urethral sphincter incompetence was diagnosed and a 4.7 mg deslorelin implant was administered, which allowed full continence for a period of 15 months. Albeit a rare event, urinary incontinence may occur in the queen and deslorelin may be considered as a treatment.

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INTEGRATIVE MEDICINE (LECTURES GIVEN IN MANDARIN CHINESE)

APPLICATIONS OF THERAPEUTIC LASER IN YOUR DAILY PRACTICE

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Introduction

A variety of technologies purported to improve tissue healing or to reduce inflammation are available to the practitioners. There is considerable growth in the use of therapeutic laser in the multimodal management of the patients. The effects of laser protocols remain incompletely understood. Nevertheless, a considerable body of scientific evidence on the possible physiological mechanisms underlying such treatment is available. Such information should guide the indications and protocols for the use of therapeutic laser in practice.

Laser is an acronym for Light Amplification by Stimulated Emission of Radiation. Light by definition is electromagnetic radiation which consists of both a waveform and of particles. Therapeutic laser or low-level light therapy, meaning the application of light energy in the red or near infrared spectrum to tissues, has been extensively studied. Instead, photobiomodulation better describes the medical interactions produced by the diversity of lasers currently available. The terminology employed in laser instrumentation is derived from these characteristics:

• **Photons**: energy transmitted via light

• **Wavelength**: determines color and classification of light energy; measured in nanometers (nm)

  • Sunlight contains electromagnetic radiation with wavelengths between approximately 100 nm and 1 mm. This is further subdivided into ultraviolet light (<400 nm), visible light (400-700 nm), and infrared (700 nm to 1 mm).

  • Wavelength is the critical variable in determining laser penetration and tissue absorption.

    • Hemoglobin and melanin are known to absorb energy (photons) at wavelengths less than 600 nm. The absorbance of water increases at wavelengths greater than 1000 nm. Therefore, ideal wavelengths for therapy are between 600 and 1000 nm, with shorter wavelengths

used for superficial tissue and longer wavelengths used to penetrate deeper tissues.

- In general, wavelengths for specific conditions are as follows:

  - 904/905nm is better for pain

  - 808-860nm is better for inflammation

  - 600-700nm is best for skin lesions and antimicrobial effects

- **Spatial coherence**: the degree to which light is focused in a beam

- **Temporal coherence**: the degree to which light has a single frequency.

  - The critical difference between sunlight and laser therapy is spatial and temporal coherence, meaning that light is focused at a particular wavelength on an intended spot. Therefore, the dose is narrowly applied to a therapeutic window as dictated by the properties of light interaction in a particular tissue.

- **Irradiance**: power of laser divided by spot size; calculated as Power (W)/Area (cm^2)

- **Power Density and Light Energy**: power is measured in Watts (W), which is defined as one joule per second (W= J/cm^2). Light energy is measured in Joule (J). A joule is therefore a unit of energy required to produce one watt of power for one second (1 Joule = 1 Watt x 1 second).

  - The total energy transferred to the tissue is reflected in J/cm^2 whereas the irradiance of a laser is expressed in W/cm^2.

  - A higher-powered laser can produce more energy per unit of time, which allows it to deliver laser energy faster, as well as to cover a larger treatment area by using a larger beam diameter.

  - Power has absolutely nothing to do with depth of penetration, or targeting of specific tissues.

  - The total dose of visible or infrared light in a human spending one hour outdoors would be about 180 J/cm^2.

  - Current Dosage Recommendations

    - Acute conditions, superficial wounds, or inflammatory conditions: 1-4 Joules/cm^2.

    - Pain, chronic conditions, or

deep seated conditions: 6-10 Joules/cm^2

- Deep seated, chronic, or severe infections: 10-30 Joules/cm^2.

- A 3 X 5 card, an average man's palm, or a CD is approximately 100 cm^2. A quarter is about 5 cm^2. An "Oreo" is about 25 cm^2.

Laser Classification

The laser classification system defines the amount of power emitted; class IIIa emits (1 to 5mW output), class IIIb (5 to 500mW output), and class IV (>500mW output). Class IIIb lasers and Class IV lasers are the most commonly- encountered in veterinary practice. The efficacy of class IIIb and class IV laser therapy has been documented.[1-3] Current thought is that physiologic effects are seen with both high- and low-power settings.[2,3] Not all therapeutic lasers are created equal, so it is important for veterinarians to be educated about the science and safety of therapeutic laser application. Veterinarians must do your homework so to not be seduced by marketing materials, as well as be prepared to continue learning as new information about applications becomes available.

Is the laser really doing anything?

Therapeutic Laser is used for three main purposes:

1. To promote wound healing, tissue repair, and the prevention of tissue death;

2. To relieve inflammation and edema because of injuries or chronic diseases;

3. As an analgesic and a treatment for other neurological problems.

   These applications appear in a wide range of clinical settings, ranging from dentistry, to dermatology, to rheumatology and physiotherapy. The primary effects of photobiomodulation based on the current experimental literature include:

1. Cellular Effects:

   a. Increase of adenosine triphosphate (ATP) production

   b. Increase of cell membrane pump function

   c. Increase of cell respiration

   d. Production of reactive oxygen species

   e. Reduces the production of substance P

   f. stimulates long term production of nitric oxide

   g. decreases the formation of bradikynin, histamine, and acetylcholine

h. stimulates the production of endorphins

2. Gross Effects:

    a. Analgesia

    b. Antiinflammatory

    c. Antiedema

    d. Circulation improvement

    e. Enhanced wound healing

    f. Enhanced healing of tendons and ligaments with superior tensile strength

    g. Nerve cell damage repair

    h. Increased collagen synthesis

    i. Slow or reversed tissue degeneration

Clinical Applications

Laser therapy has been found to offer superior healing and pain relieving effects compared to other electrotherapeutic modalities, especially in the early stages of acute injuries, and for chronic problems. Laser therapy can be used to treat muscle, tendon, ligament, connective tissue, bone and skin tissue, however, excellent results are also achieved when it is used to complement other treatment modalities, such as acupuncture, electrotherapy, therapeutic ultrasounds, and physical exercises. The current research on laser therapy in veterinary medicine is limited. A recent controlled trial showed improved ambulation times following intervertebral disc disease when laser therapy was used after surgery. Most studies, however, employ varying treatment doses and laser wavelengths. Additional clinical trials with dose standardization are needed in veterinary medicine. Laser has been shown to be effective in, but not limited to, the treatment of the following indications:

780-830nm Infra-Red Wavelengths - Deep Tissue Penetration

- Sprains & strains

- Wounds and abrasions

- Hematomas

- Ligament & tendon injuries, bowed tendon

- Inflammation (joints, ears, muscles)

- Joint injuries

- Myofascial trigger points, pain points and deep-tissue acupuncture points

- Chronic & acute pain

- Non-union & small-bone fractures

- Lick granulomas

630-700nm Visible Red Wavelengths - Shallow Tissue Penetration

- Wounds & abrasions

- Superficial acupuncture points

- Mucous membranes

- Post-surgical wounds

- Inflammation (skin, wounds)

Laser therapy effects are cumulative: Response should improve with each treatment and/or duration of response should increase with each treatment until a plateau is reached or condition is resolved. **Chronic injuries** should be treated every other day initially. A good starting protocol could be 3 times week one, then twice the following week, then once a week later. **Acute injuries** can be treated 2-3 days in a row then follow to every other day and so on. As response is noted, lengthen the time between treatments gradually until condition is resolved or acceptable patient comfort is maintained. This is often achieved in 6-10 treatments on average. **In severe or chronic conditions** treat at least weekly (twice weekly would be preferred) until resolved. Once to twice monthly intervals may be adequate for maintenance. Most patients will show at least a mild positive response in 1-2 treatments. If positive response is not noticed in 3-4 treatments with standard protocol, re-evaluate condition, treatment protocol or diagnosis.

Safety and Contraindications

Laser therapy has a WIDE margin of safety. The North American Association for Laser Therapy (NAALT) has compiled the following list of contraindications: pregnancy (over the pregnant uterus), cancers (over the tumor site), hemorrhagic areas, endocrine glands, pediatric joint epiphysis, transplant patients, or other immuno-suppressed patients, and photosensitive patients. Laser can damage optic tissue. Protective eyewear should always be worn by the clinician and the patient during treatment.

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ISFM - FELINE CARDIOLOGY

A PRACTICAL APPROACH TO CATS WITH RESPIRATORY DISTRESS

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A Practical Approach to Cats with Respiratory Distress

The cat presenting with respiratory distress poses unique challenges. Not only do clinical signs often appear to occur suddenly, creating an emotional situation for owners, but the cat is often in an unstable condition where decompensation can occur because of the stress associated with handling and procedures. Intensive care and support are vital, but the number and frequency of procedures must be minimised in order to promote patient stability. The clinician must maintain a logical and calm approach, by formulating a list of differential diagnoses that is revised on an ongoing basis as diagnostic test results are revealed.

Presenting signs, initial stabilisation and historical findings

Most cats with respiratory distress present acutely, often at an inconvenient time and with little notice. We recommend that the patient is provided with supportive oxygen therapy as soon as they are known to have respiratory distress. As a result, history taking is often curtailed early on, but this can be resumed once initial stabilisation is under way. Whilst the clinician is working with the cat, a third party (possibly a veterinary nurse) should obtain a signed consent form for stabilisation and initial procedures, including thoracocentesis.

It is imperative that the dyspnoeic cat is handled minimally, in a quiet and calm environment, with adequate oxygen provision. Intravenous access or blood work is often contraindicated early in the timeline of case management. Sedation should be considered if the patient is distressed. Pharmaceuticals that dramatically alter systemic vascular resistance or promote bronchoconstriction should be avoided. The authors typically use butorphanol (0.2-0.4mg/kg IM, SC or IV if access is present), and avoid using medetomidine or ketamine because of their profound effects on systemic vascular resistance.

Observation of respiratory pattern is a very useful way of narrowing the list of possible differential diagnoses in cats with respiratory distress (Table 1). It can be performed whilst the cat is in a carrier, whilst they are receiving oxygen, and without any patient restraint which may contribute to stress. It is worth noting that a paradoxical pattern represents non-specific respiratory fatigue, although it is common in patients with pleural

space disease and diaphragmatic rupture. Respiratory fatigue is a concern, because further decompensation may be associated with respiratory failure. When clinical signs of respiratory distress in cats are accompanied by postural adaptations (for example, orthopnoea, represented by a sternal recumbency with an extended neck and abducted elbows to reduce the resistance to airflow) or persistent open mouth breathing, the situation should be considered grave and the patient highly unstable. Owners and other staff handling the patient should be cautioned, and equipment for endotracheal intubation and cardiopulmonary resuscitation should be prepared.

Once the cat is settled in a calm, oxygen rich environment, a full history can be obtained from the owner. Alternatively, this may be carried out by another veterinarian or experienced veterinary nurse whilst the primary clinician is overseeing initial stabilisation. In most cases it is appropriate for owners to be provided with reassurance that their cat is being cared for appropriately whilst they wait. In the event that an owner has left the premises, a telephone call to update them and take a history should suffice, provided that signed consent was previously obtained.

Table 1: *The observation of respiratory pattern can be used to help localise the disease and narrow the list of differential diagnoses.*

CHF, congestive heart failure

	Description	Localisation	Common differentials	Diagnostic tests to consider
Inspiratory	Long, slow inspiratory phase, often accompanied by stridor	Upper respiratory tract	Nasopharyngeal obstruction (polyp, foreign body) Laryngeal obstruction (mass lesion, paralysis)	Upper respiratory tract visualisation and imaging
Restrictive	Rapid, shallow pattern with even effort on inspiration and expiration	Pleural space, alveoli, pulmonary interstitium	Pleural fluid (effusion, haemothorax, pyothorax) Pneumothorax Pulmonary oedema (CHF)	Thoracic ultrasonography
Obstructive	Near-normal rate with disproportionate expiratory effort, often involving an expiratory abdominal push	Lower airway disease	Lower airway obstruction (chronic bronchitis, asthma)	Thoracic radiography or computed tomography
Paradoxical	Caudal thorax and cranial abdomen move in opposite directions during both phases, often fast rate	Non-specific; represents respiratory fatigue Common in pleural space disease	Pleural space disease, Pulmonary oedema (CHF), Lower respiratory tract disease Diaphragmatic rupture	Thoracic ultrasonography
Panting	Paroxysmal: open mouth, rapid, short, shallow breaths	Non-specific; may not represent true respiratory distress if respiratory pattern normal between episodes	Stress Hyperthyroidism Right-to-left shunting cardiovascular disease	Dependent on other clinical findings

As always, a thorough history and establishing a timeline of events is vital in achieving a diagnosis and formulating an appropriate treatment plan. The duration, intensity and progression of respiratory signs should be interrogated thoroughly, as should any concurrent clinical signs. Historical reports of a persistent or chronic cough should assist in identifying lower airway disease, but may also be attributable to respiratory tract neoplasia. Coughing is rare in cats with congestive heart failure (CHF), which tend to present primarily with tachypnoea. Retching or gagging is highly suggestive of nasopharyngeal or laryngeal disease, but the sounds are easily confused with a cough by owners. For this reason, we would encourage clinicians not to be shy in performing impressions of the different respiratory noises, to aid in owner differentiation and thus assist in appropriately narrowing your list of differential diagnoses. An alternative would be to record a series of videos to demonstrate the different clinical signs to owners. Syncope, collapse, episodic weakness or even evidence of partial seizures (such as facial motor activity or periods of absence) in the history of a cat presenting with respiratory distress hint at cardiac arrhythmias, and may lead the clinician to prioritise the investigation of possible heart disease if other clinical findings support this diagnosis.

Physical examination

Once a cat has stabilised and calmed somewhat, physical examination is likely to be permitted. Again, this should be performed so that stress is minimised, potentially in the kennel or ward where the patient has acclimatised (preferably a cat only area). A quiet environment is essential for accurate auscultation and temporary cessation of oxygen therapy may be necessary to facilitate this.

The minimum physical examination of a cat presenting with respiratory distress should consist of noting the respiratory rate and pattern exhibited, assessment of mucus membrane colour and capillary refill time, noting heart rate and rhythm, auscultating heart and lung sounds, performing thoracic compression and thoracic percussion, and assessment of bilateral femoral pulses. Abdominal palpation may be useful if a diaphragmatic rupture is suspected (subjective absence of a normal volume of abdominal contents is uncommon but supportive), but should be performed with care in such patients. Also, the presence of a fluid thrill on abdominal ballottement may indicate ascites, which may occur concurrently with a pleural effusion in patients with severe heart disease, neoplasia or feline infectious peritonitis (FIP).

The presence of a heart murmur in a cat is a non-specific finding. Studies performed in a non-selected population of cats in rehoming centres suggest that only 1 in 3 cats with murmurs have any identifiable echocardiographic abnormalities. However, the specificity of murmur to identify heart disease is greater if the murmur is louder (grade III-IV/VI or above) or if the cat is older (Wagner et al. 2010). In the authors' experience, some cats with

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very severe heart disease and clinical signs of CHF do not have an audible murmur. Other auscultatory findings, such as a gallop sound or an arrhythmia are far more specific for significant heart disease. As such, the findings of a loud murmur, an arrhythmia or a gallop sound in an older cat with respiratory distress are highly supportive of a cardiogenic cause. Young cats are sadly not exempt from severe heart disease, and some cats seem to develop overt hypertrophic cardiomyopathy even in the first year of life.

Pulmonary auscultation and thoracic percussion are vital tools in identification of pleural space disease. In cats with a pleural effusion, the breath sounds are expected to be reduced and percussion should sound dull in the sternal portion of the thorax. A fluid line may be identifiable by performing auscultation and percussion at different heights. In contrast, pneumothorax may be identified by an absence of breath sounds dorsally and hyper-resonant percussion in the same region.

Abnormal airway noises are useful in identifying lower airway disease. Cats with chronic bronchitis or asthma may have loud, coarse pulmonary crackles accompanied by terminal inspiratory or expiratory wheezes. In CHF, pulmonary oedema is associated with soft, subtle crackles that may be focally distributed or even inaudible. Abnormal respiratory sounds in cats with upper-respiratory tract disease are, in contrast, less subtle. Inspiratory noise (stertor/stridor) may be audible without the use of a stethoscope, and laryngeal auscultation may detect loud or high-pitched inspiratory noise.

Thoracic ultrasonography

For cats with a restrictive, paradoxical or mixed respiratory pattern (or where the clinician is uncertain as to the localisation of respiratory compromise), thoracic ultrasonography is an invaluable initial diagnostic test. Most practices have access to a basic ultrasound machine, and the identification of air or fluid using ultrasound is a straightforward skill that is easy to learn (for a thorough review, see Lisciandro 2011). Although many clinicians are tempted to perform thoracic radiography, perhaps because of a low level of confidence in their sonographic skills, the use of ultrasound has several advantages to the patient. In contrast to relocating a patient and using manual restraint techniques for radiography, ultrasound can be performed with minimal restraint, patient-side in a kennel or ward, whilst oxygen supplementation in sternal recumbency is ongoing. This constitutes the lowest-risk handling possible, whilst a lateral radiograph would be considered very high risk for a patient with respiratory compromise (Figure 1). In addition, ultrasound may be able to identify significant cardiac enlargement, a large mediastinal mass or a diaphragmatic rupture without the necessity for additional tests.

In the absence of obvious pleural fluid, identified as an anechoic space between the thoracic wall and intrathoracic structures on ultrasound, lung ultrasound can assist in further narrowing the list of differential diagnoses. Normal lung ultrasound shows a bright pleural line in the near-field, which slides back and forth as the animal breathes (known as the "glide sign"). Deep to this line are parallel "A lines", which represent the scatter of ultrasound in normal, aerated lung (Figure 2). In patients with pneumothorax, the glide sign is absent but A lines remain present, because the pleura are no longer adjacent to the thoracic wall and so cannot be visualised beyond the scatter caused by air in the pleural space. In contrast, the presence of B-lines excludes a pneumothorax (as does the normal glide). B-lines are vertical, hyperechoic artefact caused by an air-fluid interface within the pulmonary interstitium or alveoli (Figure 2). These are highly suggestive of pulmonary oedema and should raise the concern of CHF in a cat presenting with respiratory distress. Recently, a prospective study determined that in cats with dyspnoea, thoracic ultrasound to look for B-lines had the same accuracy as thoracic radiography for both experienced and novice users (Ward et al, 2017).

Figure 1: Contrasting imaging techniques used to identify a pleural effusion: ultrasonography (left) is low-risk and may identify a mass or significant cardiac enlargement, whist radiography (right) is high-risk and further information regarding a mediastinal mass or cardiac remodelling is lost because of fluid effacement of soft tissue structures.

Figure 2: Schematic diagrams (inspired by Lisciandro 2011) of lung ultrasound in cats: the normal appearance of A lines (top left) and an abnormal appearance with B lines (top right, and below on ultrasound images) which are highly suggestive of pulmonary oedema and exclude the presence of pneumothorax. B lines move back and forth during respiration and cause a "twinkling" appearance (also see Video 6). A pneumothorax is detected when the normal glide of the pleura, moving back and forth during respiration, is absent. A lines are present in both a pneumothorax and a normal lung, due to ultrasound scatter in air.

R, rib causing hypoacoustic shadow artefact

Thoracocentesis

Drainage of pleural fluid or air is indicated in cats with respiratory compromise caused by a pleural effusion or pneumothorax. It should be considered not only a diagnostic procedure but a therapeutic one. Diuretic therapy alone is not sufficient to reduce pleural fluid volume with the rapidity required to provide patient stability, and will certainly not work if the effusion is non-cardiogenic. Thoracocentesis, on the other hand, provides a rapid benefit to the patient and will be

effective regardless of the cause of the effusion. It may also be performed blind where a clinical suspicion of pleural space disease exists but ultrasound is unavailable, as a diagnostic procedure.

Thoracocentesis may be carried out in the calm conscious patient, or one lightly sedated with butorphanol (0.2-0.4mg/kg IM or SC). We recommend pre-oxygenation during equipment set-up and flow-by oxygen administration during drainage. A butterfly needle, three way tap and 10ml syringe are the equipment of choice, with a strict aseptic technique if time and patient stability permits. At the very least, hair should be clipped and surgical spirit applied with a short contact time to reduce the likelihood of contamination by skin flora.

To drain pleural effusion, ultrasound guidance may be used to select an accessible pocket of fluid and position the needle. Alternatively, needle placement blind may be achieved by using intercostal spaces 7-9, in the ventral one-third of the thorax. Slowly inserting the needle perpendicular to the skin (tolerated better than a fast movement) whilst applying gentle negative pressure will result in fluid entering the hub of the needle and extension tubing once the pleural space is reached. At this point, the butterfly wings may be used to flatten the needle against the inner aspect of the pleural, to minimise risk of trauma to the lung surface. The procedure for draining a pneumothorax is identical, but the needle should be positioned dorsally where air accumulates. Fluid should be drained as completely as possible, sampling some in EDTA and plain tubes for fluid analysis, cytology and bacteriology. Fluid analysis can help to narrow the possible list of differential diagnoses (Table 2). A fresh, air-dried smear may assist cytologists by minimising fluid preservation artefacts. Also, if possible, a second EDTA sample should be obtained for measurement of pleural fluid NTproBNP (a cardiac biomarker, see below). It is anecdotally reported that 20ml/kg pleural fluid causes clinical signs of tachypnoea and 50ml/kg is associated with severe dyspnoea. Although these figures are likely to be a crude representation of the individual patient's pathophysiology, they may be a guide as to how much fluid the clinician should expect to drain in a particular patient. For example, in a 4kg cat with orthopnoea and a large pleural effusion on ultrasound, drainage of 80ml is unlikely to resolve clinical signs. In our experience, 200-250ml could be expected in a cat with severe dyspnoea caused by a pleural effusion.

Identifying cardiac disease

Once haemothorax, pyothorax, diaphragmatic rupture and trauma are excluded, the most likely differential diagnoses for the dyspnoeic cat are cardiac disease, lower airway disease and neoplasia. Detection of a chylothorax may be associated with CHF, neoplasia or

idiopathic disease. In cats with an obstructive pattern, lower airway disease is highly likely and empirical treatment should be considered to help stabilisation and facilitate further imaging of the thorax, such as radiography or computed tomography. In patients with either a pleural effusion or a restrictive respiratory pattern, lower airway disease can be all-but excluded.

Where analysis of a pleural effusion detects a modified transudate, or thoracic ultrasound/thoracocentesis have not yielded a diagnosis in cats with a restrictive or paradoxical dyspnoea, CHF and neoplasia should be considered the most likely causes. Significant heart disease is the easiest rule-out in this scenario, and two tests should be considered: focused assessment of left atrial size and measurement of NTproBNP (N-terminal pro B-type natriuretic peptide; a hormone released by the atrial and ventricular myocardium by the stimulus of wall stretch or stress).

Subjective assessments of atrial size in cats with respiratory distress are often preferable to absolute measurements, because standard echocardiographic views in lateral recumbency are rarely safe to obtain in dyspnoeic patients. Standard images of the left atrium are best obtained from the right hemithorax, over the palpable apical impulse. It is reassuring that the dilation of the left atrium associated with CHF is rarely subtle or equivocal (Figure 3). Whilst normal left atrial to aortic root ratio (LA:Ao - usually measured in short axis where the three cusps of the aortic valve are symmetrical and clear, like a Mercedes-Benz logo) is less than 1.5, many cats with CHF have an LA:Ao ratio over 2. Where the findings of echocardiography are unclear, cardiac biomarkers offer a good alternative to identify significant heart disease, but there is an increase observed in the circulating levels associated with respiratory distress in cats, which leads to a "grey-area" in measurements (Fox et al. 2009). For this reason, it should be considered only after thorough assessment of respiratory pattern and cardiac auscultation, and attempts to assess left atrial size using ultrasound.

Total protein (g/L)	TNCC (x10e9/L)	Fluid type	Appearance	Cause	Differential diagnoses
<25	<1000	Pure transudate	Clear Transparent	Decreased oncotic pressure	Hypoalbuminaemia
25-35	1000-5000	Modified transudate	Pink or yellow tinged Transparent or slightly turbid	Increased hydrostatic pressure	CHF Lymphatic or vascular obstruction (neoplasia)
>35	>5000	Exudate	Turbid Colour associated with pathogenesis e.g. chylous, purulent, haemorrhagic	Inflammation Increased vascular permeability	Neoplasia Chylothorax Pyothorax Haemothorax Lung lobe torsion Diaphragmatic rupture (longstanding) FIP*

Table 2: *Characteristics of pleural fluid and their association*

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with common differential diagnoses

TNCC, total nucleated cell count; FIP, feline infectious peritonitis (wet form), CHF; congestive heart failure

*Please note: cats with wet FIP typically have high protein effusions, but only moderate cell counts (500-5000 x10e9/L

Figure 3: *Normal (top) and abnormal (bottom) left atrial size in the cat. The normal feline left atrium is relatively square in appearance and its size should appear to be approximately half the area of the left ventricle in long-axis and less than 1.5 times the aortic diameter in short-axis.*

LA, left atrium; LV, left ventricle; PF, pleural fluid

NTproBNP can be measured quantitatively using reference laboratories (the best validated assay is Cardiopet proBNP Feline, run by IDEXX laboratories Ltd, United Kingdom) or qualitatively using a benchtop, patient-side SNAP test (IDEXX). The patient-side test has a positive cut-off value that is useful for detecting subclinical cardiomyopathy (around 120-130 pmol/L, Machen et al. 2014). However, it has not been validated in a population of dyspnoeic cats and this relatively low cut-off may lead to a high rate of false positive results in this population. Using the quantitative assay at a reference laboratory would be more useful in cats with respiratory distress because a significant increase (>265 pmol/L) has been shown to reliably differentiate cardiac from noncardiac causes of respiratory distress (Fox et al. 2009), but the transport time and laboratory turnaround mean that results take several days and are not useful in cats with acute dyspnoea. Despite these limitations, a negative NTproBNP SNAP test is highly likely to exclude CHF as the cause of respiratory distress. A positive test is less discriminatory.

Recent research has shown that the quantitative NTproBNP assay can be reliably run using pleural fluid (stored in an EDTA tube and handled as recommended by the laboratory for blood samples) instead of plasma (Humm et al. 2013). This is hugely advantageous in cats with acute dyspnoea, because samples for laboratory submission can be achieved from therapeutic thoracocentesis instead of taking an additional blood sample from a patient where minimal handling is a priority. Although not validated, the SNAP test should have the same reliability on pleural fluid as the quantitative test (good negative predictive value, but likely to have a high rate of false positive results).

Considerations for cats with upper airway obstruction

Patients with suspected upper-respiratory tract obstruction usually require direct visual examination of the nasopharyngeal/laryngeal area, possibly with cross-sectional imaging (e.g. computed tomography) if available. Radiography may have limited value, owing to the complex bony anatomy of the area, and ultrasonography of this area requires considerable expertise and high-frequency ultrasound transducers to obtain diagnostic images. A light plane of anaesthesia induction is required to facilitate examination of the larynx, but the clinician should be prepared for endotracheal intubation or even emergency tracheostomy if significant upper airway obstruction is detected. If detected, nasopharyngeal polyps can be removed by gentle traction (described in detail elsewhere). If suspected laryngeal neoplasia is present, fine needle aspiration might prove useful – laryngeal lymphoma should exfoliate sufficiently for a confident cytological diagnosis.

Empirical or initial treatment

Thoracocentesis is the definitive emergency treatment for stabilisation of cats with a pleural effusion or pneumothorax. It has a great therapeutic and diagnostic potential so should be performed wherever indicated.

Where a convincing obstructive respiratory pattern is present on observation, lower airway disease is most likely and empirical treatment with a bronchodilator (inhaled salbutamol or systemic terbutaline) and an anti-inflammatory dose of steroids should be considered. It is worth noting that beta-agonists can promote tachycardia and arrhythmias, which may lead to deterioration of a cardiac patient, and corticosteroids can increase circulating volume and promote congestive signs. For these reasons, if the clinician is unsure of the likelihood of cardiac disease, left atrial assessment or NTproBNP measurement should be considered prior to empirical treatment for lower airway disease. Also, steroid use may lead to uncertainty in the interpretation of diagnostic bronchoalveolar lavage samples obtained at a later date, or a worsening of clinical signs if an infectious cause of respiratory signs is present (e.g. Mycoplasma spp).

If a cat with respiratory distress has an audible gallop sound, CHF is highly likely and furosemide should be administered empirically at a dose of 2mg/kg, with reassessment of respiratory rate and effort in 30 minutes. If a pleural effusion is present, thoracocentesis should be performed. Furosemide not only reduces the severity of pulmonary oedema through its diuretic effect, but has a rapid venodilatory effect if given intravenously (if circumstances and stability permits), which helps to reduce left atrial pressure and pulmonary congestion. In addition, furosemide has mild bronchodilatory effects, so cats with signs of lower airway disease should not be put at risk by a single dose and may in fact show a favourable response even when CHF is not the cause of respiratory distress. This positive response, referred to by some as a "furosemide response test", may be interpreted as diagnostic for CHF. This test should be interpreted in light of furosemide's non-cardiovascular effects and the patient's other clinical findings (e.g. respiratory pattern at presentation).

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SVA SOFT TISSUE SURGERY

TIPS TO MAKE INTESTINAL ANASTOMOSIS EASIER

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If you would like a copy of this surgical procedure on DVD go to www.videovet.org.

Key Points

- Pay attention to basic surgical principles

- Submucosa is the layer of strength

- Use synthetic absorbable suture materials

- Appositional techniques are best

- Intestinal sutures should engage at least 3 - 4 mm of submucosa

- Intestinal sutures should be no further apart than 2 - 3 mm

- Always handle bowel wall using atraumatic technique

- Examine the integrity of your anastomsis visually

- 50 - 60% of the 'small intestine' of dogs and cats can be resected

General principles of small intestinal surgery

1) Incorporation of the collagen laden submucosal layer in the surgical closure.

2) Minimize trauma and contamination.

3) Maintain good blood supply to the surgical site.

4) Avoid tension across the suture line as this may increase the possibility of leak and/or breakdown.

5) Pay attention to your established criteria when suturing intestinal defects.

Operative Considerations

1) Proper **"packing off"** of the surgical field using moistened laparotomy pads should be performed around the **exteriorized** bowell to prevent accidental abdominal contamination from intestinal contents.

2) Keep abdominal contents warm and **moist** throughout surgery with a warm, balanced electrolyte solution.

3) Handling abdominal viscera should be kept to a minimum. **Gentle manipulation** of intestine with moistened gloves or stay sutures is helpful in preventing unnecessary tissue trauma. **DeBakey** forceps are the most atraumatic forceps for handling abdominal visceral organs.

4) The collagen laden, tough **submucosa** is the layer

of strength in the small intestine; this layer must be incorporated into any small intestinal closure.

5) It may be difficult to visualize the submucosal layer due to **mucosal eversion**. Visualization of submucosa may be enhanced if everted mucosa is trimmed away.

6) Intestinal contents should be "milked" away from the anastomosis site. **Intestinal clamps** (e.g., Doyen intestinal clampS, Alice tissue forceps with a rubber feeding tube interposed, hair clips, or Penrose drains) may be used to prevent intestinal contents from contaminating the surgical site whilst manipulating intestine during anastomosis.

7) The anastomosis should be **irrigated** prior to its return to the abdominal cavity and instruments and gloves changed prior to abdominal closure.

8) **Abdominal lavage** with 2-3 liters of body temperature, sterile, physiologic saline solution should be ac¬complished prior to closure. The ob¬jectives of repeated abdominal lavage include dilution of bacteria and endotoxin and mechanical removal of fibrin and necrotic debris. The fluid of choice is body temperature, sterile, physiologic saline solution with no additives (i.e. betadine solution, chlorhexidine, antibiotics, etc). Lavage solution is poured into the abdominal cavity using a sterile stainless steel bowl, the abdominal viscera gently aggitated, and fluid and debris suctioned out with a suction device and a Poole suction tip. Injecting antimicrobials or other products into the abdominal cavity is not recommended.

Suture Material

Absorbable suture

Catgut. Catgut is NOT recommended for any visceral organ surgery. Its unpredictable absorp¬tion and rapid loss of tensile strength in such situations may result in an unacceptably high number of anastomotic leaks and / or breakdowns. Use of catgut suture in gastrointestinal surgery is not recommended.

Dexon, Polysorb, and Vicryl. Synthetic absorbable braided suture (i.e., polyglactin, poly-glycolic acid) have become very popular. The braided nature however does result in increased tissue drag and difficult knotting ability.

Biosyn and Monocryl. These sutures have similar properties to Dexon, Polysorb and Vicryl however they are monofilament. They were developed to overcome the problem of tissue drag and knot slipping found in the braided synthetic absorbables. Their predictable hydrolytic absorption is unaffected by their immediate environment (i.e., in¬fection, contam-ination, hypoproteinemia). They retain high tensile strength for a long period of time (2 3 weeks) and have very good handling characteristics. These suture materials are ideal

for use in gastrointes¬tinal surgery. These sutures are the authors choice for gastrointestinal surgery.

PDS and Maxon. PDS and Maxon, are synthetic absorbable monofilament suture materials with similar properties to that of Dexon and Vicryl. They have been shown to retain approximately 70% of their tensile strength at 3 4 weeks, and are absorbed by hydrolysis (unaffected by infection, con¬tamination, hypoproteinemia). These suture materials are ideal for use in gastrointes¬tinal surgery. Possible disadvantages include stiffness, a tendency to kink and prolonged absorbtion time.

Nonabsorbable suture

Nylon, Polypropylene. Monofilament, nonabsorbables are excellent suture materials for use in contaminated or infected surgical sites. They have a high tensile strength, are relatively inert in tissue, noncapillary, and do not act as a nidus for infection. These materials pas¬s through tissue with essentially no tissue drag and have ex¬cellent knot tying security at sizes 3 0 to 5 0.

Silk, Mersilene, Bronamid, Vetafil. Multifilament nonabsorbable sutures should **NEVER** be used in gastrointestinal surgery. They may harbor infection for years and may result in suture related abdominal abscesses or draining tracts.

Suture size

For the majority of small intestinal surgical procedures in dogs, 3 0 or 4 0 size suture material is adequate; in cats, 4-0 is recommended. The tensile strength of this size suture is greater than the tensile strength of the tissues that are being sutured (i.e., intestinal wall). Larger size suture may contribute to anas¬tomotic failure by increased trauma to tissues and its effect on the blood supply of tis¬sue margins.

Needles

Swaged-on "atraumatic" reversed cutting, narrow taper point, or fine taper cut needles can all be used for gastrointestinal surgery. The author prefers a narrow taper point needle. Needle diameter should approach the diameter of the suture.

Suture Placement

When suturing intestine, sutures should be placed 3-4 mm from the cut edge of the intestinal **serosa** and no more than 2 -3 mm apart. It is important to recognize everted mucosa and be sure the 3 - 4 mm bite in the intestinal wall is not just in mucosa but engages all layers of the intestinal wall. Measure your intestinal wall bite from the cut edge of the serosa.

Suture Patterns

There is considerable controversy regarding specific suture pat¬tern for use in small intestinal surgery. Everting, inverting, and ap¬positional suture patterns have been used experimentally and clinically for suturing enterotomies and anastomoses. Appositional patterns are recommended as they cause little lumen compromise postoperatively.

Everting: Everting patterns (i.e., horizontal mattress) have been shown to encourage adhesions and result in lumen stenosis. This technique is **NOT** recommended. The everting technique is not to be confused with the mild eversion of mucosa that occurs in the appositional techniques described below.

Inverting: In small animals adequate lumen diameter is an important consideration with any technique. Inverting patterns result in substantial lumen compromise of the small intestine and are **NOT** recommended in dogs and cats.

Apposition: Anatomic apposition of individual layers of the bowel wall (i.e., mucosa, submucosa, muscularis, and serosa) result in primary intestinal healing. This technique is superior to inverting or everting techniques because apposition of intestinal margins eliminates lumen compromise. This is the authors preferred technique for suturing all hollow viscus organs in the abdominal cavity. Suture patterns of choice include:

1) Simple interrupted apposing. This technique involves suturing **all** layers of the intestinal wall and tying the knots on top of the serosa to approximate cut edges. The sutures should be tied tight enough to effect a watertight seal, yet not so tight as to blanch the tissue and cause ischemia of intestinal margins. This technique is simple, fast, reliable, and does not result in lumen compromise.

2) Simple continuous apposing. This technique is similar to the simple interrupted appositional technique however, a continuous suture pattern is used rather than an

interrupted pattern. Advantages include faster anastomosis, equal suture tension over the entire anastomosis, airtight-watertight seal, and mucosal eversion is minimized. This is the authors preferred suture pattern for suturing all hollow viscus organs in the abdominal cavity.

INTESTINAL ANASTOMOSIS: Intestinal anastomosis is indicated for resection of nonre-ducible intussuscep¬tion, necrotic bowel wall secondary to complete intestinal obstruction, intes¬tinal volvulus, stricture secondary to trauma, linear foreign body with multiple perforations, and intestinal neoplasia (e.g., leiomyoma, leiomyosarcoma, adenocarcinoma).

After a complete abdominal exploration, the affected length of bowel is delivered from the peritoneal cavity and isolated with the use of moistened laparotomy pads and crib towels. If possible, the intestinal anastomosis should be performed on a water resistant surface (e.g., plastic drape, crib towel) to prevent 'strike' through contamination.

Once the level of resection has been determined, the appropriate mesenteric vessels are identified and ligated, and the portion of intestine to be resected is isolated by clamping the bowel at a 60° angle away from the mesenteric border. This angle ensures adequate blood supply to the antimesenteric border.

Everted mucosa: Occasionally when the segment of intestine to be removed is amputated mucosa 'everts' from the cut edge of the intestinal wall making it difficult to visualize the cut edge of the serosa. If this occurs it is 'highly' recommended to excise the everted mucosa to enable the surgeon to easily visualize the cut edge of the intestinal serosa. It is vital that the surgeon engage at least 3 – 4 mm of intestinal wall with each suture to guarantee adequate bites in the collagen laden submucosa.

Bowel lumen diameters: In cases where the oral end of the bowel is dilated and the aboral end is normal size, several options exist to create intestinal lumens of equal diameter:

1) Increase the angle of resection on the smaller diameter segment of bowel (i.e., aboral segment). This will increase the orifice size by 5-10 mm depending upon bowel diameter (e.g., dog vs cat).

2) In larger lumen size discrepancies the antimesenteric border of the smaller diameter stoma can be in¬cised longitudinally to enlarge the lumen diameter.

3) An end to side anastomosis can be performed by closing the larger diameter stoma of the in¬testinal resection with a single layer continuous apposing suture pattern then anastomosing the smaller diameter segment of bowel to an appropriate size enterotomy made in the antimesenteric border of the larger diameter segment of bowel.

4) The larger diameter segment of bowel can be made smaller in diameter by suturing its cut edge until its lumen is equal in size to the smaller diameter intestine (this technique is often used for subtotal colectomy in cats).

Intestinal Anastomosis Technique:

See the DVD for a detailed video description of this technique (www.videovet.org).

When suturing an anastomosis, atraumatic handling of bowel wall and perfect anatomic apposition of incised margins is important. It is recommended to

begin suturing at the mesenteric border as this al¬lows adequate visual¬ization of mesenteric vessels and helps prevent encircling these ves¬sels when placing the first few sutures. Any of the appositional suture patterns previously described (i.e., simple continuous or interrupted) will result in a high success rate, both in the short-term (i.e., leakage, breakdown) and long term (i.e., stricture, stenosis).

The following tips may prove helpful when performing an intestinal anastomosis (see the anastomosis video clip at www.videovet.org for detailed description of the surgery tips below:

1) First, place a stay suture to hold the mesenteric border of each segment of bowel in apposition. Tie this suture, leave the ends long, and place a hemostat on the suture end without the needle.

2) Place a second stay suture in the antimesenteric borders of each segment to be sutured to bring the ends of the intestinal segments into apposition. Place a hemostat on the ends of this suture.

3) Place gentle traction on the mesenteric and antimesenteric stay sutures to bring the two intestinal segments into apposition.

4) Using the needled segment of suture from the mesenteric stay suture, begin a simple continuous appositional anastomosis being careful to get a 3 - 4 mm bite in the submucosa and placing each suture no more than 2 - 3 mm apart (2 mm apart in cats). When the anastomosis is complete, tie the suture to the mesenteric stay suture.

5) If a simple interrupted apposing suture pattern is used, be careful to get a 3 - 4 mm bite in the submucosa and place each suture no more than 2 - 3 mm apart.

 6) Evaluate the integrity of the anastomosis. The author's preference for evaluating the integrity of anastomotic closure is to **visually** examine each suture to be certain that suture placement is no more than 2 - 3 mm apart and that each suture has a 3 mm bite in the submucosa.

Postoperative care

Intravenous fluids to maintain hydration and ensure renal function are continued postoperatively, until the patient begins to eat and drink. Intravenous fluids should then be tapered over a 24 to 48 hour period.

Systemic antibiotics are continued postoperatively for 5-7 days; 10-14 days in cases with peritonitis and/or sepsis.

Feeding: Early return to enteral feeding is best for the overall health of the intestine. Feeding the postoperative gastrointestinal surgical patient is generrally based on the following criteria:

a) preoperative condition of the patient

b) the condi¬tion of the bowel at the time of surgery

c) surgical procedure performed (i.e., enterotomy, anastomosis, pylorectomy)

d) presence or absence of peritonitis

e) postoperative condition of the patient.

The earlier patients can be returned to oral alimenation the better.

Complications

The most common postoperative complication of small intestinal surgery is leakage; leak is either associated with breakdown of the anastomosis or improper surgical technique (i.e., improper suture placement, inappropriate suture material, knot failure, sutures to far apart, inappropriate bite in the collagen laden submucosal layer, suturing nonviable bowel).

A presumptive diagnosis may be accomplished by the following:

1) Body temperature (may be up if acute or down if moribund).

2) Abdominal palpation: periodic, gentle abdominal palpation for pain (gas or fluid?).

3) General attitude (depression anorexia).

4) Incision: examination of the patients incision for drainage (look at cytology if drainage is present)

5) CBC: leukocytosis followed by leukopenia (sepsis), or a degen¬erative left shift may imply breakdown.

6) Glucose: low glucose generally implies sepsis (this occurs early in sepsis and may be used as a screening test).

7) Abdominal radiographs: generally not helpful, they are difficult to critically assess due to the presence of postoperative air and lavage fluid. It can take 1 - 3 weeks for peritoneal air to diffuse from the abdominal cavity after routine abdominal surgery. Time variation is dependant upon the amount of air remaining in the abdominal cavity postoperatively (i.e., large deep chested animal vs a small obese animal).

8) **Abdominal tap** (paracentesis): a four quadrant abdominal tap is accomplished by aspirating fluid using a 5cc syringe and 20 gauge needle or placing a plastic IV catheter into the peritoneal cavity and allowing fluid to drip onto a slide. This may be the most sensitive diagnostic test for determining the presence or absence of intestinal leak.

9) Peritoneal lavage (if paracentesis is not productive): infuse 10-20cc/kg of sterile physiologic saline solution into the ab¬dominal cavity, then gently pal¬pate the abdomen and repeat the four quadrant paracentesis. This technique increases the sensitivity of paracentesis

to 90%.

Once fluid has been obtained, a smear should be stained and evaluated microscopically. Depending upon the cell types seen, a determination of the presence of leakage can be made.

Below are examples of expected cytology in patients with and without leak.

1) Healthy PMNs with few degenerate PMNs and a moderate number of red blood cells: This cytology may be expected in any postoperative abdominal procedure (e.g., OHE, abdominal exploratory, cystotomy). Your index of suspicion for anastomotic breakdown should be low. However, if clinical signs continue to deteriorate, repeat paracentesis (2 - 3 times daily, if necessary) to determine the "trend" of the abdominal fluid cytology is recommended.

2) Healthy polymorphonuclear leukocytes with bacteria located intra or extracellularly, degenerate PMNs with intracellular bacteria, free bacteria, or food particles imply breakdown. Exploratory laparotomy is indicated.

In a recent morbidity/mortality study of patients undergoing intestinal surgery it was found that animals requiring a second abdominal surgery to treat intestinal disorders were less likely to survive than patients requiring only one laparotomy. Also, the longer it took to determine whether or not intestinal leakage had occured the less likely the patient would survive reoperation. **The take home message is:** pay attention to detail during the first surgery and if a leak occurs, diagnose it and treat it as soon as possible.

Prognosis The overall prognosis for uncomplicated GI surgery is excellent. The surgeon must pay attention to detail when suturing any hollow viscus organ with liquid contents.

WSV18-0055

WSAVA GLOBAL NUTRITION

EVERYBODY EATS: DON'T FORGET TO FEED YOUR HOSPITALIZED PATIENTS

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The basics of critical care nutrition include an initial patient assessment, the prescribing an appropriate diet whilst being mind-full of the diagnosis and disease severity, deciding on a method of delivery of that diet, setting goals for the nutritional intervention, and finally assessing whether these goals are in fact being met. The goals of critical care nutrition are to meet resting energy requirements as well as to supply sufficient essential and conditionally essential amino acids and all other micro and macro nutrient needs of that patient. A plethora of methods are available to us to achieve this, including a variety of diet and feeding tube options for the still preferred enteral nutrition route as well as multiple solution options and peripheral and central line options for parental nutrition. In some cases, partial parenteral and enteral nutrition best supplies the patient's nutritional needs.

WSV18-0060

EVERYBODY EATS: DON'T FORGET TO FEED YOUR HOSPITALIZED PATIENTS

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Chronic intestinal protein loss is a sign of failure of digestive function that may result from severe acute or chronic inflammatory lesions or from a disruption of chyle absorption and intestinal lymph flow. While the exact mechanisms leading to intestinal protein loss have not been elucidated in the dog, the three basic mechanisms defined for humans with protein-losing enteropathy (PLE) likely also apply to canine PLE. It is frequently associated with severe chronic idiopathic inflammatory enteropathies such as inflammatory bowel disease (IBD) or with idiopathic intestinal lymphangiectasia in specific breeds. Protein loss may result from: 1) erosive or ulcerative mucosal lesions causing secondary exudation of proteins; 2) lymphatic dysfunction causing leakage of protein-rich lymph into the intestinal lumen; 3) mucosal changes disturbing the mucosal barrier, causing abnormal permeability and protein leakage into the lumen; or 4) a combination of all three of the previously listed conditions. This presentation will focus on chronic intestinal disorders associated with intestinal protein loss in dogs and the dietary and medical treatments for this condition.

WSV18-0102

SERUM CREATININE CONCENTRATION: USEFUL TIPS FOR CORRECT INTERPRETATION

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Introduction

Acute kidney injury (AKI) and chronic kidney disease (CKD) are common in dogs and cats and are associated with substantial morbidity and mortality. Serum creatinine is the most commonly utilized marker for renal function, but is **neither sensitive nor specific for early detection of kidney injury.**

Limitations of serum creatinine concentration

Despite the diagnostic advancements made in other medical fields (e.g., the use of biomarkers in cardiology), serum creatinine is still being used as the marker for kidney function despite its multiple shortcomings.

The main limitation of serum creatinine include the following:

1. Serum creatinine is affected by extra-renal factors, like hydration status and muscle mass, therefore it lacks specificity. Creatinine may be increased in dehydrated animals in face of normal kidney function. In cachectic animals (common in animals with advanced CKD) creatinine concentration will overestimate kidney function.

2. Creatinine is influenced by muscle mass therefore its baseline concentration varies among dog breeds. The attempt to include all dogs under one reference range results in a very wide reference range, which decreases its sensitivity.

3. The nonlinear relationship between glomerular filtration rate (GFR) and creatinine (Fig. 1) also decreases its sensitivity since substantial changes in GFR at the early stages of the disease, are associated with only small changes in serum creatinine concentration, thus creatinine will not increase above its reference range until substantial loss in GFR has already occurred. This phenomenon is more pronounced in dogs with low baseline creatinine concentration (dogs with low muscle mass). In most dog breeds creatinine is not expected to rise above the reference range until ~75% of nephrons become non-functional.
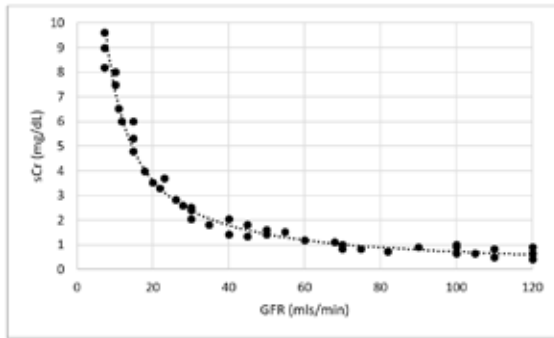
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Fig. 1: The relationship between GFR and sCr levels

4. Serum creatinine is a functional marker thus it is "blinded" to kidney injury that is not accompanied by decreased kidney function. In the liver, different markers are indicative to liver injury compared to liver function. Increased activities of alanine aminotransferase for example indicate liver injury but are not indicative of liver function. Other markers (i.e., urea, glucose, cholesterol, albumin, Bilirubin, bile acids etc.) indicate presence of liver failure. Thus the clinician is aware of the presence of liver injury and can intervene before failure ensues. Conversely, in the kidney, there are no readily available and sensitive markers of kidney injury, thus with the first indication of kidney disease, substantial decrease in kidney function has already occurred.

5. Serum creatinine does not represent the severity of the dysfunction until a steady-state has been reached. For example, if GFR drops to zero and serum creatinine is measured immediately, it will still be within the reference range. In time serum creatinine will increase but it will take days until steady state has been reached, and the severity of the injury can be accurately assessed. Consequently, substantial changes in GFR at the early stages of AKI are associated with relatively small changes in serum creatinine.

The above limitations of serum creatinine are being reflected by the findings of several studies in human medicine indicating that small, and even transient, increase in serum creatinine concentration in human patients is detrimental. In one study, as little as 0.5 mg/dL increase in serum creatinine was associated with increased in-hospital mortality (1). In another study, a transient increase in serum creatinine was also associated with increased odds ratio for in-hospital mortality (2). Finally, even a small and transient increase in serum creatinine in patients that were discharged from the hospital, was associated with the need for chronic dialysis over the ensuing three years (3). In a study of heatstroke in dogs, the median creatinine concentration was only mildly increased at presentation and third of the dogs presented with normal serum creatinine concentration (4), however, concurrent evaluation of kidney function using GFR measurement revealed that kidney function was markedly decreased. Moreover, when kidney injury was assessed using sensitive markers, presence of kidney injury was identified in 100% of the dogs (4).

The aforementioned limitations and studies imply that relying on serum creatinine as the only marker of kidney function does not provide the entire information needed to accurately assess kidney function. When relying on serum creatinine, AKI is characteristically recognized only when the disease is within the maintenance phase, when clinical signs are overt. This is one of the speculated reasons for the high mortality rate among animals with AKI, as late recognition of the disease provides only a narrow window of opportunity for therapy. In CKD, due to the wide reference range, the nonlinear relationship between GFR and serum creatinine and because the disease is initially subclinical, the diagnosis is delayed until most of nephrons are already lost.

Improving the interpretation of serum creatinine

Understating renal physiology and the limitations of serum creatinine improves interpretation of serum creatinine concentration. As oppose to the common believe that serum creatinine does not increase until 75% of GFR is decreased, any decrease in GFR is expected in be reflected by an increase in serum creatinine concentration. Thus, following small trends in serum creatinine concentration within the reference range increases its sensitivity. In fact the International Renal Interest Society defines Grade I AKI as 0.3 mg/dL (26.4µmol/L) increase in creatinine concentration compared to the baseline, even when creatinine is within the reference range. The same can be applied to animals with CKD. Once the baseline serum creatinine concentration is a known, 0.3 mg/dL increase in the same dog and under the same conditions (fasted sample, normal hydration status, same methodology) represent decrease in kidney function and presence of CKD, even if creatinine is still within the reference range. The normal baseline creatinine concentration for a specific dog should be determined in a relatively young age, when the dog is fasted and is well hydrated. Any subsequent measurements of creatinine should be related to the baseline concentration and not for the reference range of dogs.

One of the aforementioned limitations of serum creatinine relates to its variability among dog breeds (5). Therefore, increased awareness for the breed specific reference range can sensitize the clinician to elevated creatinine even if the latter is still within the reference range. For example, creatinine of 1.2 in a Yorkshire Terrier likely represents ~75% decrease in kidney function, despite the fact that it is well within the reference range of most laboratories. Conversely, a serum creatinine

concentration of 1.5 mg/dL in a dog with high muscle mass might be normal for the dog even of this value is above the reference range.

Serum creatinine should be interpreted with additional markers of kidney function. Blood urea nitrogen is a non-specific marker of kidney function, since it is influenced by multiple non renal parameters (e.g., dietary protein, gastrointestinal bleeding, catabolism, polyuria and polydipsia, dehydration, liver function), yet the urea to creatinine ratio should always be evaluated. For example, when the urea/creatinine ratio is increased, a potential reason might be decreased body mass, thus, in this case, creatinine is likely underrepresents the decrease in kidney function. Another marker that can be used in conjunction to serum creatinine, is symmetric dimethylarginine (SDMA). SDMA is a methylated form of the amino acid arginine and is primarily eliminated through the kidneys by renal filtration and excretion (6), therefore it is a potential endogenous marker of GFR. SDMA is superior to creatinine since is it not influenced by muscle mass, thus these two markers should be used in conjunction.

Summary

Serum creatinine has multiple limitations, however, understanding renal physiology and its limitations improves its' interpretation. There is a need for sensitive renal biomarkers that will facilitate the diagnosis of kidney diseases and will allow earlier intervention. Renal biomarkers that can identify kidney injury even in the absence of decreased kidney function will likely become available in the near future and have the potential to aid in the diagnosis of AKI and CKD.

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SVA EXOTICS

PEDIATRIC DEVELOPMENT OF THE PARROT CHICK

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Abstract

Parrot chicks are altricial i.e. totally dependent on their parents (or hand rearer) for warmth, food and psychological development. This paper explores the normal development of psittacine chicks and the factors that impact this development. Understanding this development leads the clinician to a better understanding of the impact of disease (and its treatment) on these chicks.

Normal development

Chicks grow at a rapid rate until they begin to wean, at which time they peak their weight and then lose weight until they reach their adult weight and plateau. The weight and age will vary between species but the growth rate, expressed as a chart (see Figure 1) remains similar.
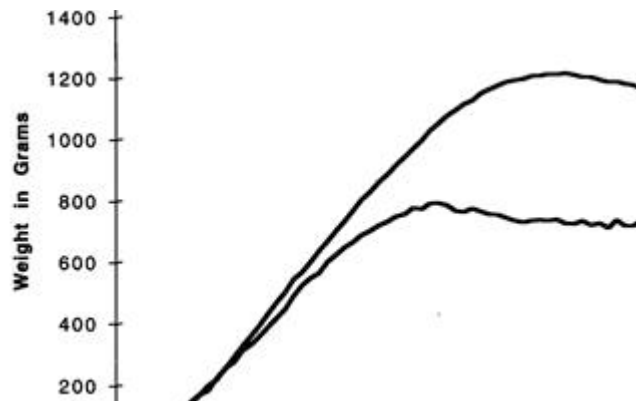
Figure 1. Growth rate graphs of two macaw species (from Abramson et al, 1996)

Prior to full feathering the normal chick's skin should be pink or pink–yellow in colour, and soft and warm to the touch. Depending on the species, some chicks are hatched naked or with down feathers, which may be white, yellow or grey in colour. A second wave of down feather growth begins at 1–3 weeks of age and, sometimes, even later in some species.

Pin feathers begin to emerge at 2–3 weeks of age. The body contour feathers emerge over the shoulders first; the pattern of emergence after that varies between species, although usually the body contour feathers emerge at the same time or shortly before the secondary

flight feathers on the wings. Primaries may begin to develop before secondary feathers, but usually mature after them. Final feather maturity is usually not complete before the bird has weaned.

The eyes begin to open at 10–28 days and take several days to open completely. Most Australian and African parrots hatch with their ears open. The ears of Eclectus and South American species should be open within 2–3 weeks after hatching.

The normal crop should have some food in it at most times. It should not be over-distended, nor should it have significant amounts of air or gas in it. Rhythmic contractions of the crop should be visible in neonatal chicks, and the crop should nearly empty in 4–6 hours in all chicks.

In neonatal chicks the abdomen should be large and convex relative to the rest of the body. The liver may be visible through the skin in very young chicks, and rhythmic contractions of the ventriculus should be visible. The duodenal loop may be visible. There should not be bruising or haemorrhage visible. As the chick grows, the abdomen reduces in size relative to the rest of the body.

Pre-weaning chicks should be either resting, sleeping or calling for food. As they get older, the chicks still spend a lot of time sleeping, but are more interested in their environment and in socializing with nursery mates. A feeding response (vigorous extension and bobbing of the head and neck) should be easily elicited by pressing gently at the commissures of the beak.

Hand rearing

Hand rearing can be done for several reasons: to increase production from rare or valuable species; when the chick is orphaned; or in an attempt to make a better socialised chick. Recent studies suggest that the latter is not always achieved by hand rearing, but many aviculturists maintain that hand reared chicks make better pets.

The timing of commencement of hand rearing varies between the owner and the individual circumstances. Some chicks, artificially incubated, are reared from the time of hatch; others are left with their parents for 2-3 weeks and are 'pulled' for hand rearing just before their eyes open, while others are taken just before fledging and are then reared until ready to wean.

The frequency of feeding is dependent on the age of the chick:

1. Hatch to 1 week – every 2 hours

2. 1 week to 3 weeks – every 4 hours

3. 3 weeks to 6 weeks – every 6-8 hours

4. 6 weeks to weaning – every 8-12 hours

A variety of quality commercial diets are available today and it is uncommon to see 'homemade' recipes. The volume fed is between 8-12% of the chick's bodyweight. The food should be mixed fresh for each feed and fed at approximately 41oC (normal body temperature for birds).

Weaning is a stressful time for both the chick and the hand rearer. The chicks will often start to refuse feeds and may regurgitate part of what is been offered, A good selection of a wide variety of foods should be available for the chicks at this time – formulated diets, vegetables and fruit – and intake of these foods monitored carefully.

Health determinants

As most companion birds are altricial (i.e. totally dependent on their parents or rearer and the environment), their health status is determined not only by their current environment, but also by the 'input' from their parents and how the eggs were incubated. We can divide these factors into three broad groups (see Table 1).

Pre-laying factors	Parental genetics become a major issue in the breeding of mutations, where closely related birds are mated to develop certain characteristics such as colour. Unfortunately, along with desirable characteristics such as new colour, undesirable physical characteristics, such as decreased body size or physical deformities, can also occur.
	The maturity of the parents will affect the size of the eggs laid, the immunity passed through the egg, and the number of eggs laid.
	The health of the parent birds can affect the chick via pathogens transmitted through the egg to the chick, and by the degree of transference on maternal immunity via the albumen and yolk.
	The diet fed to the parents, particularly the levels of calcium and vitamins, will have a direct effect on the vitality, development and health of chicks – even before hatch.
Incubation factors	Artificial vs. natural incubation: as a general rule parent-incubated eggs tend to hatch out stronger chicks. Advances in incubation techniques are, by and large, resulting in better chicks. However, skill and experience are required to match the results seen in natural incubation.
	Temperature, humidity and hygiene in the nest box or incubator will have direct effects on the development of the chick and its hatchability and strength after hatch.
	Care in handling of the eggs by either the parents or the incubator operator, as well as the frequency and degree of rotation during artificial incubation, can affect the degree of difficulty in hatching, as well as an effect on the incidence of malpositions.
Post-hatch factors	Temperature extremes can force the chick to divert resources from its growth and health towards maintaining a constant body temperature. Humidity extremes can predispose a chick to dehydration, respiratory infections and skin problems (e.g. toe constriction).
	Poor hygiene will substantially increase the concentration of pathogens (or potential pathogens) in the chick's environment. With the chick's immune system still developing, this creates a high probability of infectious disease.
	Although the era of homemade hand-rearing diets is passing, nutrition of the chick is still a major factor in the health of the chick. The practice of adding ingredients to a well-balanced formula (often based on anecdotal information from other aviculturists) can have major detrimental effects on the chick.
	Management of the nursery, in particular biosecurity and the quarantine of new chicks from different sources, will determine the likelihood of introduction of infectious diseases such as Avian Polyomavirus.

Table 1. Determinants of health for chicks

Detailed knowledge of hand-rearing practices, including weaning ages, can be obtained from reputable aviculture literature.

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Examination of the chick

History

As with any other medical case, a good history of the chick is an essential key in the diagnostic workup. Factors to consider stem from the earlier discussion on determinants of a chick's health. They include:

- The parents - their genetics, diet, maturity and health status.

- Incubation – was it artificial or natural? The hatchability of fertile eggs is a key indicator of incubation performance, and hopefully it can be found in the aviculturist's records.

- Hatching: if the eggs were artificially incubated, were there any problems with hatch?

- How is the nursery managed regarding hygiene, biosecurity, and the source of eggs and/or chicks?

- What type of food is been fed, how is it pre-pared, what volume is fed and how frequently?

- If there are any siblings or other chicks, have there been any problems or deaths within the group?

- What records does the aviculturist keep? Details on hatch dates, hatch weights, growth rates, mortalities, medications, and previous medical problems are valuable sources of information, but are sadly lacking in many cases.

Physical examination

Weight

The chick should be weighed and its weight compared with the expected weight for that age, found in growth charts (if available). All chicks will gain weight each day – it is the rate of weight gain that is more important than the actual weight. This can be monitored by expressing the chick's weight as a percentage of the expected weight for that age. If a problem exists, improvement can be seen as a gain in percentage of expected weight, rather than actual weight gain. (Figure 2)

Figure 2. Using growth charts

Posture

It is important to be aware that chicks sleep and rest in what seems to be 'awkward' positions. (For example, conure and macaw chicks often sleep on their on their backs.) These positions change as the chick moves; one should look for postures that do not change with movement.

Conformation

The positioning and conformation of the limbs and the spine should be checked. Common conformational abnormalities include a kinked (or 'wry') neck, scoliosis, kyphosis, tibiotarsal or femoral rotation, and anteroflexion of the toes.

Body condition

The toes and elbows in a well-nourished, healthy chick should be 'plump'. Thin toes and elbows are a good indicator in neonatal chicks of dehydration, malnourishment or disease. Palpation of the pectoral muscles is helpful; the soft keel bone at this age should be well fleshed with soft (but poorly developed) pectoral muscles.

Behaviour

Restlessness could indicate incorrect environmental temperature or stress (e.g. excessive lighting). Failure to elicit a feeding response can be an indication of disease, hypothermia or weakness.

Skin

Pallor of the skin can indicate hypothermia, anaemia or illness. Erythematous skin can indicate hyperthermia or illness. Heavily wrinkled skin indicates dehydration.

Crop

The normal crop should have some food in it at most times. It should not be over-distended, nor should it have significant amounts of air or gas in it. Rhythmic contractions of the crop should be visible in neonatal chicks, and the crop should nearly empty in 4–6 hours in all chicks.

Head

The size of the head should not be excessively large in relation to body size. The beak should have a normal conformation. There should be no sinus swellings. The nares should be open and symmetrical.

The eyes should be symmetrical and healthy in appearance. They begin to open at 10–28 days and take several days to open completely. Most Australian and African parrots hatch with their ears open. The ears of Eclectus and South American species should be open within 2–3 weeks after hatching.

Oral cavity

The oral cavity should be examined for diphtheritic plaques or other abnormalities. Its colour should be noted; it is normally pale pink.

Abdomen

There should not be bruising or haemorrhage visible. The abdomen can be trans-illuminated with an intense focal light for closer inspection. As the chick grows, the abdomen reduces in size relative to the rest of the body. It should be concave when palpated; a convex abdomen could indicate a degree of abdominal distension.

Feather growth

Abnormalities include:
- Feathers erupting in an unusual pattern (e.g. in a circular pattern on the crown of the head, rather than running parallel along the line of the body).
- Stress bars in the opened vane.
- Abnormal colouring.
- Haemorrhage in the calamus.
- Dystrophic development.

Droppings

The faecal portion should be relatively well formed, light brown in colour, and not malodorous. A degree of polyuria is normal (especially in hand-reared chicks), but this should lessen as the chick ages. Excessive or persistent polyuria warrants further investigation.

Diagnostic testing

Microbiology is an important tool in assessing gastrointestinal flora. Gram stains and cultures are frequently used to assess crop or other gastrointestinal problems. Normal bacterial flora includes **Lactobacillus, Streptococcus, Staphylococcus** and **Bacillus** spp. Low numbers of **E. coli** are often normally cultured as well. Other gram-negative bacilli and **Candida** are rarely cultured from healthy chicks.

Clinical pathology can be used readily on chicks. It is important to note that compared to adults of the same species, chicks normally have:
- Lower PCV and higher white cell count.
- Lower total protein and uric acid.
- Higher CK.

Radiography is an essential tool for assessing the status of the skeletal system, but the low density of the bones and the cartilaginous growth plates in very young chicks can make this difficult.

Common problems

Crop stasis ('sour crop')

This is a commonly seen problem in paediatric medicine, with most sick chicks having varying degrees of crop stasis. All too often hand rearers attempt to diagnose and treat this problem without seeking to understand the underlying pathology.

Aetiology

Causes include:
- Generalized ileus (e.g. systemic illness, foreign bodies, chilling, heavy metal toxicosis, dehydration).
- Crop disorders (e.g. foreign bodies, overstretched/ atonic crop, infectious ingluvitis, fibrous food impaction, or crop burns).
- Dietary problems (e.g. cold food, excessively watery food, food that settles out in the crop, overfeeding, overly dry food).

Clinical presentation

Signs include the crop failing to empty in more than six hours, regurgitation and loss of feeding response. Most chicks will be dehydrated on presentation (erythematous wrinkled skin, tenting of the skin and sunken eyes).

Aspiration of the crop contents usually reveals the sour –smelling fermenting ingesta ('sour crop') or thickened ingesta (hand-rearing formula with the fluid drawn out of it).

Faecal output may be reduced, and the stool may be unformed or pasty.

Diagnosis

This is based on crop and faecal cytology (Gram stain) and culture, haematology and biochemistry, and radiography.

Management

The cause should be identified using the means outlined above, and corrected where possible.

The crop should be emptied with a feeding tube and repeatedly lavaged with warm saline until a clear wash has been obtained. (In some extreme cases, e.g. foreign bodies, it may be necessary to perform an ingluviotomy.) It should always be assumed that these chicks are dehydrated, and they should be treated with parental fluids until crop motility has been restored.

Appropriate antimicrobials should be given as indicated by crop and faecal cytology/culture. It is overly simplistic to assume that these cases are always yeast infections; in the author's experience bacterial overgrowth is usually more common.

A crop 'bra' can be used if needed. This is a non-adhesive bandage placed under the crop and around the wings to 'lift and support' the atonic crop in order to allow gravity to assist with crop emptying.

Once the crop has been emptied, in many cases it may be advisable to leave it empty for a few hours while dehydration is corrected. Initial feeds should be of small volumes of isotonic saline. If this moves through, solids can be added. Small, watery meals should be

fed often. Pre-digesting the hand-rearing formula with a small amount of pancreatic enzymes can liquefy the diet without diluting it.

Motility modifiers (e.g. metoclopramide or cisapride) may assist in restoring motility, although their efficacy is poor if used without other supportive measures. Metoclopramide, in particular, is usually ineffective unless given as a constant rate infusion. In uncomplicated cases (where the chick is otherwise bright), a strong solution of fennel tea given by crop drench may assist in restoring motility.

The prognosis is good, provided prompt and appropriate therapy is provided.

Stunting

This is a common condition where chicks in the first 30 days of life do not grow normally and become stunted.

Aetiology

It is usually associated with improper feeding techniques (poorly balanced or incorrectly mixed diets, inadequate amounts fed, etc.), poor environmental conditions (temperature and humidity extremes) or disease (e.g. renal disease).

Clinical presentation

Signs include subnormal weight gain, reduced muscle mass (toes, wings, back should be checked), abnormal feathering (e.g. head feathers develop in a circular pattern on the crown) and oversized head relative to the size of the body. Eyelids fail to open normally or when expected, and there is delayed ear opening or narrowing of the ear canal. The affected bird may suffer with chronic, recurrent infections, and be constantly calling and begging for food. As the chick gets older it often develops a globose head with an elongated slender beak. The eyes may appear exophthalmic because of the misshapen skull.

Management

The predisposing cause should be identified and treated. Nutritional inadequacies should be corrected. The prognosis is good if the problem is diagnosed early and treated successfully.

Infectious disease

Infectious diseases are quite common in young chicks; their low level of immuno-competence combined with often substandard rearing practices leaves them highly predisposed to infection. This same lack of immuno-competence means that the progression of an infectious disease in young birds is often rapid. Prompt and aggressive therapy is needed to save the patient.

Aetiology

Infections may be bacterial (**Pseudomonas**, **E. coli**, other gram-negative bacteria); fungal (**Candida**, **Aspergillus**); viral (polyomavirus, adenovirus, PBFD); **Chlamydia psittaci**; parasitic: protozoa (Cryptosporidia, Trichomonas, Cochlosoma, Coccidia and Atoxoplasma [in young canaries]); and nematodes (ascarids, Capillaria and Acuaria).

Clinical presentation

Signs include lethargy, loss of feeding response, pallor or erythema of the skin, dehydration, crop stasis, vomiting/regurgitation, weight loss or failure to thrive, subcutaneous haemorrhage, feathering abnormalities and sudden death.

Management

The aetiological agent should be identified and the chick treated accordingly. The patient will require supportive care.

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IMAGING PORTOSYSTEMIC SHUNTS IN DOGS: COMPARING RADIOGRAPHY, ULTRASONOGRAPHY AND COMPUTED TOMOGRAPHY

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IMAGING OF PORTOSYSTEMIC SHUNTS IN DOGS: COMPARING RADIOGRAPHY, ULTRASONOGRAPHY AND COMPUTED TOMOGRAPHY

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There are multiple factors that will influence sensitivity and specificity for the diagnosis of intrahepatic and extrahepatic anomalous vesselsin dogs. The 3 most common factors are theavailability of equipment, the operatorand the patient/owner.

Radiography is one of the most commonly use imaging modality for screening of animals with suspected portosystemic shunt. The sensitivity and specificity is low. We may see microhepatia and enlargement of the kidneys. Occasionally, mildly mineralized calculi in the urinary system is visualized. Contrast radiography such as intraoperative cranial mesenteric venography will be able to outline the anomalous vessels, but this is an invasive procedure. Cranial mesenteric angiography which need a fluoroscopy machine is another method of choice. Again this is also an invasive procedure. The advantage of these two procedures are the ability or visualize small intrahepatic portal veins otherwise may not be seen on computed tomography (CT).

Patient preparation for ultrasound is very important in search for both intrahepatic and extrahepatic anomalous vessels. Most of the patient with anomalous vessels has microhepatia, thus it is not easy to visualize the liver if the patient has excessive movement and panting. Sedating or putting the animal under GA will help to increase the sensitivity of detecting any anomalous vessel. If a patient has a postprandial stomach, this may reduce the acoustic window and interfering with detection of the normal and abnormal blood vessels. There is variation of the type of the shunt vessels, which include the different sizes and lengths of the anomalous vessel. It is easier to detect a large diameter anomalous vessel, thus the sensitivity and specificity are higher. It is also easier to trace a short anomalous vessel, and

to be able to recognize the origin and insertion of the anomalous vessel. A small diameter, long and tortuous anomalous vessel could be detected, but most of the time it is difficult to trace the entire vessel. The operator needs to have a very strong knowledge of the normal anatomy of the blood vessels, and also familiar with the various type of anomalous vessels. The last factor is the ultrasound machine used. As a general rule, we need a good ultrasound machines with a better resolution and good Colour Doppler sensitivity to detect any anomalous vessel. This is especially true when the blood vessel(s) is small. In the last decade, the availablility of better resolution and colour Doppler ultrasound machines and literatures of various anomalous vessels has promote the use of ultrasound in the investigation of intrahepatic and extrahepatic anomalous vessels.

The sensitivity and specificity of ultrasound detection of intrahepatic shunt has been reported to be 100% and 100% while the sensitivity and specificity of detection of extrahepatic shunt has been reported to be 90% and 97% respectively.

There are 2 steps in ultrasonographic examination of the anomalous vessel, the screening and the confirmation. It has been reported that there is usually a change in size of the blood vessels (portal vein and caudal vena cava) with occurrence of extrahepatic shunt. Thus a comparison of portal vein and caudal vena cava (PV/CVC), and also portal vein and aorta (PV/Ao) ratio has been published. A PV/Ao ration of 0.7 to 1.25 is considered normal. Thus, dogs and cats with PV/Ao ratio of ≤0.65 were found to have either an extrahepatic portocaval shunt or idiopathic, noncirrhotic portal hypertension. Dogs and caats with a PV/Ao of ≥0.80 may have other types of shunts or normal. It is good to use the PV/Ao ratio as a screening procedure before looking for an anomalous vessel. When a patient with a PV/Ao ration of <0.65 and has clinical suspicious of anomalous vessel, the portal vein and caudal vena should be examined carefully. If not anomalous vessel is identified, then abdominal contrast CT is recommended to rule out any small anomalous vessel. If an anomalous vessel is identified ultrasonographically, contrast CT may not be needed.

The anomalous vessel should be classified as congenital or acquired, intrahepatic or extrahepatic, and singular or multiple. Most anomalous vessels are congenital, and singular. Large breed dogs tends to have intrahepatic while smaller breed dogs to have extrahepatic anomalous vessels. In patients with acquired anomalous vessels, this normally occurs secondary to severe liver disease, hepatic AV malformations, portal vein thrombosis or hepatic vein outflow obstruction with portal hypertension.

There are three types of anomalous vessels in intrahepatic shunts: The left divisional, central divisional

and right divisional shunt. The central divisional anomalous vessel is the most difficult to trace due to its short length. Both left and right divisional anomalous vessels are wide and tortuous.

There are many types of extrahepatic anomalous shunt vessel. The most commonly seen are splenoazygus, splenocaval and splenophrenic. Less commonly seen anomalous shunt are splenocaval, right gastric-caval, double right gastric-caval, double right gastric-azygus. The presence of extra blood vessel(s) adjacent to the aorta making splenoazygus shunt readily recognized with both intercostal and subcostal approaches. However, due to the long and tortuous nature of the anomalous vessel, it is not easy to trace the entire vessel back to the splenic vein. Anomalous splenocaval shunt vessel is normally short and could be trace at the region of the portal hepatis. Due to the cranial position of the anomalous splenophrenic shunt vessel adjacent to the diaphragm, this type of shunt vessel is more challenging to visualize due to movement of the diaphragm secondary to respiration.

Acquired shunt is normally secondary to portal hypertension, and multiple shunt vessels or varices are present. Most of the time, these varices are short, small in diameter and tortuous. They are commonly located cranial to the left kidney or at the region of the splenic vein. Esophageal varices has been reported. Whenever, there is suspicious or confirmation of an acquired shunt, measure of portal blood flow velocity should be performed. Normal portal blood flow velocity is between 10-25 cm/sec. Any patients with portal blood flow velocity of <10cm/sec has higher probability of developing acquired extrahepatic shunts.

Computed tomography has become the imaging modality of choice for most clinician nowadays. This is because it is not invasive and the detail anatomy of the anomalous blood vessels could be outline with post procession 3D reconstruction. The information of diameter, length and the actual insertion of the shunt vessel could be acquired from the CT study. Computed tomography normally is fast (less than 10 minutes), and with 64 slice machines, occasionally this study could be performed under heavy sedation. The only disadvantage of this is the intrahepatic portal veins may not be identified.

The ultimate choice of modalities/technique used in the investigation of anomalous vessels is again depending on the patient preparation, available of equipmentsand also the operator experience and preference. Recently, the clinician/surgeon' preference has become a deciding factor of the choice of the techniques. Some surgeons want to know the distant between the portal hepatis and the anomalous vessel, the size and length of the anomalous vessel and also the exact connection point of the anomalous vessel for surgical planning. A

3 D reconstruction of the anomalous vessel is always requested by surgeons prior to surgical correction.

NURSES (HILLS)

NUTRITIONAL ASSESSMENT: A CRITICAL COMPONENT OF PATIENT CARE

R. Lumbis[1]

Nutrition is one of the most important considerations in the maintenance of health and plays a critical role in the management of diseases, patient recovery and hospital outcome; a reflection of its acknowledgment as the fifth vital assessment (after temperature, pulse, respiration and pain). The veterinary healthcare team (veterinarians, veterinary nurses/technicians and patient care assistants) play an instrumental role in implementing nutritional support to hospitalised animals and educating pet owners about nutrition. Yet, findings suggest that, while 90% of pet owners would like a nutritional recommendation from the veterinary healthcare team, only 15% of pet owners identify receiving one (AAHA, 2003). An understanding of basic nutritional principles and the application of nutrition in optimising the health and wellbeing of both fit and clinically affected companion animals is therefore essential.

As with any medical intervention, there are always risks of complications and this is no different with nutritional interventions. Minimising such risks depends on careful patient selection and assessment. Nutritional assessment identifies malnourished patients requiring immediate nutritional support as well as those at risk of developing malnutrition.

In 2011, the WSAVA Global Nutrition Committee (GNC) launched Nutritional Assessment Guidelines for Dogs and Cats to help the veterinary healthcare team and pet owners ensure that dogs and cats receive optimal nutrition, tailored to their needs (Freeman et al, 2011). They have been endorsed by the BSAVA, together with numerous other global veterinary organisations, published in a variety of journals and translated into many languages since. They are available for free download from the WSAVA website (www.wsava.org) and have become one of its most frequently accessed WSAVA resources.

These guidelines provide a framework for the veterinary healthcare team to assist them in making a nutritional assessment, and specific nutritional recommendations, for every patient at every visit (Figure 1).
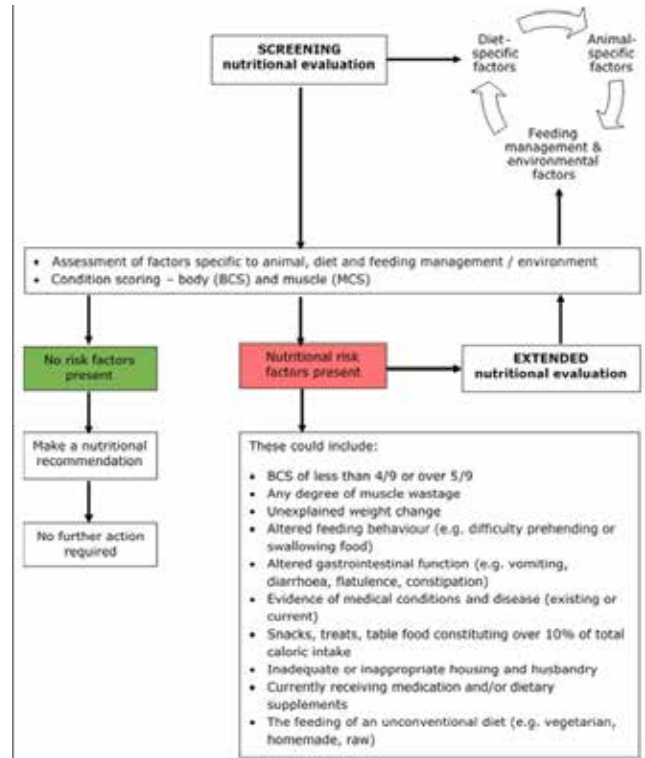
Figure 1: The nutritional assessment process using the WSAVA Nutritional Assessment Guidelines for Dogs and Cats (modified from Freeman et al, 2011).

The first stage of this process involves making a systematic screening evaluation of the animal as well as identification of the diet fed and any feeding management and environmental factors. This includes information obtained from the history and a physical examination and should include measurement and/or consideration of these additional factors:

• Current and previous bodyweight

• Body Condition Score (BCS)

• Muscle Condition Score (MCS)

• Diet (including type and brand, frequency of feeding and amount being fed)

• Medications

• History of vomiting, diarrhoea or other

• Temperature, Pulse, Respiration, Pain

• Any abnormal physical findings

The identification of any abnormalities in the history, diet, or physical exam and nutrition-related risk factors should prompt a more in-depth extended evaluation of each of these factors and will impact on how, and when, the nutritional plan can be implemented (Table 1). If no risk factors are identified, owners should be advised accordingly and given an appropriate recommendation, for example, to continue with the existing diet.

Table 1: Examples of factors to consider when completing a nutritional assessment.

	Patient factors	Dietary factors	Feeding and environmental factors
Initial screening evaluation	• Age? • Physiological status? • Activity levels and daily exercise? • Body condition? • Body weight? • Existing medical conditions?	• Conventional or unconventional? • Suitability for the species and life-stage? • Complete versus complementary? • Composition (including ingredients)? • Feeding guidelines?	• Frequency, timing, location and method of feeding? • Food container and material e.g. Metal bowl, food dispensing toy • Multi-pet household? • Quality of surroundings and husbandry? • Pet's access to space?
Extended evaluation	• Changes in food intake or behaviour? • Alterations in gastrointestinal function and faecal production? • Condition of the skin? • Presence and effect of any medical conditions and/or medications? • Laboratory abnormalities?	• Other sources of nutrients, e.g. access to treats, snacks and table food? • Type, formulation, energy density, texture and flavour of diet? • Storage of the diet?	• Primary feeder of pet? • Other providers and sources of food? • Extent of enrichment? • Environmental stressors? • Presence of conditions and/or circumstances causing fear and anxiety?

Nutritional assessment identifies risk factors influencing how, and when, the nutritional plan can be implemented. In some situations, focus must be placed on resuscitation and stabilisation with the possibility of feeding being delayed until the patient is haemodynamically stable and any major electrolyte, fluid, and acid-base abnormalities have been corrected. Appropriate laboratory analysis may be performed and any concurrent conditions such as renal or hepatic disease may require dietary adjustments. A nutritional plan should prevent (or correct) overt nutritional deficiencies and imbalances. Further to this, a detailed dietary history, including information about the petThe provision of supportive care to address problems involving hydration and electrolyte status, pain, body temperature, vitamin B deficiencies and nausea can result in appetite being re-established in many anorexic patients (Delaney, 2006).

In addition to the nutritional assessment guidelines, the GNC has prepared a non-branded Global Nutrition Toolkit, again available for free download and containing a number of resources including:

• A body condition scoring chart

• A muscle condition scoring chart

• 'How to' videos for performing a feline and canine body condition score

• A diet history form

• A nutritional assessment checklist

• Calorie guidelines for healthy adult dogs and cats

• A hospitalised patient feeding guide and nutrition monitoring chart

• An advice sheet for pet owners on selecting the right diet

• A pet owner's guide to nutrition on the internet

Additional resources are added to the toolkit on a regular basis to reflect the latest thinking on nutrition.

The vision of the GNC is to help the veterinary healthcare team and the public understand the importance of nutrition in companion animal health by providing an expert source of accurate nutritional information and recommendations. Its campaign aims to ensure that a nutritional assessment and recommendation is made on every patient during every visit to the vet. All members of the veterinary healthcare and reception team are integral to promoting the value and importance of nutrition in their practice. As acknowledged by the American Animal Hospital Association (AAHA, 2009, p4),

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WSV18-0225

CLINICAL PATHOLOGY

HOW TO GET THE MOST FROM CYTOLOGY SUBMISSIONS

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HOW TO GET THE MOST FROM CYTOLOGY SUBMISSIONS

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Successful microscope evaluation of a specimen depends on the quality of the sample. This seminar will focus on how to collect and process a sample such that a good quality slide and/or fluid sample amenable to cytologic evaluation and fluid analysis is prepared. A systematic approach to the evaluation of a cytologic specimen will be introduced. By the end of this seminar, you will walk away with practical tips to avoid non-diagnostic results, an approach to diagnosing disease from fine needle cell aspirates of lumps and bumps and harvesting cells present in body fluids and washes.

WSV18-0175

ISFM - FELINE CARDIOLOGY

FELINE CARDIOMYOPATHY: AN UPDATE AND REVIEW OF RECENT UNDERSTANDING

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In this lecture, we compare human and feline cardiomyopathy and discuss controversies in current thinking and how to classify feline cardiomyopathy.

Diagnosis of feline cardiomyopathy

The classification system for feline cardiomyopathy requires updating, and there is poor agreement between cardiologists using the current system. One problem is that the current system does not account for the changes seen as heart disease progresses over time. For example, a cat may start being classified as hypertrophic cardiomyopathy (HCM), but as the myocardium becomes ischaemic and myocardial function deteriorates with time, the same individual may be classified as restrictive cardiomyopathy and then dilated cardiomyopathy over the coming years. Since many cats do not undergo serial echocardiography, and most patients have a single echo at the time of diagnosis, it is likely that many individuals are misclassified ("misdiagnosed") at the outset, when they are in fact a case of advanced HCM.

Hypertrophic cardiomyopathy:
- Left ventricular myocardium measures $\geq$6mm in any 2D view, long or short axis.
- No identifiable cause of LV hypertrophy; i.e. exclude hyperthyroidism, hypertension, infiltration (if possible), acromegaly, Cushing's disease.

Restrictive cardiomyopathy:
- Non-hypertrophic, non-dilated LV
- Normal systolic function
- Left or bi-atrial dilation
- Restrictive inflow pattern on Doppler interrogation of mitral valve (can be present in any advanced heart disease with increased left atrial pressure)
- Bridging scar across the left ventricle (classified as endomyocardial form of RCM)

Dilated cardiomyopathy:
- Dilated, non-hypertrophic LV
- Subnormal systolic function

Arrhythmogenic right ventricular cardiomyopathy:
- Disproportionate right heart dilation
- Normal appearing LV
- Arrhythmias – usually ventricular – detected on ECG

Unclassified cardiomyopathy:
- Case which does not fit into any of the above classifications

WSV18-0257

SVA SOFT TISSUE SURGERY

PERINEAL HERNIA

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Perineal hernias result from weakening or complete failure of the muscular diaphragm of the pelvis. Although the development of hernias may be multifactorial – the predominance of intact males suggests a hormonal balance contributes to the weakening of the pelvic diaphragm. Female dogs are less frequently afflicted but, in these cases, additional measures (such as abdominal ultrasound) should be taken to rule out concurrent predisposing diseases. Successful long-term management of hernias relies upon eliminating the underlying cause (concurrent castration in most cases).

Presentation typically includes swelling adjacent to the rectum, signs of constipation, tenesmus, lethargy, stranguria (if bladder or prostate are involved) and altered tail carriage. Differential diagnoses include perianal/pelvic canal/subcutaneous masses.

Rectal examination should be performed with both left and right fingers as it is difficult to palpate on the right with the right and on the left with the left hand. Even if one side of the perianal region appears normal it should be thoroughly palpated as bilateral herniation is common. Attempts should be made to localize the bladder and prostate either within the abdomen, pelvic canal or within the hernia itself. Hydration should be carefully assessed. Diagnostic workup includes blood work and a positive contrast cystourethrogram as indicated.

Pre-operative medical management can include: a low residue diet, lactulose to effect, and intermittent deobstipation. However, continued medical management will often result in worsening of the condition risking bladder entrapment and rupture or fistula development. Furthermore, chronic hernias are typically more inflamed and have a greater number of adhesions risking inaccurate tissue apposition or neurovascular damage during repair.

Surgical procedures often include castration, cystopexy and/or colopexy in addition to the definitive hernia repair. Surgery involves careful identification of the following structures; external anal sphincter, levator ani (if still present), coccygeus, internal obturator, pudendal bundle, and sacrotuberous ligament. Care should be taken to identify the exact location of the hernia (ie between which tissues) as this will affect how the repair is performed.

Most cases of hernia can be managed with direct apposition of the external anal sphincter to the levator ani/coccygeus dorsally and elevation of an internal obturator flap ventrally. However, additional reconstruction options include the use of the superficial gluteal, semitendinosus muscle or prosthetic implants. Repairs can be carried out with relatively large gauge monofilament sutures, either absorbable or non-absorbable suture materials are appropriate.

Following surgical management of the hernia, a diet higher in insoluble fibre (such as diets for canine diabetes, feline hairball control or weight management) can be used to allow passage of softer bulked faeces.

Complications include potential recurrence, faecal incontinence, infection and/or contralateral herniation.

WSV18-0243

PROTEIN-LOSING ENTEROPATHY: INTEGRATING DIET AND MEDICAL MANAGEMENT

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WSV18-0061

PROTEIN-LOSING ENTEROPATHY: INTEGRATING DIET AND MEDICAL MANAGEMENT

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Feeding a complete and balanced diet is known to promote wellness in dogs and cats, but a number of highly publicized pet food recalls, as well as a growing appreciation for the role of diet in health and disease, has raised questions about the use of commercial pet foods to meet this end. Additionally, concerns over specific ingredients, such as corn or wheat, have been promoted through advice columns and pet food marketing companies and has caused some owners to become leery of manufacturers that incorporate these ingredients into their foods. Many owners, and some veterinarians, also advocate feeding dogs and cats home-prepared foods exclusively (raw or cooked, or both) and either cite perceived health benefits or a general mistrust of the pet food industry. At its most basic concept, food is a means of getting essential nutrients into the body. How these nutrients are delivered or which specific ingredients are being used (barring ingredient sensitivity) is much less important than ensuring that the optimal amount is provided on a regular basis. Whatever the reason for a particular diet strategy, it is important for veterinarians to understand the motivations, risks and benefits of that diet type to ensure that the nutritional needs of the individual animal are being met.

WSV18-0100

NEPHROLOGY

MANAGEMENT OF THE PROTEINURIC DOG

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Introduction

Dogs and cats may normally have small amounts of protein in their urine; however the term "proteinuria" usually refers to the presence of an abnormal amount of protein in the urine. The term "microalbuminuria" refers to the presence of albumin in the urine in a concentration of 1-30 mg/dL, which is considered abnormal, but is below the detection limit of the urine dipstick. A persistently high magnitude proteinuria is usually an indicator of chronic kidney disease (CKD); however it may be a secondary consequence of infectious, inflammatory, metabolic or neoplastic disorders. Proteinuria is also a prognostic marker and is associated with a more rapid progression of CKD, a higher frequency of uremic crises as well as an increased mortality rate (1, 2). Thus, early detection of proteinuric animals will allow close monitoring as well as early therapeutic intervention, which may decrease the magnitude of proteinuria and the disease progression rate of these patients. Screening for proteinuria should be performed in any animal diagnosed with CKD or with any other disease known to be associated with proteinuria.

Methods of detection and interpretation of test results

The urine dipstick colorimetric test is the most commonly used method to screen for proteinuria. The urine dipstick is more sensitive to albumin compared to other proteins, and its lower detection limit is 30 mg/dL. Interpretation of any result should be done in light of the urine specific gravity. A positive result in highly concentrated urine reflects a smaller degree of protein loss compared to the same amount of protein in diluted urine; thus, the latter is more alarming. Both false positive and false negative results occur using the urine dipstick. Once persistent proteinuria has been confirmed or when a high magnitude proteinuria is suspected, it should be quantified using urine to creatinine ratio (UPC). Its results are used as a guideline for diagnostic investigation, therapeutic intervention, and monitoring response to therapy. Urine protein to creatinine ratio <0.2 in dogs and cats is considered normal, and a ratio between 0.2-0.4 in cats and 0.2-0.5 in dogs is considered borderline proteinuria.

The origin of proteinuria

Once proteinuria has been documented, its origin should be identified as a first step towards the diagnosis of the underlying disease. Proteinuria can be classified as urinary system or extra-urinary system in origin.

Extra-urinary system proteinuria may result from either pre-renal or post urinary system (i.e. genital system) conditions. Pre-renal proteinuria results from presence of excessive amounts of either normal (e.g., hemoglobin, myoglobin) or abnormal (e.g., Bence Jones) blood proteins, which can be freely filtered through the glomerulus. Thus, pre-renal proteinuria can occur with normal kidney structure and function and treatment is aimed at identifying and eliminating the underlying disease. Urinary system proteinuria can be classified as renal (functional or pathological) or post renal. Functional renal proteinuria represents a transient change in the permselectivity characteristics of the glomerulus, and thus should not be treated. Pathological renal proteinuria may result from glomerular (decreased permselectivity), tubular (decreased re-absorption) or interstitial (exudation of proteins to the urinary space) abnormalities. Glomerular proteinuria is the most common cause of persistent high magnitude proteinuria. It requires close monitoring, and often warrants diagnostic workup and therapeutic intervention. Post-renal proteinuria relates to the entry of proteins into the urine from the renal pelvis, ureters, urinary bladder, or urethra, and results from disorders along the urinary excretory system (e.g., infection, urolithiasis, neoplasia).

Diagnostic approach

The diagnostic workup is directed towards the detection of the origin of proteinuria and the underlying disease, and includes complete history and physical examination as well as diagnostic tests such as arterial blood pressure measurement, complete blood count, serum chemistry, urinalysis and urine culture, serologic testing and PCR for infectious diseases, diagnostic imaging, and kidney biopsy. Initially, post renal proteinuria is excluded by evaluating the urine sediment for presence of inflammation and hemorrhage. Then, extra urinary system causes are excluded. Post urinary (genital system) proteinuria is easily excluded by performing urinalysis on urine obtained by cystocentesis, and pre renal proteinuria is ruled out by evaluating the plasma protein concentration and excluding dysproteinemia and presence of specific proteins in the urine (e.g., hemoglobinuria, myoglobinuria and Bence Jones proteins). Glomerular proteinuria can be of any magnitude, but is particularly suspected when persistent high magnitude (UPC≥2) proteinuria is present, and after ruling out extra renal and post renal causes. Glomerular proteinuria can be diagnosed by obtaining a kidney biopsy which can additionally help in sub categorizing the disease, using light microscopy, electron microscopy and immunofluorescence.

Treatment of proteinuria

Therapy of proteinuria should be directed towards elimination of any underlying disease and decreasing the magnitude of proteinuria. Successful therapy

of the underlying disease may resolve proteinuria; however, some patients will remain proteinuric due to the presence of irreversible damage. When proteinuria persists after elimination of the underlying disease or when the latter neither can be identified nor be eliminated, therapy is merely symptomatic. Treatment goals include decreasing the magnitude of proteinuria to the reference range to minimize progressive kidney damage, as well as preventing and treating the secondary consequences of the protein loss (e.g., thromboembolism).

Azotemic patients require therapeutic intervention at a lower magnitude of proteinuria compared to non-azotemic patients. Current guidelines recommend treating non-azotemic patients when UPC ratio is ≥2.0, while azotemic dogs and cats are to be treated when UPC≥0.5 and UPC≥0.4, respectively.

Standard therapy

Dietary modification, angiotensin converting enzyme inhibition (ACEi) and angiotensin receptor blockade (ARBs) are the mainstay of therapy. Protein restriction is one of the dietary modifications recommended for patients with protein losing nephropathy. Even though counterintuitive, increasing dietary protein amounts is associated with increased albuminuria and may result in decreased serum albumin concentration. ACEi and ARBs decrease the efferent glomerular arteriolar resistance, resulting in decreased glomerular transcapillary hydraulic pressure. Administration of ACEi and ARBs should be exercised with caution, especially in severely and acutely azotemic patients. Low dose aspirin (0.5 mg/kg, PO, q12-24 hr) may also decrease proteinuria in dogs. It has been shown that glomerular damage may be prevented by thromboxane release inhibition, thus preventing platelet aggregation and neutrophil chemotaxis. An additional potential advantage of low dose aspirin therapy is decreasing the risk of thromboembolism, especially in animals with a decreased hypoantithrombinemia.

Immunosuppression

When kidney biopsy is obtained and there is evidence for immune complex deposition, immunosuppression should be initiated. Empirical application of immunosuppressive/anti-inflammatory therapy should be considered for animals with severe, persistent, or progressive glomerular disease in which there is renal biopsy supported evidence of an immune pathogenesis and no identified contraindication to immunosuppressive therapy. For diseases associated with profound proteinuria, attendant hypoalbuminemia, nephrotic syndrome, and/or rapidly progressive azotemia, single drug or combination therapy consisting of rapidly acting immunosuppressive drugs is recommended.

Based on preliminary, uncontrolled clinical experience with mycophenolate and its low rate of serious complications, mycophenolate is recommended for therapy of dogs with glomerular disease of an apparent immune pathogenesis. Short-term administration of glucocorticoids may be recommended in fulminate cases where immediate immunosuppression is required if their use is adjusted to minimize their adverse effects. However, on the basis of current practice perceptions and anecdotal experience, the use of glucocorticoid therapy should be tapered to the minimally effective dose as quickly as possible due to predictable side effects (3).

Monitoring of proteinuria

When the degree of proteinuria is mild and therapeutic intervention is not indicated, periodic monitoring should include urinalysis, UPC ratio, and serum creatinine and albumin concentration at least every 3-6 months. When therapy is applied, closer monitoring should be performed. In high-risk patients serum creatinine should be monitored 3-5 days after initiation of ACEi/ARBs to identify a significant decrease in glomerular filtration rate. Urinary protein to creatinine ratio should be monitored periodically and therapy should be adjusted. Due to day to day variation, not every change in UPC ratio would be considered significant. At least a 35% or 80% change should be demonstrated when the UPC ratio is high (around 12) or low (around 0.5), respectively. In animals with progressive kidney disease, the magnitude of proteinuria may decrease in late stages of the disease due to a reduction in the number of remaining nephrons through which protein loss can occur.

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43RD WORLD SMALL ANIMAL VETERINARY ASSOCIATION CONGRESS AND 9TH FASAVA CONGRESS

WSV18-0033

SVA EXOTICS

ANTIBIOTIC SELECTION IN EXOTIC PETS

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Antibiotic selection should ideally be guided by the results of culture and sensitivity tests performed on swabs from clinically relevant sites and organs. However, where that is not possible due to lack of client compliance or infections in difficult to access sites, antibiotic selection based on first principles can be the difference between resolution of the infection or death from septicaemia.

Enrofloxacin is a popular choice of antibiotic in exotic pets. However, using a fluoroquinolone as a first line antibiotic can have consequences for development of antimicrobial resistance. As antibiotic stewardship campaigns become more prominent in the veterinary community, veterinarians should consider other antibiotics when faced with infections in exotic pets.

In birds, doxycycline can be used as broad-spectrum antibiotic against aerobic gram positive and gram negative bacteria, including Chlamydia and Mycoplasma sp. Clavulanic acid/amoxycillin can also be used against aerobic and anaerobic gram positive and gram negative bacteria. There are both oral and injectable formulations of each antibiotic.

In reptiles, ceftazidime can be used as a broad spectrum antibiotic against aerobic gram positive and gram negative bacteria. It also has action against a few anaerobic bacteria. When treating a primarily aerobic gram negative bacterial infection, another alternative is gentamicin. The main advantage of both these antibiotics is that they do not require daily administration when injected and has less tissue irritation effects at the site of injection compared to injectable enrofloxacin.

Small mammals such as rabbits and guinea pigs can suffer dysbiosis if an inappropriate antibiotic, e.g. oral clavulanic acid/amoxycillin is used. It is possibly for this reason that veterinarians would rather prescribe a safer antibiotic such as enrofloxacin as a first option. However, other antibiotics, such as trimethoprim/sulfamethoxazole may have a broader spectrum and equal safety profile as enrofloxacin.

Another antibiotic that can be considered in all common exotic pet species is metronidazole, but it has action only against anaerobic bacteria, and is also anti-protozoal.

In conclusion, there are non-fluoroquinolone antibiotics that can be used safely in exotic pet species and should be considered where possible. The dose rates and frequency for each antibiotic has deliberately been left out of this abstract so that changes in dose rates from newer pharmacological studies does not alter the accuracy of the content.

WSV18-0035

DIAGNOSTIC IMAGING AND GASTROENTEROLOGY
(SIMULTANEOUS TRANSLATION INTO MANDARI

PRINCIPLES AND CLINICAL APPLICATION OF COMPUTED TOMOGRAPHY

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PRINCIPLES AND CLINICAL APPLICATIONS OF VETERINARY COMPUTED TOMOGRAPHY

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Computed tomography is a diagnostic imaging modality that is widely used because it can provide axial sectional or slice-oriented imaging of the patients. This helps to detect abnormalities in areas where there is a lot of superimposition on plain radiography such as the nasal turbinate, skull, and the spine. Two additional advantages of performing CT study is its capability of performing multi-planar and three-dimensional image reconstruction. This helps in precise determination of anatomical location of abnormalities detected. This is especially very helpful in computed tomography angiography and the evaluation complex fractures.

CT unit is comprised of a gantry, couch (patient table), hardware equipment and an operator console. The gantry houses an x-ray tube, collimators and the X-ray photon detectors. The X-ray tube will rotate around the patient table while the incident photon from the X-ray tube will be recorded by the detectors. The X-ray photon will then be transformed to electrical signal and converted to a digital image. The patient table normally will move according with the movement of the gantry. For large animals, a special custom designated table is needed.

Once the raw data is acquired, the images can be displayed using different reconstruction algorithms. Different window settings, defined as window width and level, can be used to view the study. The window width (W) determines the contrast and the window level (L) determines the grey scale of the images. Usually the CT images are displayed using several different window and level settings for each study.

Computed tomography measures and computes the spatial distribution of the linear attenuation coefficient in each pixel and it is displayed relative to the attenuation of water. These values are called CT numbers or Hounsfield Units (HU). The normalized HU ranges from about -1000 to +3000. Typically, the HU of water is 0, air is -1000, lung is -845, fat is -100, brain is + 30 to +40, muscle is +50 to +70, and bone and mineral are +100 to + 1000.

One of the most common applications of CT is the investigation of nasal disease. This is normally performed when there are chronic clinical signs and a suspicious of nasal neoplasia. Computed tomography will show detail anatomical changes such as bone and turbinate lysis, presence of soft tissue attenuating material and the pattern of contrast enhancement. Computed tomography will often be able to differentiate inflammatory rhinitis, fungal rhinitis and nasal neoplasia due to their distinctive features. Two important aspects to evaluate are to rule out invasion of the brain (especially by the nasal neoplasia) and the presence of tooth root abscess. Computed tomography will also be able to assess the mandibular and retropharyngeal lymph nodes to rule out metastasis of the nasal neoplasia.

Two major objectives of the CT evaluation of the lungs are to screen the patients for metastatic disease (1) and to define the location of an abnormality seen on the radiography. Computed tomography is more sensitive than radiography for the detection of small pulmonary nodules. In patients with a continuous pneumothorax, CT examination is performed to locate a possible lung bulla that lead to this condition.

In recent years, CT examination of intervertebral disc disease (IVDD) has been used widely to replace the use of myelography (2). It has also become the first choice of imaging modalities to investigate IVDD when the cost and availability of MRI prohibit the use of MRI. Hyperattenuating mineralized disc material is readily detectable on CT examination and with conjunction with the history and clinical signs, a diagnosis of IVDD is easily made. Normally radiography of the spine is performed before the CT examination to rule out any obvious changes such as lysis of the bones.

For the abdominal cavity, CT examination is normally performed after an abdominal ultrasound to investigate the margins and resectability of an abdominal mass, or to confirm suspected ectopic ureters. Investigation of the invasiveness of an adrenal tumor to the surrounding blood vessels is also an important indication of abdominal CT (3). The investigation of portosystemic shunts using dynamic contrast study has become more common with the available of multislice CT machine (4).

In conclusion, CT can be used to investigate various organ systems and diseases. It is mostly performed after radiography and ultrasonography due to its cost and the need of general anesthesia and/or deep sedation.

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CLINICAL PATHOLOGY

IMMUNOHISTOCHEMISTRY AND MOLECULAR BIOLOGY TESTING FOR CANCER CASES

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IMMUNOHISTOCHEMISTRY AND MOLECULAR TESTING FOR CANCER CASES

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Introduction

The basis for histopathological diagnosis and characterization of tumours has for many years been the evaluation of sections stained by haematoxylin and eosin (HE) and examined by light microscopy. This procedure still remains the cornerstone for diagnosis and the starting stage for subsequent application of the techniques that will be described in this lecture. One major advance with routine HE microscopy has been the availability of slide digitization for computer-based analysis, which facilitates remote working by diagnostic pathologists and the ability to hold case discussions at a distance.

Immunohistochemistry

Immunohistochemistry (IHC) is defined as the use of monoclonal or polyclonal antibodies to detect and localize antigen within a tissue section. In contrast, immunocytochemistry (ICC) is the use of such reagents to detect and localize antigen within a cell monolayer. There are now numerous variations of procedures for IHC based on using different tissue samples (fresh frozen or formalin-fixed), pre-treatment protocols (antigen retrieval and blocking steps) and detection reagents coupled to enzymes (for an enzyme–substrate reaction and colour change viewed by light microscopy) or fluorochromes (for emission of fluorescence when excited by light of particular wavelength under a fluorescence microscope). Details of these methodologies is beyond the scope of this presentation.

Immunohistochemistry (generally using immunoperoxidase enzyme–substrate based techniques) is now usually automated for high throughput and standardization between laboratories. Reagents are available for the detection of a wide range of structural or secreted molecules or the identification of infectious agents. The majority of reagents are broadly cross-

reactive between animal species, which has allowed the application of numerous antibodies raised against human or rodent proteins to be used with tissue derived from companion animals. Testing laboratories now offer a wide menu of target antigens to the veterinary practitioner.

Immunohistochemistry for tumour phenotyping

The use of IHC in small companion animal tumour phenotyping began over 25 years ago with the first studies distinguishing between T- and B-cell lymphoma in the dog. Kaplan-Meier survival curves clearly showed that canine T-cell lymphoma had worse clinical prognosis compared with B-cell lymphoma. There are now numerous studies validating these concepts and further phenotyping the tumours by application of multiple markers [1]. More recent studies have extended immunophenotyping and correlation with survival to feline lymphoma [2]. One of the most useful applications of immunohistochemistry has been in helping to make the distinction between chronic lymphoplasmacytic enteritis and alimentary lymphoma; particularly in the cat [3]. Different approaches have been taken to classification of feline alimentary lymphoma [4, 5], but consideration of low-grade and high-grade alimentary lymphoma and large granular lymphocytic lymphoma of the intestine is a useful framework. Characterization of feline alimentary diseases can be improved when IHC is used in combination with molecular analysis of lymphoid clonality (see below) and a useful diagnostic algorithm has been published [3]. IHC has now been widely applied to many other tumour types and has revolutionized diagnostic histopathology.

One further example of a tumour type in which IHC has proven value is that of canine mast cell tumour. Evaluation of the Ki67 labelling index (an indication of mitotic activity) and assessment of membrane versus cytoplasmic expression of KIT are associated with survival and metastasis of these tumours. Mutations in the KIT gene are further linked to the potential of the tumours to respond to tyrosine kinase-inhibiting therapy [6–8].

Molecular testing of biopsy samples

Analysis of rearrangements in genes encoding chains of T- and B-cell receptor molecules is now widely available for determining 'clonality' within a lymphocytic infiltration of tissue. A reactive or chronic inflammatory lesion involves a polyclonal infiltrate of lymphocytes of numerous different antigenic specificities; however, a neoplastic infiltrate is comprised of a clonal population with a restricted or monoclonal receptor type. It is recommended that clonality testing be performed only as an adjunct procedure following routine histopathological evaluation and IHC [9].

The next advance in tumour diagnosis is larger scale molecular screening evaluating the expression of genes (i.e. mRNA production) encoding structural or metabolic molecules associated with particular types of tumour. One such technology applies the quantitative nuclease protection assay to analyse the small and fragmented mRNA that might typically be found in formalin-fixed and paraffin wax-embedded tissue samples. The same blocked tissue sample of tumour that was used for HE microscopy (and IHC) can be utilized for this technique that screens for a panel of tumour-associated genes. This method can not only phenotype the tumour (as for IHC), but can also provide prognostic and therapeutic information permitting customized therapeutic options for the patient [10].

Serum biomarkers

Although strictly not related to the subject of the present discussion (tissue-based diagnostics), there is active parallel research into serum biomarkers of animal tumours. For example, one study reports the evaluation of a diagnostic/prognostic algorithm based on clinical evaluation of canine lymphoma together with measurement of the serum concentrations of haptoglobin and C-reactive protein [11].

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CARDIAC BIOMARKERS IN CATS: USEFUL TEST OR NEEDLESS DISTRACTION?

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In this lecture, we will briefly review two cases of cardiac neoplasia in cats and how tumours of the heart differ in cats compared to dogs.

Whereas in dogs, cardiac neoplasia typically presents as effusive pericardial disease, cats tend to have less obvious signs of cardiac compromise. Clinical signs are often less specific, which can lead to late or missed diagnosis.

Cardiac neoplasia seen in cats
- Lymphoma; nodular or infiltrative (associated with FeLV infection in some cases)
- Chemodectoma (handful of case reports)
- Haemangiosarcoma (one case reported)
- Metastasis of extra-cardiac tumours; pulmonary and mammary adenocarcinomas reported

Lymphoma makes up at least 80% of identified cardiac tumours. Most cases have a pericardial effusion, and diagnosis is straightforward because the lesion tends to exfoliate and effusion cytology is diagnostic. However, this relies upon the clinician identifying a suspicion of lymphoma – the myocardium often appears thickened with abnormal relaxation, indistinguishable from hypertrophic cardiomyopathy, which is far more common in cats. If cardiac tamponade is identified in a cat, pericardiocentesis should be performed and submitted for cytology, as lymphoma is one of the very few causes of true cardiac tamponade in cats (where signs of right heart failure are present secondary to the pericardial fluid).

Case examples

Cardiac lymphoma

9-year old male Maine Coon cat, presented with signs of left-sided congestive heart failure

Echocardiographic evidence of LV hypertrophy and reduced function

Small pericardial effusion, suspected to be secondary to heart failure

Treated for congestive heart failure, positive clinical response for 4 weeks

Progressive weight loss, evidence of acute kidney injury, anorexia

Euthanasia performed and diagnosis of myocardial and renal lymphoma evident on histopathology

Particularities of the disease and a literature review will be discussed

Heart base tumour

7-year old female Domestic shorthair cat, presented with signs of increased respiratory effort

Radiographic evidence of mass at the heart base

Cytology with ultrasound guidance of cardiac mass showed evidence of a neuroendocrine tumour

CT imaging showed bronchial obstruction

Bilateral bronchial stents deployed via minimally invasive technique

Clinical signs relieved for some months, until tumour invaded oesophagus and patient euthanised because of regurgitation

WSV18-0297

SVA SOFT TISSUE SURGERY

SUBTOTAL COLECTOMY

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Key Points

- Pay attention to the unique blood supply to the colon
- Increase collagenase activity occurs 5 - 7 days after colotomy/anastomosis
- Colon is a high pressure conduit system
- Subtotal colectomy may be curative for megacolon in cats

If you would like a video of this surgical procedure on DVD, go to **www.videovet.org** or email VideoVet at videovet@me.com.

SURGICAL MANAGEMENT OF MEGACOLON

Clinical presentation: Megacolon is a condition in which the ascending, transverse, and descend-ing colon are chronically large in diameter and filled with inspisated stool. Patients generally present with a history of chronic constipation (i.e., weeks to years), tenesmus, and weight loss. Males are more commonly affected than females and the age ranges from one year to 12 years.

Etiology: The etiology of megacolon is a functional defect of the colonic smooth muscle. It is thought to be either congenital, acquired, or idiopathic. The idiopathic form is the most common type seen in the cat.

Diagnosis: Diagnosis of idiopathic megacolon in cats is usually made on the basis of history, abdominal palpation, and radiography. Confirmation is based on ex¬ploratory laparotomy.

Treatment: The decision to operate is generally made on the basis of the constipation becoming progressively worse and responding only to multiple enemas and manual deobstipation. Exhaustive medical therapy is generally performed prior to surgical intervention using a varitey of diets and colonic motility modifiers.

Preoperative management: Preoperative bowel preparation, using antibiotics administered orally or multiple cleansing enemas is probably useless in cases of severe constipation or obstipation. A parenterally administered antimicrobial agent, with a spectrum of activity directed toward coliforms and anaerobes, is probably the most efficacious preoperative management. Compacted stool from chronic obstipation is best removed at surgery rather than trying to remove it pre operatively.

Subtotal colectomy: Subtotal colectomy is the surgical procedure of choice in cats with megacolon. This technique is performed regardless of how much of the colon appears diseased. The surgical objective is to remove all of the colon except what is necessary to reestablish bowel continuity. When the ileocecocolic valve is removed (i.e., which is done if the cecum appears grossly abnormal), a 1.5 - 2 cm segment of descending colon just proximal to the pubis (i.e., colorectal junction) is saved to accommodate the ileo-colonic anastomosis. When the ileocecocolic valve is retained (i.e., which is done if the cecum appears grossly normal), a 1 cm segment of ascending colon is preserved to accommodate the colonic anastomosis.

Several techniques have been described for performing the colonic anastomosis. The author's technique of choice is an end-to-end anastomosis. The procedure is performed using a single layer simple continuous or simple interrupted appositional pattern with 3-0 or 4-0 synthetic absorbable suture. Because of lumen diameter differences between the ileum and colon, it is necessary to place several sutures in the larger diameter bowel (i.e., colon) in order to create similar size lumen diameters thus resulting in a watertight anastomosis.

After the anastomosis is completed, the peritoneal cavity is thoroughly lavaged with 200 - 300 ml/kg of warm, sterile physiologic saline solution prior to closure. In situations where the anastomosis is under any question, particularly with respect to color and blood supply (i.e., tissue viability), it is advisable to place an omental patch over the anastomotic area to help provide a source of blood supply and lymphatic drainage, as well as to help support the anastomosis.

Postoperative care: Immediately postoperatively patients should be supported with a balanced electrolyte solution intravenously until they are able to maintain their hydration status. Antimicrobial treatment is generally continued for five to seven days post operatively. Patients are returned to their normal diet within 24 hours and allowed water ad libitum.

Results: Long term results have been somewhat variable from case to case, but generally:

1) patients generally maintain fecal continence post-operatively.

2) after a 10-15% weight loss 2 - 3 weeks postoperatively, body weight is regained within 3 - 7 weeks

3) watery to mucoid stools occur during the first 3 - 7 weeks followed by mucoid to semi-solid to formed stools by 3-6 months. This is thought to be due to ilieal adaptation thus allowing efficient water absorbtion.

4) frequency of stools is approximately six per day initially followed in 1-2 months by four per day, then at six months to 2-3 stools per day (range 1-4 stools per day).

5) owner satisfaction has been excellent in the majority of cases.

WSV18-0159

CHEW ON THIS – NUTRITION FOR DENTAL DISEASE

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CHEW ON THIS: IMPLICATIONS OF NUTRITION AND DENTAL HEALTH

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Introduction

Periodontal disease is the number one health problem in small animals. By two years of age, 70% of cats and 80% of dogs have periodontal disease. There are generally little to no outward signs of early disease process, so therapy typically comes late. Consequently, periodontal disease may be the most undertreated disease in our patients. Unchecked periodontal disease has numerous local and systemic consequences.

Pathogenesis:

Periodontal disease is initiated by oral bacteria which adhere to teeth in a substance called plaque. Plaque is a biofilm, made up almost entirely of oral bacteria, contained in a matrix of salivary glycoproteins and extracellular polysaccharides. Calculus (tartar) is basically plaque which has secondarily become calcified by the minerals in saliva. **Plaque and calculus may contain up to 100,000,000,000 bacteria per gram.** Bacteria within a biofilm do not act like free living or "planktonic" bacteria; they are 1,000 to 1,500 times more resistant to antibiotics than planktonic bacteria. Plaque on the tooth surface is supragingival plaque. Once it extends under the free gingival margin and into the gingival sulcus (between the gingiva and the teeth or alveolar bone), it is subgingival plaque. Supragingival plaque likely affects the pathogenicity of the subgingival plaque in the early stages of periodontal disease; however, once the periodontal pocket forms, the effect of supragingival plaque and calculus is minimal. Therefore, control of supragingival plaque alone is ineffective in controlling the progression of periodontal disease.

Bacteria in the subgingival plaque secrete toxins as well as metabolic products. Cytotoxins and bacterial endotoxins can invade tissues causing inflammation of the gingival and periodontal tissues. This inflammation damages the gingival tissues and initially results in gingivitis. Eventually, the inflammation can lead to periodontitis, i.e. the destruction of the attachment between the periodontal tissues and the teeth. In addition to directly stimulating inflammation, bacterial metabolites elicit a host inflammatory response. The progression of periodontal disease is determined by the virulence of the bacteria combined with the host response. It is the host response that often damages periodontal tissues.

The inflammation produced by the combination of the subgingival bacteria and host response damages the tooth's soft tissue attachment and decreases bony support via osteoclastic activity. This causes the tooth's periodontal attachment to move apically (towards the root tip). The end stage of periodontal disease is tooth loss; however, the disease creates significant prior problems.

Periodontal disease is generally in two stages, gingivitis and periodontitis. Gingivitis is the initial, **reversible** stage in which inflammation is confined to the gingiva. The gingival inflammation is created by plaque bacteria and may be reversed with a thorough dental prophylaxis and consistent homecare. Periodontitis is the later stage of the process defined as an inflammatory disease of the deeper supporting structures of the tooth (periodontal ligament and alveolar bone) caused by microorganisms. The inflammation results in the progressive destruction of the periodontal tissues, leading to attachment loss. This can be seen as gingival recession, periodontal pocket formation, or both. Mild to moderate periodontal pockets may be reduced or eliminated by proper plaque and calculus removal. However, periodontal bone loss is **irreversible** (without regenerative surgery). Although bone loss is irreversible, it is possible to arrest its progression.

Clinical Features:

Normal gingival tissues are coral pink in color (allowing for normal pigmentation), have a thin, knife-like edge, and a smooth and regular texture. There should be no demonstrable plaque or calculus on the dentition. Normal sulcal depth in a dog is 0 to 3mm and in a cat is 0 to 0.5mm.

The first clinical sign of gingivitis is erythema of the gingiva. This is followed by edema, gingival bleeding during brushing or after chewing hard/rough toys, and halitosis. Gingivitis is typically associated with dental calculus but is primarily elicited by PLAQUE and can be seen in the absence of calculus. Alternatively, widespread supragingival calculus may be present with little to no gingivitis. Calculus itself is essentially non-pathogenic; therefore, the degree of gingival inflammation should be used to judge the need for professional therapy. As gingivitis progresses to periodontitis, the oral inflammatory changes intensify.

The hallmark feature of established periodontitis is attachment loss. As periodontitis progresses, alveolar

bone is also lost. In some cases, the apical migration results in gingival recession. Consequently, tooth roots become exposed and the disease process is easily identified on conscious exam. In other cases, the gingiva remains at the same height while the area of attachment moves apically, thus creating a periodontal pocket. This form is typically diagnosed only under general anesthesia with a periodontal probe. As attachment loss progresses, alveolar bone loss continues, resulting in tooth exfoliation. After this occurs, the area generally returns to an uninfected state, but the bone loss is permanent.

Severe local consequences:

- **oral-nasal fistula (ONF)**
- **class II perio-endo abscess**
- **pathologic fracture**
- **blindness**
- **oral cancer**
- **Osteomyelitis**

Severe systemic manifestations:

Systemic ramifications of periodontal disease are well documented. The inflammation of the gingiva and periodontal tissues that allows the body's defenses to attack the invaders also allows these bacteria to gain access to the body. Studies suggest these bacteria negatively affect the kidneys and liver leading to decreased function. Furthermore, it has been suggested that these bacteria can become attached to previously damaged heart valves (i.e. valvular dysplasias) and cause endocarditis. Oral bacteremias have also been linked to cerebral and myocardial infarctions. Human studies have linked periodontal disease to an increased incidence of chronic obstructive respiratory disease, pneumonia and increased insulin resistance, resulting in poor diabetic control and increased severity of diabetic complications (wound healing, microvascular disease). Diabetes is also a risk factor for periodontal disease. Periodontal disease and diabetes appear to have a bidirectional interrelationship where one worsens the other.

Diet and periodontal disease:

Dry vs moist petfoods

A common perception is that feeding dry pet foods decrease plaque and calculus and canned foods promote them. It would seem that the crunching action of biting into a hard kibble should clean the teeth. Moist foods may have a similar effect to a typical dry food on plaque and calculus accumulation. As the pet bites into a typical kibble it shatters and crumbles, which provides no mechanical cleaning.

In a study of 10 cats, those consuming a dry cereal-based kibble compared to a higher protein/lower carbohydrate wet diet had a more diverse oral microbiome, but with an enrichment of bacteria associated with both gingival health and periodontal disease.

Dental diets and treats

Some dental foods and snacks have a texture which maximizes the contact with the teeth. Foods with the right shape, size and physical structure can provide plaque, stain and calculus control. A 6-month study comparing a dental diet to a typical maintenance diet showed about a third less plaque and gingival inflammation with the dental diet. When a dental diet was fed to Beagles with pre-existing plaque, calculus and gingivitis, there was a significant decrease in these, whereas they increased in the Beagles eating a maintenance diet. The type of fibre in the dental diets exercises the gums, promotes gingival keratinisation and cleans the teeth. Dental treats need to be very hard, and can sometimes fracture teeth.

Additives

Some diets and treats contain antibiotics or additives to retard or inhibit plaque or calculus, e.g. sodium hexametaphosphate (HMP) forms soluble complexes with calcium and decreases the amount available for forming calculus. Adding HMP to a dry diet decreased calculus in dogs by nearly 80%, although another study showed no difference in plaque or calculus when HMP-coated biscuits were fed to dogs for 3 weeks.

Vitamin deficiencies

Deficiencies in vitamin A, C, D and E and the B vitamins folic acid, niacin, pantothenic acid and riboflavin have been associated with gingival disease. These are adequate in diets which meet AAFCO or FEDIAF guidelines but can be deficient in diets which don't meet those guidelines, such as many homemade diets.

Natural diets and feeding raw bones

Proponents of natural foods or of feeding raw bones have claimed that this will improve the cleanliness of pet's teeth; further claims are sometimes made that feeding commercial petfood contributes to the high prevalence of periodontal disease in cats and dogs. However, a study in foxhounds fed raw carcasses, including raw bones, showed that they had varying degrees of periodontal disease and a high prevalence of tooth fractures. The skulls of 29 African wild dogs eating a "natural diet", mostly wild antelope, showed evidence of periodontal disease (41%), teeth wearing (83%) and fractured teeth (48%).

Small feral cats on Marion Island (South Africa) which had been eating a variety of natural foods (mostly birds) showed periodontal disease in 61% cats, although only 9% had calculus. In Australia feral cats eating a mixed natural had less calculus compared to domestic cats

fed dry or canned commercial food, although again there was no difference in the prevalence of periodontal disease. These studies show that a natural diet or raw bones, does appear to confer some protection against dental calculus, but not against the more destructive periodontal disease. There is also the risk of fractured teeth.

Probiotics

Nitric oxide (NO), an important inflammatory mediator, has been shown to be increased in human periodontitis and agents blocking the production of NO or its effects might be valuable. **Lactobacillus brevis (L brevis)**, is a probiotic bacteria containing high levels of arginine deiminiase (AD). High levels of AD inhibit NO generation by competing with NO synthase for the same arginine substrate. In people, topical application of probiotics containing **L brevis** decreased inflammatory mediators involved in periodontitis. Topical **L brevis** CD2 in dogs showed reduction of gingival inflammatory infiltrates,

Summary/conclusion

While the common idea of dry food cleaning the teeth is appealing, many dry foods do not decrease the risk of periodontitis. Similarly, feeding natural foods or raw bones may decrease dental calculus, but does not decrease the risk of periodontitis.

References available upon request

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STATE-OF-THE-ART LECTURE UPDATE ON THE DIAGNOSIS AND MANAGEMENT OF ACUTE KIDNEY INJURY IN THE DOG

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Introduction

The term 'acute kidney injury' (AKI) has been adopted in recent years in both human and veterinary medicine and replaced the old term "acute renal failure". The term AKI is more accurate, as not every injury is associated with a measurable failure. The new terminology also sensitizes clinicians to early identification and intervention. Currently, an increase in serum creatinine concentration as little as 0.3 mg/dL or decreased urine production for more than 6 hours is classified as AKI, even if concentration creatinine is still within the reference range. Yet, in many animals, the baseline creatinine concentration is unknown at presentation and urine production is not quantified. Thus any animal with acute onset of clinical signs, increased creatinine concentration, low urine specific gravity, without any evidence of previous chronic kidney disease, should be suspected for AKI until proven otherwise. Presence of urinary markers as glucose or casts might aid in the diagnosis, however, these are present only in ~30% of the cases, thus are insensitive. Novel sensitive and specific biomarkers are currently under investigation.

The most common etiologies for AKI in dogs can be categorized as follows:

1. Nephrotoxicity, including endogenous nephrotoxins (e.g., hemoglobin, myoglobin) and exogenous nephrotoxins, including ethylene glycol, lilies (cats), grapes and raisins (dogs), medications (e.g., gentamicin) and contrast agents.

2. Infectious agents – leptospirosis, pyelonephritis, sepsis, and FIP in cats.

3. Ischemia / inflammatory.

Medical management

AKI is a systemic disease affecting many body organs, thus the treatment should be focused on the whole patient rather than on the kidney alone. The treatment should focus at identification and elimination of any inciting causes (if possible), controlling the ensued clinical signs and clinicopathologic abnormalities (i.e., retention of uremic toxins, acid-base and electrolyte disorders, changes in blood pressure, decreased erythropoietin production) as well as addressing complications (e.g., respiratory disorders, pancreatitis, disseminated intravascular coagulation etc.). The goal is

to maintain homeostasis and control clinical signs until the kidney recovers.

Hydration status

Animals with AKI are often dehydrated at presentation but may also be overhydrated, mostly due to previous medical treatment that included excessive fluid administration. Although changes in hydration and volemia often parallel, there are some exceptions. An overhydrated patient may be hypovolemic and vice versa. Assessment of hydration status is based mostly on the patient's skin turgor and the mucous membranes, however in patients with uremia this assessment is often challenging. Uremia can lead to decreased saliva production and dry mouth (xerostomia) or alternatively to nausea and salivation. There are only few objective measures that can be used to assess the hydration status. The most readily available tool to assess changes in hydration status is serial measurements of body weight.

The initial treatment should focus in correcting the patient hydration status. Unless there is a contraindication for rapid fluid administration (e.g., heart disease), dehydration should be corrected over 4-6 hours, using the following formula:

$$\text{Volume (L)} = \text{Body Weight} \times \text{\% Dehydration}$$

When calculating the amount of fluids to be administered thereafter, one has also to consider urine production, ongoing losses, and insensible losses. The maintenance requirements of patients with AKI cannot be extrapolated from the required maintenance fluids of normal patients, as some of the AKI patients are an/oliguric while others are polyuric. Therefore, fluids rate should be adjusted individually for each patient.

Patients with AKI should be weighed at presentation and routinely during the treatment period. Once a patient has reached its goal weight, it should be maintained on this weight throughout the hospitalization time. Fluid rate is determined by the rate of fluid loss (urine production, insensible losses and ongoing losses). Fluid administration without close monitoring may result in life threatening overhydration, especially in those patients with low urine production that cannot excrete the fluids administered. Clinical signs of overhydration include serous secretions from the nose, peripheral edema, lung edema, chemosis, pleural effusion and ascites. Overhydration is a common cause for morbidity and mortality in patients with AKI.

Recovery from AKI is often associated with the transition from anuria/oliguria to polyuria. This is a critical point, in which the risk for dehydration is high, and consequently further damage to the kidneys may occur. Polyuric patients may lose a substantial amount of fluids and typically do not drink enough to compensate for the fluid loss, thus fluids have to be administered as needed to maintain normal hydration status.

Diuretics

Once the animal has reached normal hydration status and urine production is still low, the use of diuretics should be considered. Under no circumstances, diuretics should be used to promote urine production in a dehydrated patient. The most commonly used diuretics are mannitol, furosemide and dopamine. Despite their wide use, there is no solid scientific evidence to advocate their use. In addition, like any other drug, diuretics may be associated with side effects, therefore their use should be considered for each case individually.

Mannitol is an osmotic diuretic; therefore, it is active along the entire nephron. Mannitol increases urine production, blood flow to the kidney, and promotes urea excretion. Mannitol is also a free radical scavenger. It is administered initially as a bolus (0.5-1.0 gr/kg over 20 minutes) which is followed by a constant rate infusion at 1 mg/kg/min. Prior to Mannitol administration blood pressure should be controlled, as mannitol may increase the intravascular volume and worsen hypertension.

Furosemide is a potent loop diuretic, which acts on the thick ascending loop of Henle. Furosemide increases the blood flow to the kidney and promotes urea excretion, but it does not have any effect on the glomerular filtration rate. Furosemide is administered as an initial bolus or as a constant rate infusion. In addition to its diuretic effect, furosemide promotes potassium excretion.

The use of dopamine is highly controversial, both in human and veterinary medicine. The recommended dose for AKI is a low (0.5-3.0 µg/kg/min), which, at least in theory, activates only the dopaminergic receptors without activation of α and β adrenergic receptors. Recent studies performed in human medicine demonstrated that although dopamine can increase urine production in some patients with AKI, its use did not change the outcome of these patients (death or the need for dialysis). Fenoldopam is a selective dopaminergic agonist which has been shown to promote urine production and solute excretion in normal animals but in a recent study did not change the outcome or kidney dysfunction in animals with heatstroke related AKI (1).

Controlling clinical and clinicopathologic signs associated with uremia

Gastrointestinal signs result from a direct effect of the uremic toxins on the chemoreceptor trigger zone and from gastrointestinal damage caused by these toxins (e.g., ulcers). The control of gastrointestinal signs is achieved by the use of gastrointestinal protectants (H2 blockers, proton pump inhibitors) and antiemetic

medications (e.g., metoclopramide, ondansetron, Maropitant).

Metabolic acidosis

Metabolic acidosis is a common acid-base disorder of patients with AKI. Metabolic acidosis results from retention of acids in the blood, decreased bicarbonate reabsorption in the proximal tubule, decreased bicarbonate production and decreased hydrogen excretion. Correction of dehydration will eliminate lactic acidosis but patients that remain acidotic need to be treated with bicarbonate, as follows:

$$HCO_3 \text{ (mEq)} = 0.3 * \text{Body weight (kg)} * \text{Base deficit}$$

Treatment should be performed cautiously and over few hours. Rapid bicarbonate administration may lead to paradoxical cerebellar acidosis or metabolic alkalosis.

Hyperkalemia

Hyperkalemia is a common complication of patients with AKI, which results mostly from decreased renal potassium excretion and acidosis. The treatment is determined by the severity of the hyperkalemia and the degree of cardiotoxicity. In mild cases, potassium-free fluids may correct hyperkalemia. In more severe cases dextrose (with or without insulin) and bicarbonate administration are used to shift the potassium intracellularly. When cardiotoxicity is already present, calcium is indicated to protect the heart.

Hypertension

Hypertension is a common complication in AKI. Hypertension may cause severe damage to end organs (eyes, heart, kidneys and brain). In most cases treatment with the calcium channel blocker (e.g., amlodipine) controls hypertension. In refractory cases, hydralazine and nitroprusside can also be considered. Angiotensin converting enzyme inhibitors are recommended as part of the management of hypertension in patients with chronic kidney disease, but should be carefully considered in cases of AKI as they may further decrease GFR and worsen azotemia.

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SVA EXOTICS

RABBIT AND GUINEA PIG DENTISTRY

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Learning objectives for Rabbit and Rodent Dentistry

This lecture will cover the pathophysiology, clinical presentation and approach to staging dental disease in rabbits and commonly seen herbivorous rodents such as guinea pigs and chinchillas. Diagnostic modalities include extraoral and intraoral radiographic techniques, endoscopy as well as computed tomography scans. Treatment of dental disease will be described but prognosis of patients varies greatly based on the stage in which the diagnosis is made. Prophylactic diet adjustments to a high fibre diet should be recommended for all pet rabbits and herbivorous species as dietary habits must be considered as one of the major potential factors resulting in acquired dental disease.

Introduction

Rabbits and herbivorous rodents such as guinea pigs and chinchillas have completely elodont dentition. The incisors, premolars and molars are continuously erupting and the clinical crown will gradually wear down and be replaced by the reserve crown. Dental disease in these species are caused by varying factors however emphasis should be placed at addressing inadequate dietary fibre as this is the leading cause of acquired dental disease. Treatment of affected animals consists of dental procedures such as occlusal adjustments, incisor height adjustments, extractions of severely affected incisor/cheek teeth and surgical treatment of any periapical abscessations. The goal of treatment for dental disease is restoration of elodont teeth to normal length and shape; therefore, improving function. Secondary control of inflammation and infection should also be initiated. Diet adjustments post operatively is also necessary for the full recovery of patients.

Clinical presentations

Rabbits affected by dental disease may present with poor body condition, anorexia, chewing disturbances, changes in food preferences (soft and palatable foods) and hypersalivation. Dental disease can also be accompanied by the development of facial abscesses, wet dermatitis, epiphora, exophthalmia and damage to the temporomandibular joint. Guinea pigs affected by dental disease most commonly present with anorexia and emaciation. They rarely show skin or ocular presentations. Chinchillas with dental disease usually present for weight loss, reduced appetite, poor fur quality, epiphora and hypersalivation. Signs of gastrointestinal disease signs should be closely

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monitored for all species. Dental disease may result in concurrent gastrointestinal stasis due to excessive fur chewing/self-grooming, poor appetite, pain and lack of dietary fibre.

Dental disease in rabbits

There are three main causes proposed for acquired dental disease (ADD) in rabbits: 1) congenital factors (mandibular prognathism, malocclusion), 2) nutritional (reduced fiber intake) or 3) metabolic bone disease (reduced sunlight exposure causing lower calcium levels and higher PTH) These factors may result in improper or insufficient wearing of incisor and/or cheek teeth (CT) as well as formation of periapical abscessations and osteomyelitis. Staging of dental disease in rabbits is performed commonly by performing extra oral radiography and endoscopy. In early stages, mild or absent clinical signs may be seen. However radiographically, elongation of CT crowns is present with slight curving of first premolars. There is slight deformation in the ventral mandibular cortex and interproximal space of mandibular CT may begin to widen. The incisor teeth usually appear normal. In the later stages, the occlusal surface of the CT will be abnormal ("wave mouth") and excessive CT crown elongation and differences in crown height is present ("step mouth"), curvature and deformation of the mandibular or maxillary CT roots may occur, ventral mandibular cortical deformity will become more obvious, eventual root perforation of CT1 and CT2 into cortical bone will occur, fractures of elongated CT frequently occur, abnormal curving and malocclusion of incisor teeth is present. In the late stages, an endoscopic oral exam will reveal excessive crown elongation, deviation of CT and spurs may be seen as well as resulting ulcerations. In the end stages, radiographically there is radiolucency of the mandible, compensatory bony calcifications of the mandible, general lack of normal CT anatomy and incisors and clinical crowns are non-growing/absent or fractured.

Other diagnostic modalities

Intraoral dental radiographic techniques have been described for rabbits and are advocated to allow early detection of lagomorph dental pathology. This technique can be performed in conjunction with extraoral views to aid in visualization and guide veterinarians in providing proper treatment and prognosis.

Computed tomography scans (CT scans) should be considered for advanced to late stages of dental disease to provided more details about the extent of the dental pathology, which is likely to be important for establishing a more precise prognosis and treatment plan for the patient.

Dental disease in guinea pigs

Malocclusion of incisor teeth is less frequent but when seen is typically secondary to CT disease. Hence, they serve as excellent indicators of CT abnormalities. Excessive CT elongation and malocclusion is likely due to poor nutrition causing improper wear. Radiographically, malocclusion and elongation of the incisors may be apparent, deformities of the cortical bone may be seen but are subtler than in rabbits. These lesions are usually more painful in guinea pigs and may not be relieved by coronal reduction. On endoscopic exam, the occlusal angle is sloped more than the normal 45 degree angle from buccal to lingual in severe cases of malocclusion. There will be crown elongation and malocclusion of CT and one or both mandibular CT1 is commonly affected in early stages. This may cause entrapment and discomfort of the tongue. Food and hair impaction is very common. Buccal spurs of maxillary CT may also cause ulcerations.

Dental disease in chinchillas

Malocclusion of incisors are not frequently encountered and usually secondary to dental disease of the cheek teeth. Traumatic fractures of incisors may be encountered and may result in long term overgrowth and deviation. Crown elongation and malocclusion is caused by abnormal wear due to poor nutrition. Radiographically the occlusal surface is uneven, elongation of CT crowns with CT 1 being most commonly elongated in early stages, partial resorption and abnormal curvature of CT may occur and in later stages deformity of the ventral cortical bone is seen with wider interproximal spaces. Malocclusion and elongation of incisors and completely abnormal occlusal surfaces are also common in late stages. Endoscopic exam will reveal excessive elongation of crowns with increased alveolar crest and gingival margins and molar spurs. Proliferation of gingiva may also be present and are usually associated with pain and poorer prognosis. There is excessive wear and even absence of clinical CT crowns in end stage disease.

Dental procedures

Specialised dental equipment such as cheek dilators, mouth gags, rodent table restrainer, periodontal probe, high speed dental handpiece or Dremel and appropriate burs, molar cutters, Crossley's luxators for cheek teeth and incisors, small curved hemotstat and extraction forceps are necessary for the successful dental procedures which may include coronal height reduction, dental extractions, and incisor trimmings. Surgical magnification and light sources are also highly recommended to allow better visualization of the inner oral cavity. The patient should be deeply anesthetized for all dental procedures to prevent accidental trauma due to head and tongue movements. The soft tissue such as the tongue or buccal mucosa should be shielded using an instrument such as a spatula. Moistened cotton tip applicators can be used to periodically clean the working surface of the teeth and remove tooth dust. Spikes and

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elongated crowns should be filed using abrasive burs on a low/high speed handpiece. Overgrown incisors are similarly filed back using an abrasive bur. After reduction in height, the teeth are then filed and shaped to restore normal chisel shaped appearance of the teeth. Extractions should be performed using the specialized Crossley's luxators due to the long tooth roots and care should be taken to ensure entire tooth and its pulp tissue is removed. Extraoral extractions can be attempted when an intra oral extraction technique is not feasible. Indications of extraoral techniques may include retained dental tips, severe root deformities, abscessations and osteomyelitis especially of the mandibular cheek teeth.

Facial abscesses and osteomyelitis

A common sequale of ADD in rabbits and herbivorous rodents are the gradual formation of abscesses resulting from periapical infections. Abscesses are usually not painful, cool to touch, encapsulated in a thick wall and contain a very dense, white and creamy pus. Treatment of abscesses involve aggressive surgical debridement, marsupialization and long-term antibiotic therapy. Mandibular abscesses may develop under the masseter muscles (especially in guinea pigs) and even result in osteomyelitis and jaw deformities.

Dental disease in rabbits and rodents can be very rewarding to treat and most patients will have a good outcome if diagnosis is reached early and timely treatment performed. However in chronic and advanced stages, treatment may also be performed periodically as a form for palliative care. Post-procedural supportive therapy would include analgesics such as opiods and non-steroidal anti inflammatories, antibiotics, fluid therapy, and gastrointestinal motility drugs. Anorexic patients should also be supplemented with a commercial hand feeding formula such as Oxbow Critical Care (Oxbow Pet Products Murdock NE, www.oxbowhay.com).

Diet counselling should be performed for all owners of pet rabbits and herbivorous rodents to prevent ADD. Rabbits and herbivorous rodents should be fed a pelleted diet and ad libitum hay and grass. Commercial mixtures with seeds and other additives are not recommended due to their selective feeding behaviour which will favour ingredients which are high in energy, high in phosphorus, low in calcium and ultimately this does not support optimal chewing patterns. A rabbit's optimal diet should be high in fibre (20–24%) and with optimal calcium and phosphorus ratio of 1.5–2:1. Therefore, only commercial pellets as well as hay and grass with balanced mineral content and ratio should be selected for feeding. Wellness exams and regular weight checks are recommended for all rabbit and herbivorous rodent patients.

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DIAGNOSTIC IMAGING AND GASTROENTEROLOGY (SIMULTANEOUS TRANSLATION INTO MANDARI

APPROACH AND MANAGEMENT OF CHRONIC ENTEROPATHIES IN DOGS

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OVERVIEW

The general term "chronic enteropathies" (CE) has been used frequently in recent years to describe dogs and cats with chronic intestinal diseases of unknown origin. Diet-responsive diarrhea (DRD), antibiotic-responsive diarrhea (ARD), and inflammatory bowel disease (IBD) are different forms of chronic enteropathies. They may form a continuum, with DRD being a mild form of CE, and IBD representing the severe form of the disease.

DEFINITIONS

The following definitions apply to diseases affecting the small and/or the large intestine.

* Chronic enteropathies (CE): a term encompassing all chronic inflammatory intestinal diseases of unknown origin. This includes diet-responsive CE, antibiotic-responsive diarrhea or intestinal dysbiosis, and inflammatory bowel disease (IBD).
* Diet-responsive CE: a form of CE that responds to a dietary trial with an elimination diet based on a novel protein source or the use of hydrolyzed peptides.
* Antibiotic-responsive diarrhea or idiopathic small intestinal dysbiosis: a form of CE associated with a severe imbalance of intestinal microbiota that responds to treatment with select antimicrobials
* Inflammatory bowel disease (IBD): a term used by some as synonymous to CE. Other authors prefer to use IBD to define a more severe form of CE that does not respond to a dietary trial or to a course of antimicrobials, and requires the use of immune-suppressive drugs. IBD is associated with moderate to severe infiltration of the intestinal mucosa with inflammatory cells (such as lymphocytes, plasma cells, eosinophils, neutrophils, macrophages and/or a combination of 2 types). In addition, changes in mucosal architecture such as stunting of the small intestinal villi are also common. Severe IBD may cause protein-losing enteropathy (PLE). Granulomatous colitis of boxers is a particular form of IBD that affects young boxers and bulldogs.

ETIOLOGY

Canine CE are thought to occur as a consequence of a combination of factors that include dysregulation of the intestinal mucosal immune system and its interactions with intestinal microbiota and/or dietary components, and compromised integrity of the intestinal mucosal barrier.

CLINICAL PRESENTATION

CE are characterized by chronic or chronic-intermittent

diarrhea of more than 3 weeks' duration. Mild CE may cause intermittent clinical signs, whereas progressive and severe clinical signs are common in severe cases. Poor body condition with poor hair coat is frequent with severe disease. Some animals may regularly vomit and dehydration is possible. Thickened small intestinal loops may occasionally be palpated. Animals may show pain or discomfort on abdominal palpation. Ascites, hydrothorax and peripheral edema may occur in case of significant protein loss (PLE).

DIAGNOSIS

Diagnosis of CE consists in an elimination process. The aim is to rule out other diseases of known etiology that may cause similar clinical signs: fecal flotation and **Giardia** antigen test should be performed in all dogs. Alternately, empiric parasiticide treatment can be administered (e.g. fenbendazole 50 mg/kg q24h for 3-5 days). Subsequently, the diagnostic process is different for dogs with mild clinical signs and no evidence of systemic complication such as hypoproteinemia, and for dogs that are more severely affected.

Dogs with mild to moderate disease can undergo a treatment trial with a novel protein or hydrolyzed peptide diet, while severely affected dogs showing significant systemic repercussions of their intestinal disease should be evaluated more thoroughly with collection of a minimal database including CBC, serum biochemistry and urinalysis. Presence of hypoalbuminemia, often accompanied by hypoglobulinemia, suggests PLE. Sensitivity of abdominal ultrasound is intermediate, and scans may be normal or show focal or diffuse loss of wall layering, presence of mucosal striations or spicules, wall thickening, enlarged and/or hypoechoic mesenteric lymph nodes. If lesions are present, localization to a specific intestinal segment may be helpful.

Procurement of biopsy samples with upper GI endoscopy or exploratory celiotomy is necessary to further evaluate disease severity in dogs undergoing an in-depth work up. Each method offers different advantages and drawbacks. Biopsy specimen of adequate quality and quantity are required for accurate interpretation by pathologists. The most important justification for histology is to rule out a neoplastic infiltrate. However, it is also useful to evaluate the magnitude of intestinal mucosal inflammation based on the severity and type of the infiltrate and on the severity of the architectural mucosal changes. Current scoring systems are based on results from a WSAVA working group, and take both architectural and inflammatory mucosal changes into account. Inflammatory infiltration may be of varying severity and consist of lymphocytes and plasma cells (lymphoplasmacytic enteritis), eosinophils, neutrophils or macrophages or combinations thereof. Examples of small intestinal mucosal architectural changes include villus stunting, surface epithelial injury, crypt distension, lacteal dilation, and mucosal fibrosis.

DIFFERENTIAL DIAGNOSES

General differentials include intestinal parasitic diseases, bacterial enteritis, fungal enteritis, chronic intestinal foreign body, and diseases originating outside the GI tract such as chronic kidney disease, chronic liver disease, chronic pancreatitis, exocrine pancreatic insufficiency, and atypical hypoadrenocorticism.

MANAGEMENT

In most instances, the goal of treatment is to manage the clinical signs. Full recovery may be possible in mild cases.

After endoparasites have been ruled out (see above), the standard approach for a dog with mild to moderate chronic recurrent diarrhea of unknown origin without significant systemic repercussions is to initiate a food trial with a novel protein or hydrolyzed peptide diet. It is important to collect a detailed dietary history in order to identify the best-suited diet which may be different for each animal. At this time, many clinicians prefer hydrolyzed diets to novel protein diets to treat their patients with diet-responsive CE, although there is no evidence to support their superiority. A thorough discussion about the implementation of the dietary trial with the animal's owners is an indispensable pre-requisite. During the trial, the dog should not receive any other food or treats than the prescribed diet. All dogs with diet-responsive CE show at least a partial response within 10 to 14 days. In dogs with partial or complete response, the dietary trial should be pursued for several months. There does not appear to be any benefit in adding probiotics to the treatment, although the studies published to date included only small numbers of dogs.

In dogs that do not respond to the elimination diet, the next step may consist of another treatment trial with another novel protein or hydrolyzed peptide diet. Antimicrobials may also be used in conjunction with the new diet. Small intestinal dysbiosis (change in the composition of the intestinal microbiota) occurs in most dogs with IBD. Young large breed dogs with CE (particularly but not exclusively German Shepherd Dogs) respond to prolonged treatment with antimicrobials such as tylosin (20 mg/kg PO BID) or metronidazole (10-15 mg/kg PO BID). This may result from an inability of these dogs' immune system to interact adequately with their intestinal microbiota. Clinical experience accumulated over the past decades indicates that prolonged treatment (4 to 8 weeks) is necessary, and that relapses are common. In refractory cases, and in dogs with severe disease and evidence of systemic involvement a more thorough work up with acquisition of a minimal database should be initiated (see diagnosis above).

Dietary management is also an essential part of the

treatment for most other forms of canine CE. Feeding a novel protein or hydrolyzed diet may also contribute to decreased mucosal inflammation in dogs with IBD receiving immune-suppressive treatment. In dogs with lymphangiectasia, a cause of protein-losing enteropathy, treatment with a highly digestible diet with low to very low fat content (10-15% on a dry matter basis) may prevent further dilation and rupture of lacteals by reducing the flow of chyle. This diet significantly contributes to the success of treatment, which often includes anti-inflammatory doses of glucocorticoids. Additionally, the diet should contain highly bioavailable dietary proteins and be low in crude fiber.

All dogs with chronic small intestinal inflammation have limited absorptive capacity, and feeding them with a highly bioavailable diet is necessary in order to improve metabolic state and stop ongoing catabolism. As stated above, the goal of treatment in dogs with various forms of IBD is to successfully manage the disease. Therefore, appropriate dietary therapy should be administered long term and regularly adapted to the dog's condition in order to decrease the risk of recurrences.

PROGNOSIS

The prognosis for diet-responsive CE is good. It appears that 77-80% of these dogs are still controlled only with an elimination diet one year after diagnosis. However, several studies showed that the prognosis of dogs with CE that do not respond to a dietary treatment trial is significantly worse, with up to 1/3 of patients euthanized within 3 months of diagnosis. Moreover, severe hypoalbuminemia (serum albumin < 2g/dL or <20 g/l) at the time of presentation was identified as a negative prognostic factor.

VALUE OF DIET IN OTHER CHRONIC ENTEROPATHIES

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NURSES (HILLS)
THE ART AND SCIENCE OF BANDAGING

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THE ART AND SCIENCE OF BANDAGING
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Classification of wounds

The appropriate treatment of a wound is dictated by the condition of the tissue at time of presentation. Wounds are classified in the following manner:

- Clean
- Clean-contaminated
- Contaminated
- Dirty

Clean wound - Example: planned surgical incision.

- A 'clean' wound is a relatively non-traumatic surgical wound, made under controlled conditions, aseptic preparation, minimal trauma and in a very clean or sterile environment.
- 'Clean' wounds are sutured soon after they have been created.
- Incisions entering the oropharynx, urogenital, respiratory or alimentary tract are not classed as clean wounds.

Clean-contaminated wounds - Example: laceration less than a few hours old

- There is some trauma to the tissues and the conditions are non-sterile.
- The wound is contaminated with environmental bacteria – either from the object causing the wound or from the normal flora of the animal.
- These wounds are able to be cleaned and sutured if they are presented before the bacteria has had chance to proliferate the tissue, known as the 'golden period'.

Contaminated wound - Example: degloving from road traffic accident

- The wound is clearly soiled or contaminated with bacteria.
- It has often been exposed for longer than 6-8 hrs.
- This type of wound often requires aggressive cleaning and staged treatment techniques to facilitate an uncomplicated healing.

Dirty Wounds - Example: Cat fight abscess

- Also termed 'infected' wounds
- An abscess has formed as a result of infection
- A wound care plan should be implemented for each individual wound depending on its cause and classification.

Types of wounds

The cause and type of the wound is a major factor in the treatment technique and long-term plan. They can be initially classified as open or closed.

Open wound

- Incision
- Laceration
- Puncture
- Abrasion
- Burns and scalds

Closed wound

- Contusions
- Crush injury

Incised wound

- Caused by sharp cutting instruments, e.g. knives, glass
- Edges are clean cut and defined
- Generally the wound will gape open
- The wound is usually quite deep
- The wound can be of any size

Lacerated wound

- Generally caused by road accidents, dog-fights, tearing by barbed wire etc
- Wounds are irregular in shape and generally gape open
- Edges are jagged
- Will usually be contaminated – dirt, debris etc
- Very painful

Puncture wound

- Caused by small sharp pointed objects, e.g. fish hook, cats teeth
- Small wound – can easily be overlooked
- Generally causes a deep wound – tracks down through tissues

Abrasions

- Also known as 'grazes'
- Caused by such incidents as road traffic accidents and animal is dragged along the ground
- Wound does not penetrate the whole of the skins thickness – it is superficial, can be of any size
- Very painful
- Wound is generally contaminated.

Wound Care

The initial goals of wound care are to prevent contamination and protect the tissues. On presentation the wound should be covered. Saline soaked swabs are effective to prevent contamination but also protect exposed tissue from drying.

Clip Hair

The hair surrounding the wound should be clipped. Hair should be prevented from entering the wound. Instilling sterile gel into the wound prior to clipping should prevent this. The sterile gel can then be flushed from the wound with sterile saline once the clip is completed.

Flush wound

The wound should be lavaged with fluids under moderate pressure to remove debris and bacteria. Warm isotonic solutions (0.9% NaCl) are preferred. Hypertonic solutions may be used if oedema is present.

Antiseptics and soaps are NOT recommended, as they are irritating to the tissues and delay healing. However, to reduce bacteria a very dilute chlorhexidine or povidine-iodine solution may be used.

Debridement

Debridement of necrotic or devitalised tissue may be required to enhance healing. It may be performed layer-by-layer or a large excision in one area. The wound should be surgically prepared, placing sterile gel in the wound during preparation so prep solutions do not enter the area. Once debridement is performed, the closure method may be decided.

Closure

Closure may be performed (depending on the type of wound) by suturing the wound. It may be necessary to delay wound suture until further debridement's are performed. If the wound cannot be closed then healing takes place by second intention.

Drains

Drains may be placed in the wound. They are generally made of flexible non-absorbable tubing. They are placed to:

- Establish drainage of fluids from 'dead' space
- Prevent accumulation of fluids or exudates in the wound
- Maintain drainage of fluids during the debridement stage of healing
- Drain exudates from an infected wound.

Dressings

There are many types of dressings or gels available to promote wound healing. They are the primary part of the bandage applied to the wound.

The type of dressing or gel used depends upon the current stage of healing and the objective of the wound treatment.

- For infected wounds use dressings that reduce the bacterial load (e.g Acticoat™ , Iodosorb™). Use in conjunction with systemic antibiotics.

- For wounds with heavy exudate, use dressings that lift the discharge away from the wound and store it in the

dressing (e.g Allevyn™, Absorb Plus™)

- For dry wounds which are at risk of desiccation, use dressings which restore moisture balance (Duoderm™, Instrasite Gel™, sterile saline soaked swabs)

Wound Healing

There are several stages of healing that occur sequentially over a period of time. The healing may occur at different rates in various areas of the same wound.

Stages of Healing

Divided into the following phases:

- Inflammatory
- Debridement
- Repair
- Maturation

Inflammatory Phase

6-8 hours post wound occurring

- Immediate vasoconstriction and then vasodilation
- Clot is formed at the site of the injury to prevent further haemorrhage
- Plasma like fluid is produced to assist healing
- Erythema, heat, swelling and pain develop

Debridement Phase

6 hrs – 5 days post wound occurring

- Cellular activity is stimulated by the inflammatory process.
- This results in the production of exudates and discharge from the wound.
- The discharge has a cleansing effect as necrotic debris, white blood cells and tissue fluids are removed with the discharge
- Discharge = 'pus' which can be sterile or indicative of a bacterial infection.
- Performing cytology and looking at the discharge can determine proper wound care.

Repair Phase

Starting 3-5 days post wound occurring and lasting 3-12 days

- Blood vessels grow into the wound
- Granulation tissue forms to fill the wound area and epithelium begins to form along the edge of the wound
- Collagen is laid down to give the wound strength
- The wound becomes smaller in diameter as the granulation tissue starts to contract.
- Healthy granulation is red and shiny; it bleeds easily but is not painful.

Maturation Phase

- Begins 17-20days post wound occurring and can last up to 2 years
- This is when the collagen fibres in the connective tissue become replaced and realigned to give the area greater strength. The scar becomes pale and less obvious.

Wound Strength

0-5 days	Very little strength during the inflammatory and debridement stages
5-21 days	During the repair and maturation phase the wound strength starts to increase
21+ days	As the scar matures during the later maturation stage the strength will increase, but the final strength of the area is about 20% weaker than the original tissue.

Factors in Wound Healing

Wound healing can be influenced not only by the type and depth of contamination (if present) but also the condition of the patient.

In order for healing to take place efficiently it is essential that:

- The area has a good blood supply. Functioning white blood cells, fibrinogen and numerous other substances need to get to the area. Debris and waste products need to be removed. Oxygen is need by all the cells to function well
- There is a good supply of the materials needed to conduct the repair.

Many factors that delay wound healing involves interference with the above two factors.

Wound factors that delay healing include:

- Poor blood supply – fails to carry healing cells, chemicals and oxygen to the areas as well as waste products and debris away from the area
- Dead space with accumulation of fluid – interferes with local blood supply
- Infection – negatively affects white blood cell function
- Foreign bodies or debris present – creates a persistent infection
- Oedema – interferes with blood supply.

Patient factors that can delay healing include:

- Age (geriatric) – reduced circulation and immune function
- Systemic disease (diabetes, liver or renal dysfunction) reduced immune function
- Obesity – impaired circulation
- Malnutrition (low protein levels) – poor supply of repair materials
- Cancer and cancer treatments – reduced immune function
- Some medications – reduced immune function
- Self-trauma – impairs local blood supply.

Wound Healing Complications

Recognising wound complications can assist in appropriate treatment being implemented as soon as possible. Complications may include:

- Oedema

The area becomes puffy or spongy and pale in colour. It may produce a watery fluid. When pressed gently with

the finger an imprint may be left for a few moments – this is called pitting oedema.

- **Devitalised Tissue**

If the tissue displays an abnormal colouring such as purple, grey, white, green or black – the area is becoming devitalized. This means that the blood supply to the tissue is decreased or absent. The devitalized tissue will undergo necrosis and may liquefy or become dry and leathery. When the tissue becomes black and leathery, this is termed 'eschar'.

- Dehiscence

This occurs when the wound repair breaks down and the wound becomes open again.

- 'Proud' flesh

Also termed exuberant granulation, this occurs when the granulation tissue is above the level of the skin edges.

- Infection

The wound will discharge an odourous exudate, with inflammation at the site and bacteria contained within the fluid.

- Seroma

Fluid accumulation at the wound site. A fluctuant swelling or mass containing fluid is palpable at the site. This can be a seroma, haematoma or abscess. A Fine needle aspirate would be required to determine the type of fluid and treatment course.

- Fistula

This is a persistent non-healing opening that often produces exudates.

- Contracture deformity

This most often occurs when a wound is around or near a joint. As the wound contracts and undergoes maturation, the scar tissue may restrict the range of movement.

- Hypertrophic Scar

Over time the scar should become less obvious, however, a hypertrophic scar will become more obvious appearing raised, thickened and prominent.

Bandaging

The general objectives of a bandage are to:
Provide support for:
- Fractures
- Disclocations
- Sprains, strains the fractures

Protect against
- Self-mutilation
- Infection
- Environment
- Further injury

Pressure to:
- Stop haemorrhage
- Prevent or control swelling

Provide comfort and pain relief Immobilise
- Limit joint movement
- Limit mvement of fracture sites

Bandage Formula

A basic bandaging formula is:

Initial layer – dressing (applied directly over the wound)

Primary layer – padding (for comfort, support and absorbtion of any exudate)

Secondary layer – conforming (provides strength and conforms to the contours of limb and secures dressing and padding)

Tertiary layer – protection (protects the bandage and provides further security).

Types of Dressings (Initial layer)		
Dry	Sterile plain gauze swabs	
Impregnated	E.g. with petroleum gel or antibiotic	Jelonet®
Semi-occlusive	Usually have a layer of permeable non-stick material on one or both sides. May have absorbant core. Some have adhesive section around the edge to enable stable and accurate placement of dressing	Melolin® Cutiplast® Cutilin®
Absorbant	these can be made of various materials and are usually quite thick – used for wounds with large amount of exudate	Allevyn®

Types of Padding (Primary layer)		
Cotton Wool	Natural or man-made	Cotton Wool Rolls
Padding bandage	Natural absorbant material supplied in rolls Easier application Great for limbs	Velband® Soffban Natural ®
Synthetic padding	Thinner and lighter than padding bandage and cotton wool	Soffban®
Cotton wool and gauze	Cotton wool that is sandwiched between layers of gauze and supplied in a roll	

Types of Conforming Bandage (Secondary layer)		
Conforming	has an elastic component to enable 'conformation' care must be taken not to apply too tightly	Handyband®
Loose open-weave	has no elastic component loose weave assists with bandage conforming	Handygrip®
Crepe	washable cotton fibre on a role not commonly used	Elastocrepe®
Tubular	elastic net bandage supplied in tubular format	Surgifix®

Tertiary Layer		
Adhesive	thick, cotton based material with an adhesive side	Elastoplast®
Cohesive	material containing latex that 'sticks' to itself but not to the skin or hair	Co-flex®

Bandage Assessment

When the bandasge has been placed, regardless of the type or area, it should be assessed using the following questions:

- Is the bandage achieving its aim?

(Immobilising the correct joint, not slipping, in correct position)

- Is the bandage comfortable?

(Check that the bandage is not too tight or causing discomfort to the patient – no chewing or interference from patient)

- Is the bandage suitable?

(The bandage is not interfering with the other general movement of the patient).

Bandage Care

Once a bandage has been applied and assessed it should be frequently checked until removal, this includes:

- Ensure the bandage is not too tight
- Check that it is not casuing the patient discomfort / pain
- Check in correct position (not slipped)
- Check for:
    - Odour
    - Discharge
    - Wetness

- **The bandage should be removed and the source investigated**
- Contamination / dirt
- Check above and below the bandage for:
    - Skin inflammation / redness
    - Oedema
    - Heat or coldness

It is vital that the bandage is kept clean and does not become soiled or wet from environmental factors (urine, faeces, wet ground etc). If soiling occurs the bandage must be changed.

If a limb is bandaged, the bandage can be protected from the environment (e.g. when toileting outside) by placing a protective covering over it. For example, a clean dry used drip bag can have the bottom cut from it and tied over bandage or use a plastic bag in a similar manner.

WSAVA GLOBAL PAIN

MECHANISMS OF PHARMA, NON-PHARMA AND SUPPLEMENTS IN TREATING PAIN

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A thorough review of pain physiology MARKEDLY lubricates the conversation around the mechanisms of analgesia- both pharmacologic and non-phamacologic.

In this SHORT lecture on the topic we will quickly cover the major contributors of pain physiology, and how treatments address these sub-categories in the generation (or treatment) of pain.

Periphery: The triad of elements making up nerve ending are all modifiable.

Nerve endings in the periphery are blocked with a laundry list of sodium channel blockers (lidocaine, bupivacaine, etc) that block a particular subtype of sodium channels known as tetrodo-toxin sensitive channels. These drugs are relatively indiscriminate about which types of nerves that are blocked (bupivacaine and ropivacaine may slightly favor sensory), although the kinetics of drug penetration tends to favor blockade of thinner, less myelinated and less deeply bundled nerves. Sensory fibers carrying pain, heat and touch sensations are readily altered while thick, myelinated motor fibers are slower to affect.

As with the local anesthetics, a wide variety of nerve endings are present in the periphery and not only pain fibers are affected by interventions. Stimulating heat or cold-sensing receptors, touch sensing receptors, etc sends competing signals to the dorsal horn of the spinal cord. These stimuli decrease the amount of afferent stimuli reaching being received. This is one method by which heat, cold, touch, massage, and acupuncture play a role at the nerve endings. This particular aspect of descending inhibition was targeting by early pain pioneers Melzak and Wall in their 'gate theory'. Thermotherapy (ice/cold) also chemically inhibit pain signals through their effect on TrP channels in peripheral sensory nerves.

Finally, the mast cells and capillaries are critical. Local release of inflammatory mediators set an avalanche of events into action. Interventions at this level include peripherally acting anti-inflammatories such as non-steroidal drugs (NSAIDs) and steroids. A large laundry list of nutritional supplements act as anti-oxidants and have some effect and reducing inflammation (Boswellia, Curcumin, etc) Cooling, photodynamic treatments such as low-level laser therapy and acupuncture also are likely to have direct affects on both decreasing inflammation

and improving lymphatic and capillary drainage from the region of the triad.

Axon The most effective way to interfere with electrical signaling is to interfere with sodium channels, so here we see the return of the local anesthetics.

When used along a major axon use of local anesthetics is generally termed 'regional anesthesia' and this is clearly a hallmark of food animal practice. However, a huge resurgence has occurred across all types of human and veterinary species with fantastic new applications. The advantage to targeting the axon with local anesthetics is that it avoids placing drug right at the site of the injury. Both the biology of injured tissue (acidic, edematous, etc) and the pharmacology of the local anesthetics (pH sensitive, anti-inflammatory, painful on injection, nerve damage with repeated administration) provide interactions that are avoided by placing drug remote to the injury. A disadvantage to using a local anesthetic along the axon is that, although the signal is blocked from entering the dorsal horn, the activity continues and the peripheral site of injury. Therefore the release of inflammatory mediators, the uptake of these mediators into the bloodstream and passing of the signal to the DRG still occurs. Therefore the interference into the pain processing is not as complete as once thought when a regional technique is used. Some of the exciting advances in regional techniques have come about through improved accuracy of drug placement. Nerve stimulation and/or ultrasound are fantastic methods for verifying needle placement. An excellent review by Luis Campoy titled: Fundamentals of Regional Anesthesia using Nerve Stimulation in the Dog can be found on www.ivis.org.

Photobiomodulation (low level laser therapy) plays an important role at the axonal level. Pain signaling has been shown to decrease with photodynamic therapy, and one of the proposed mechanisms is disruption of axonal flow due to disruption of flow along the filaments within axons. Furthermore, cytochrome c activity is amplified, which provides a pro-metabolic, immune modulatory effect.

Likewise, using sound for biomodulation with shock wave therapy has been shown to improve tissue healing and reduce pain in tendon, bone and skin injuries.

Cell Body (in the DRG) Therapies directed at the cell body generally fall into the chronic pain category. Increased expression of a variety of ion channels and receptors accompany chronic pain states. Opioid receptors are synthesized, packaged and sent to peripheral locations. Sodium channel sub-types are slowly switched to tetrodo-toxin resistant types (not-responsive to traditional sodium channel blockers). Likewise, calcium channels are upregulated and different sub-types emerge. Cyclo-oxygenase subtypes increase

(COX 2 specifically) in the cell body and terminals.

Therefore, the cell body in the DRG is an important target for transcriptionally directed therapies. NSAIDs play some of their pain-relieving role here and both the centrally acting and peripherally acting NSAIDS probably have some access to the DRG. Steroids work in this location both by interfering with COX and also by causing transcriptional changes. Acetaminophen serves a COX 3 modifying role in the DRG and dorsal horn, although it does not serve as an anti-inflammatory in peripheral tissues.

Anti-epileptic drugs often target high-use receptor subtypes of sodium and calcium channels. Drugs such as Phenytoin, Carbemazepin, Zoneisamide and Lamotrigene target high-use sodium channels. Gabapentin and Pregablin target calcium channel expression.

Although most of the cell body therapies are directed at chronic changes, it has recently been recognized that the cell body is highly involved in the effect of low-concentration systemic lidocaine administered via constant rate infusion (Lidocaine CRI). While this method has become increasing popular due to analgesia and pro-motility effects on the gastro-intestinal system, the mechanism of action has been unclear as it has been shown that the doses are too low to justify a blocking effect on peripheral tissues, and the drug is excluded from the central nervous system by the blood-brain barrier. Meclizine is an orally-available sodium channel blocker of the same class as lidocaine and can sometimes be useful in pain states that respond to lidocaine infusions.

Dorsal Horn An entire graduate course can be filled with treatments directed at decreasing pain signaling through the dorsal horn. We have already discussed descending inhibition from activation of non-pain sensory fibers sending competitive signals through lamina 1-5 in the axon section. We have discussed several of the transcriptionally mediated changes to ion channels above in the cell body section. The bulk of the cell body discussion plays out in the dorsal horn, which is, after all, an extension from the cell body.

Glutamate and substance P are major players in synaptic conductance; however, their roles are too diverse throughout the body to serve as good specific targets for pain therapy. The major receptors for glutamate are AMPA, NMDA and metabotropic glutamate receptors. The NMDA receptor tends to remain quiescent in the dorsal horn (it is much more active in the brain as it serves an important role in learning). It becomes activated only when enough activity passes through the synapse to dislodge a Mg ion that occupies the pore. However, 'sustained activity' to a nerve is really only 10-15 minutes or less, once again pointing out

some different perceptions between physiologists and clinicians (who tend to see chronic stimulation as occurring over weeks to months). Once activated this receptor massively amplifies calcium handling and glutamate receptor activation - like a supercharger for a pain stimulus. Thus, it is a great target. Ketamine, Amantadine, dextromethorphan and methadone have NMDA antagonist effects.

Inhibitory receptors are also routinely expressed at both the pre-synaptic and post-synaptic membranes. Three major examples of these ligand-receptor pairs are opioids (Mu Kappa and Delta receptors), serotonin (5HT), and Norepinepherine (Alpha-2 receptors). When bound to the channel these ligands activate second messenger systems that modify channel kinetics- hyperpolarizing membranes, closing ion channels and interfering with cellular messenging. GABA is an important messenger at the dorsal horn that unfortunately has had mixed results when targeted for pain modulation. For example, benzodiazapines are GABA agonists, but have a variable effect on pain perception ranging from anti-nociceptive to pro-nociceptive in different studies and at different drug concentrations.

Opioid agonists (morphine, meperidine, hydromorphone, oxymorphone, fentanyl) and partial agonists (butorphanol, buprenorphine, nalbuphine) bind to receptors that are repleat through all levels of the pain processing system. Opioid receptors work via channels (as mentioned above) as well as stimulating inhibitory interneurons. The number of recognized receptor subtypes continues to expand, shedding some light on some important differences in individual responses to different opioids. In general, agonists tend to provide more potent analgesia than partial agonists. It is important to note, however, that opioids may bind to either inhibitory linked g-proteins (decreasing pain signaling) or excitatory linked g-proteins. With chronic administration the excitatory-linked receptors are increasingly manufactured- leading to tolerance and forms of dysphoria or excitement. Several opioids have been shown to directly stimulate glial activation (see section below). So, while opioids are the cornerstone of acute pain management, they express increasing limitations in the treatment of chronic conditions. Dogs have an extremely effective first pass metabolism of narcotic drugs, and the only oral narcotic that has shown any levels in the plasma of dogs is codeine.

Tramadol has been utilized as an alternative to opioids because of its oral bio-availability in humans. It is an opioid-like drug with about 10-20% of the efficacy of morphine at mu receptors (in people- likely very minimal opioid effects in dogs). It's effectiveness is improved via additional effects on the noradrenergic and seratonergic systems in humans. In dogs, there is controversy about the efficacy or oral tramadol, although parenteral

tramadol has been shown to be analgesic. Like tramadol, drugs commonly regarded as 'anti-anxiety' medications generally target noradrenegic and seratonergic systems in the brain and spinal cord. Amitriptyline and Imipramine are common examples in veterinary medicine.

Specific alpha-two agonist drugs such as dex-medetomidine, romifidine and xylazine also cause a tremendous amount of sedation, making them very useful for acute pain therapy in individuals who would benefit from sedation but less useful in the management of chronic or ongoing pain.

Before leaving the topic of the dorsal horn, a more direct way to maximize drug effects in this location (or to have drug effects in the case of drugs that cannot penetrate the blood-brain barrier) is to administer the drugs by epidural or spinal routes. The most common drugs used in this way are opioids, local anesthetics, alpha-two agonists and ketamine. Epidural catheters may be placed for long-term administration of drugs through this route. Finally, mild electrical stimulus applied to the spinal cord dramatically reduces some forms of neuropathic pain. Spinal cord stimulators are available for this purpose, although application in non-speaking populations poses some difficulty for post-placement assessment.

Brain stem and cortex Serotonin, norepinepherine and endorphins/opioids are the most well-understood modulators of pain processing in the brain. In addition, psychological state has a profound influence on pain processing. So, in addition to the generally recognized analgesic drugs, anxiolysis becomes a critical feature in modifying pain messages in the higher centers. Acepromazine is a profound anxiolytic that has a solid place in pain management despite the fact that it is not independently analgesic. However, natural forms of anxiolysis are probably far superior and include touch, companionship, being at home, reduction of stressful inputs, etc. Acupuncture studies using functional MRI have shown reduced activation of brain areas commonly associated with stress.

Glia Glia are constitutively active support cells that can be upregulated to perform immune functions in the central nervous system. They are activated by transmitter over-flow from the synaptic cleft as well as specific compounds (fractaline) released by active neurons. Astrocytes communicate among one another of great distances by non-synaptic gap-junctions and in turn activate microglia through the release of glutamate, cytokines and other proteins. Opioids can directly stimulate glial activation as well. In addition to some less common therapies (such as pentoxyphylline), Centrally acting NSAIDs slow glial activation. Glial activation secondary to opioid use can be slowed or prevented by co-treatment with NMDA antagonists, gabapentin, or low-dose opioid antagonists. Much information has yet to be gained as the glial component has only been reported in

the last 5 years.

Immune system Already covered at each step in pain processing, the immune system is implicitly linked to even the most acute pain signaling. Systemic reductions in inflammation with steroids, NSAIDs, and many supplements can reduce tissue damage, to both the nervous system and the other collateral systems (musculoskeletal, myofascial). Acupuncture provides immune modulation both systemically and regionally. Cooling and gentle massage have regional effects on immune function.

Muscle and connective tissue Muscle and connective tissue sequella are inevitable with any sort of amplified pain processing, both through guarding of the painful region as well as bystander activation from neuronal and glial amplification. Systemic muscle relaxants such as methocarbamol may aide in reducing muscle tension. Specific regional techniques to reduce muscle tension are generally superior and include: acupuncture, low-level laser therapy, ultrasounds therapy, transcutaneous electrical stimulation, massage and physical therapy. Fascia is recently recognized as having proprioceptive and sensory capacity, and also plays a major role in both the generation of pain, and the treatment of pain and proprioceptive deficits by physical medicine modalities.

Caution is advised when adding these modalities to your practice as there is significantly less regulation of these affiliated professions. Verify an appropriate evidence-based training or go through validated training programs yourself.

Bone and joint Clearly, the most physiologically function a body region, the less pain and accommodation will need to occur. Definitive surgical correction should always be pursued when available. Additionally, many methods are available to augment bone and joint function. Inflammation is a key component to the demise of cartilage, and anti-inflammatory products are irreplaceable in this setting. Other products that may have an impact in reducing joint inflammation are glycosamino-glycan products such as adequan. While the presence of articular cartilage may improve the effect of GAGs, there is also evidence that decreased inflammatory mediators (such as IL-1) follow treatment and may help joint comfort even when little normal cartilage remains. Intra-articular administration of Hyaluronic acid takes this approach to a more direct level. Intra-articular steroids have some potential harm for damaging cartilage (depo-medrol), although other studies (especially in horses) have shown a cartilage sparing effect of triamcinolone when combined with HA. These injections may have an important role to provide comfort in end-stage joints and facilitate physical therapy. Biologicals such as PRP or stem cells may also improve joint comfort, although more research is needed in these areas. Using local anesthetics in joints has recently come under fire, as changes in cartilage healing has been found with infusions of bupivacaine directly into joints.

Nutritional supplements directed at cartilage and joint function include fatty acids, soy and avocado insaponifiables, glucosmine, chondroitin, MSM, elk-velvet antler, green-lipped mussle, milk-based products such as duralactin, myristol, herbals such as dandelion, boswelli, turmeric: etc. Many of these products have merit, some more validated than others. Omega-three fatty acids (50 mg/kg EPA+ DHA) have the highest level of evidence, and are therefore commonly recommended. There are many supplements available, and while they may be helpful, they may also serve to direct money and energy away from validated therapies. Those of animal origin have other concerns, such as disease transmission and ethical harvesting. Whenever possible, it is recommended to favor products from companies that are pursuing scientific validation for their products. Consider avoiding glucosamine/chondroitin containing products in spinal cord until further evidence is found that it won't inhibit central nerve healing.

WSV18-0226

CLINICAL PATHOLOGY

CASE STUDIES IN CYTOLOGY

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CLINICAL PATHOLOGY: CASE STUDIES IN CYTOLOGY

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Can you identify those common tumors in the skin and subcutis? Do you feel a little overwhelmed when trying to differentiate inflammation from certain tumors? And what makes mesothelial cells so confusing? Whether it comes to FNAs of common skin lumps and bumps and/or cells in body fluids, this seminar will focus on a series of case studies which will go through several common and some unique cases you may or may have not seen in your clinic. By the end of this seminar, you should leave feeling more comfortable differentiating inflammation from neoplasia and interpreting cells in body fluids (cavitary effusions, nasal, bladder, and prostatic washes), including those mysterious mesothelial cells.

WSV18-0172

ISFM - FELINE CARDIOLOGY

WHICH CATS WITH ARTERIAL THROMBOEMBOLISM IS IT ETHICAL TO TREAT?

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Arterial thromboembolism: what we know

Arterial thromboembolism (ATE) is a condition associated with high morbidity and mortality in cats, most commonly with an acute and distressing presentation. For clinical purposes, it is often defined as thromboembolism to ≥1 limb. In most cases, the thrombus originates in the left side of the heart; feline ATE is most commonly associated with cardiomyopathy, although cardiac disease is not present in all affected cats. Although pulmonary thromboembolism also involves arterial occlusion, it usually is classified as a separate syndrome. Presenting signs of limb ATE are easily recognized. Loss of peripheral pulses, tissue pallor, lower motor neuron signs and cool extremities in the presence of neuromuscular pain provide a highly suggestive clinical picture. Smith et al reported that 1/175 (0.6%) of their hospital feline population presented with ATE, a similar prevalence to 1/142 cats (0.7%) reported by Buchanan et al from a different centre, almost 40 years earlier. These data were obtained from cats treated in referral practice and do not necessarily represent the general feline population. Borgeat et al (2014) published findings in 250 cats with ATE in general practice, and prevalence was 0.26% - less than half that reported from referral centres.

It is widely acknowledged that ATE has a poor prognosis, although no prospective studies have reported the outcome of cats presenting with a first episode of acute clinical signs. Several retrospective studies suggest that euthanasia at presentation is common and <50% patients survive to discharge. Smith et al reported that hypothermia, ≥2 affected limbs, absence of motor function, hyperphosphataemia and bradycardia were associated with a decreased rate of survival to discharge. Moore et al also reported that hypothermia and ≥2 affected limbs were associated with death or euthanasia before discharge. It has been suggested that cats in congestive heart failure (CHF) have a shorter survival time after discharge (Smith et al, 2003). Among cats discharged from the clinic, recurrence of ATE is common.

In first opinion clinics, rectal temperature is also vital to assess at presentation: lower rectal temperature was crucial in predicting death at 24h and before 7 days of an ATE episode (Borgeat et al, 2014). Interestingly, this retrospective study also reported that treatment with an anti-platelet agent (aspirin, clopidogrel, or both drugs)

was associated with an increased likelihood of survival to 7 days. We believe that immediate analgesia and anti-platelet agents are the standard of care for cats with an ATE, and if rectal temperature is particularly low or pain cannot be controlled, we recommend euthanasia as a viable treatment option.

Of cats treated for ATE in general practice, survival to 7 days is estimated at 55%. Of those cats alive at 1 week after an acute ATE episode, 1 year survival was 20%. Despite mortality being high in this population, and recurrence of ATE approaching 80%, quality of life in between episodes is often good and longer-term survival is possible (Borgeat et al, 2012).

Approach to treatment of ATE
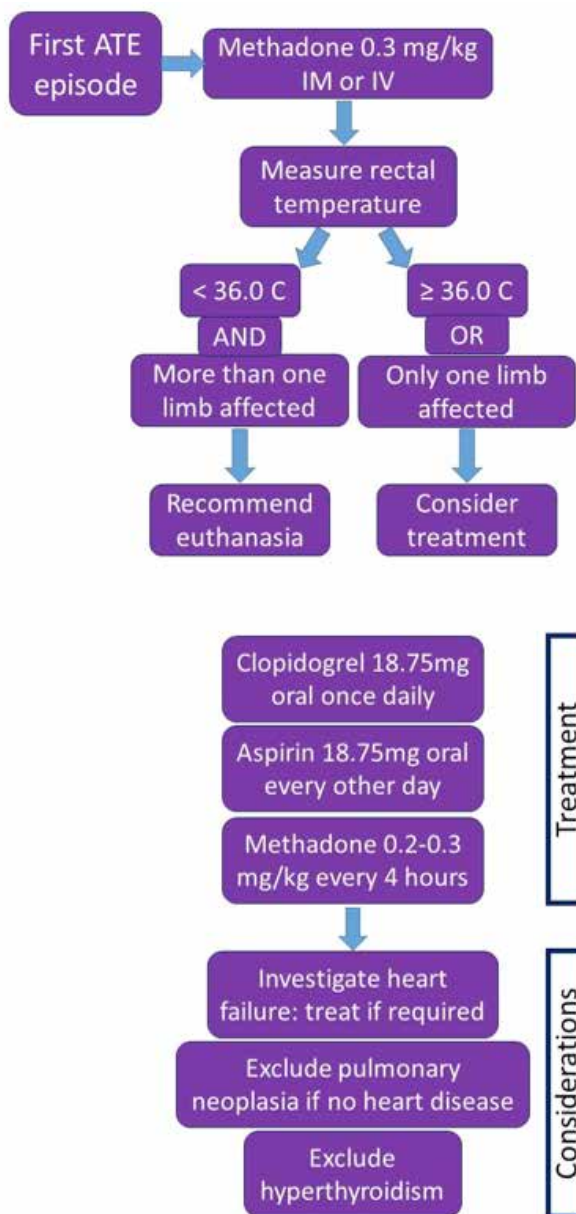
Why we should choose clopidogrel over aspirin: the FAT CAT study (Hogan et al, 2015)

Over several years, the feline arterial thromboembolism clopidogrel vs. aspirin trial, or FAT CAT study, recruited cats that were at risk of ATE. Because identifying risk is difficult, the researchers recruited cats that had suffered an episode of ATE and had survived for 3 months (so they were known to have stable disease, and would not die or be euthanised as a result of acute consequences of the first ATE). Cats were randomised to receive either clopidogrel (ADP receptor antagonist) 18.75mg once daily or aspirin (thromboxane receptor antagonist) 18.75mg every third day. Cats were followed until either ATE recurrence, or death because of heart disease.

This study showed that cats receiving clopidogrel lived for an average of 442 days before ATE recurrence, compared to an interval of 192 days observed in the aspirin group. Despite its more frequent dosing interval (and apparently a bitter taste to around 10-20% of cats), the drug is highly effective at preventing ATE, so should be considered the standard of care in cats with heart disease at risk of ATE: i.e. those with LA dilation or poor atrial function.

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43RD WORLD SMALL ANIMAL VETERINARY ASSOCIATION CONGRESS AND 9TH FASAVA CONGRESS

WSV18-0295

SVA SOFT TISSUE SURGERY

ANAL SACCULECTOMY

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Key Points

- knowledge of anorectal anatomy and neuroanatomy is important to protect vital

- structures

- remove all anal sac epithelium during anal sacculectomy

- use of a Mila Anal Sac catheter or Foley catheter will facilitate anal sacculectomy

If you would like a video of this surgical procedure on DVD go to www.videovet.org or contact videovet@me.com.

Introduction: Disorders involving the anus and rectum occur frequently in small animal practice. In order to appropriately diagnose and treat these disorders, knowledge of the regional anatomy, physiology, common clinical signs they produce, and proper physical examination techniques are necessary.

Anatomy: The location and function of the following anatomic structures should be reviewed prior to surgical management of diseases of the anus and rectum: internal and external anal sphincter muscle, anal sac and duct, circumanal glands, caudal rectal artery, vein and nerve, and columnar zone of the anus. These structures are commonly involved in many of the disease processes discussed below and their preservation or removal plays an important part in the patient's ultimate recovery.

The Anal Sphincter Muscle (From the introduction of a report on hemorrhoidectomy written by WC Bornemeier and published in Am J of Proc, Feb, 1960.): "The prime objective of a hemorrhoidectomy is to remove the offending varicosity with as little damage as possible to the patient. Of all the structures in the area, one stands out as the king. You can damage, deform, ruin, remove, abuse, amputate, maim, or mutilate every structure in and around the anus except one. That structure is the sphincter ani. There is not a muscle or structure in the body that has a more keenly developed sense of alertness and ability to accommodate itself to varying situations. It is like the goalie in hockey...always alert."

"They say man has succeeded where the animals fail because of the clever use of his hands yet, when compared to the hands, the sphincter ani is far superior. If you place into your cupped hands a mixture of fluid, solid, and gas and then, through an opening at the bottom, try to let only the gas escape, you will fail. Yet the sphincter ani can do it. The sphincter apparently can differentiate between solid, fluid, and gas. It apparently can tell whether its owner is alone or with someone, whether standing up or sitting down, whether its owner has his pants on or off. No other muscle in the body is such a protector of the dignity of man, yet so ready to come to his relief. A muscle like this is worth protecting."

Physiology: The rectum has little importance in digestion, and acts as a reservoir or collecting tube for undigested waste. The most important physiologic function of the rectum and anus is in the controlled act of defecation (i.e., continence).

Clinical Signs: Common clinical signs associated with diseases of the anus and rectum include: dyschezia, hematochezia, tenesmus, anal licking, ribbon-like stools, matting of anal hair, anal discharge, scooting, excessive flatulence and diarrhea. Patients that present with any of the above clinical signs should have a thorough physical examination with emphasis on the anorectal region, including a digital rectal examination.

Physical Examination: A complete physical examination should be performed in all patients with clinical signs specific for anorectal disease in order to rule-out systemic disorders that manifest themselves with anorectal abnormalities (i.e., pemphigus).

Specific examination of the anorectal region should include close visual examination of the perineum, circumanal area, and base of the tail, as well as careful digital rectal palpation. In many instances this may be all that is necessary to obtain a definitive diagnosis. If a more detailed examination is needed, the use of an anal dilator or proctoscope may be indicated.

These techniques require heavy sedation or general anesthesia to adequately perform. Epidural anesthesia has proven to be an effective anesthetic regime for examination of the anus and rectum. Excellent muscle relaxation allows easy anal sphincter dilation and visualization of the anal canal and rectal mucosa. The patient is placed in a perineal position for examination.

Sphincter muscle atonia or areflexia: This form of incontinence occurs when the peripheral nervous supply to the external anal sphincter muscle or the muscle itself has been partially or totally severed. The external anal sphincter muscle is made up of striated muscle fibers, and is partially responsible for the voluntary control of defecation.

Isolated injury of the pudendal nerve to the external anal sphincter is uncommon, but may occur from iatrogenic causes. Injury can occur during the following surgical procedures:

1. Perianal fistula repair-cryosurgery or excision

2. Perianal gland adenoma removal-cryosurgery or excision

3. Perineal hernia repair

4. Anal sacculectomy

5. Anoplasty procedures

6. Removal of malignant neoplasm

When this type of injury occurs unilaterally, the patient may still maintain enough sphincteric continence to be considered an appropriate house pet. With partial loss of anal sphincter tone, fine control of defecation is lost, but the patient still has the ability to sense the urge to defecate and can position properly. However, the fine control necessary to terminate a bowel movement without dropping a piece of stool is compromised. Also, when the patient is excited, startled, or barks loudly causing increased intra-abdominal pressure; a piece of stool may drop out of the rectum. The important thing to remember is that the patient retains the urge to defecate and can control, to some extent, bowel movements.

Anal Sacculitis: Anal sac impaction and abscessation is the most common anorectal disorder diagnosed by the small animal practitioner. Diagnosis is confirmed by clinical signs, visual and digital rectal examination. Relief of impaction by digitally expressing the anal sacs is easily performed during rectal examination. If an anal sax abscess is present, infusion of an antibiotic preparation may be sufficient to eliminate the infection. Systemic antimicrobial treatment may be required in resistant cases. If the anal sac abscess becomes a chronic recurrent problem, surgical excision of both anal sacs is the treatment of choice. Surgery should be delayed however until the immediate infection or abscess has been controlled medically as described above.

Surgical Techniques: There are a variety of techniques currently used to successfully remove anal sacs. One such technique includes using a pair of Metzenbaum scissors to cut into the anal sac through the duct. The sac is opened to expose the glistening greyish colored interior lining. Hemostats are used to grasp the full thickness of the anal sac wall, being careful to avoid the external anal sphincter muscle fibers. A number 15 BP scalpel blade is used to carefully scrape the gland from the underlying external anal sphincter muscle. The external anal sphincter m., subcutaneous tissue and skin are closed with a synthetic absorbable suture material in a simple interrupted pattern.

An alternate method is to incise over the anal sac, dissect through the subcutaneous tissue, locate the sac and excise it toward the duct.

Regardless of the procedure used, if the entire anal sac is removed and the caudal rectal nerve avoided the prognosis is excellent.

Mila Anal Sac Catheter Technique (the authors A novel approach for safely and completely removing anal sacs relies on the use of a 6 French balloon catheter with a 3cc bulb (Foley or Mila). The balloon catheter is placed into the anal sac through the anal sac orifice and its cuff inflated. Once introduced into the sac, the catheter bulb is inflated with 2-3 cc of air or saline. The bulb distends the anal sac making identification and palpation of the gland simple. The protruding catheter allows the surgeon, or the surgeon's assistant, to place gentle traction on the gland during dissection. A 360-degree skin incision is made around anal sac duct and the protruding catheter. Care is taken to leave at least 2mm of skin from the anal sac duct and the incision. Metzenbaum scissors (curved) are then used to dissect to the plane of tissue between the anal sac wall and external anal sphincter. Identification of the anal sac wall is made by identifying its grayish color in comparison to the deep red color of external anal sphincter muscle fibers that will be carefully dissected off of the anal sac wall. As the dissection progresses constant traction is placed on the Foley catheter to accentuate to sac. When performing the deep dissection of the sac wall care is taken to make certain the dissection does not go deep to the sac wall. This is the location of the caudal rectal nerve fibers. Dissection is continued until the sac is completely dissected free and removed from its surrounding tissue.

Closure consists of suturing together any cut fibers of the external anal sphincter muscle with 3-0 Maxon and the skin closed with 4-0 Biosyn using an intradermal technique. This is the authors preferred technique for anal sacculectomy.

This technique is illustrated on the Anal Sacculectomy video located in the GI Surgery I DVD. Check it out at www.videovet.org.

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ISFM - FELINE CARDIOLOGY

APPROACH TO CATS WITH SYNCOPE

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Syncope is defined as a "transient loss of consciousness and postural tone, associated with reduced cerebral delivery of oxygen and/or metabolites". The commonest cause for syncope is cardiovascular (arrhythmias, outflow tract obstruction, etc), but in rare cases it can be caused by transient hypo/hypertension or episodic hypoglycaemia.

Obtaining a detailed history, including the timeline of the episode and its association with recent events (exercise, respiratory signs, food intake, etc.), is absolutely crucial to choosing an appropriate diagnostic course for a cat presenting with syncope. If the clinician does not accurately identify the clinical signs – for example, if syncope is mistaken for a seizure episode – further testing may result in an incorrect diagnosis and treatment plan which influences patient well-being and further decision making over the coming months or years.

In dogs, syncope is generally rather easy to differentiate from a neurological or neuromuscular event. However, in cats, clinical signs perceived as neurological are often associated with a cardiogenic syncope event. In my experience, cats with bradyarrhythmias are commonly presented to the neurology referral service rather than a cardiologist in the first instance. History findings associated with syncope in dogs: association with exercise or exertion, falling over with flaccid muscles, unresponsive with eyes open, rapid recovery within 1-2 minutes. In cats, syncope is a more challenging event to identify so conclusively. It can occur at exertion or when lying still, is commonly associated with opisthotonus and limb rigidity, and occasionally is reported to feature vocalisation, salivation, facial motor activity or urination. These so-called "autonomic" signs are often suggestive to the clinician of a neurological event, and therefore heart disease could easily be overlooked. However, despite these clinical signs may overlap with neurological diseases, other features of syncope remain – namely the rapid recovery and return to normal activity. We will look at some video examples in the lectures.

One feline idiosyncrasy worth mentioning is that cats with bradyarrhythmias commonly present with facial motor activity alone, with no other signs of weakness or collapse. In this scenario, a diagnosis of a partial seizure is easily made. We would strongly recommend that any patient with "partial seizures" undergoes an ECG recording during an episode – whether this is lucky enough to be caught on a paper or digital ECG in-clinic, a Holter ECG or an implantable loop recorder.

Clinical sign	Body system	Example causes
Syncope	Cardiovascular	Arrhythmia, e.g. ventricular tachycardia
		Obstructed cardiac output, e.g. systolic anterior motion of the mitral valve
		Pulmonary hypertension, e.g. caused by patent ductus arteriosus
		Intra-cardiac right-to-left shunt, e.g. right to left VSD causing systemic hypoxaemia
	Metabolic	Transient hypoglycaemia
		Transient hypo/hypertension
Generalised weakness	Systemic disease	Anaemia, acute haemorrhage, sepsis, etc
	Cardiac	Persistent tachy- or brady-arrhythmia
	Neurological or neuromuscular disease	Myaesthenia gravis
Hind-limb weakness	Cardiac	Right-to-left PDA, persistent tachy- or brady-arrhythmia
	Neurologic or neuromuscular disease	Myaesthenia gravis
	Other	Aortic thromboembolism

Diagnostic Investigation of Syncope

1. History and video recording of episodes; VITAL

2. Physical examination: mucus membrane colour/CRT, arrhythmia, heart murmur, pulse quality/deficits, respiratory signs, behaviour and interactions in-clinic

3. Non-invasive blood pressure: Doppler systolic method

4. Blood tests: haematology, serum biochemical profile, cardiac biomarkers (NTproBNP and cardiac troponin I), endocrine testing (age dependent)

5. ECG

6. Echocardiography – especially if murmur or arrhythmia

7. 24h Holter ECG – next step to evaluate rhythm outside of the clinic, but poorly tolerated by most cats

8. Implantable loop recorder (Reveal device) – owner activates a subcutaneous ECG device to record during an episode of syncope
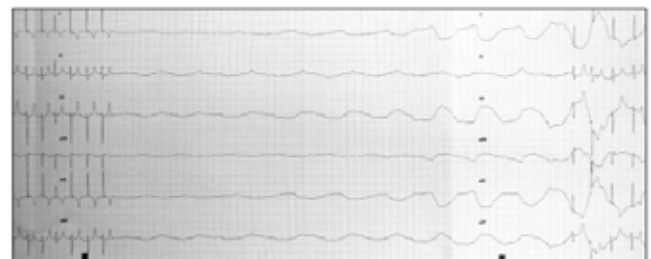

Figure 1: Paroxysmal asystole in a cat with episodic syncope

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ORAL RECONSTRUCTION

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Prior to any oral surgery or reconstruction familiarization with local nerve blocks is essential. Unlike some other areas of the body, good options exist for local analgesia. Local anaesthetics include:

- 0.5% bupivicine (up to 2mg/kg, onset within 30 minutes - effective for 6-10 hours)

- lidocaine 2% (up to 5mg/kg in dogs and 1mg/kg in cats, onset within 5 minutes - effective for <2hrs)

The **Infraorbital** nerve bock is applied at the infraorbital foramen or inside of the infraorbital canal and will block tissues rostral to the infraorbital foramen. The **inferior alveolar** nerve block is Injected around the mandibular foramen situated on the ventromedial aspect of the mandibular ramus. The area blocked includes the mandibular body, mandibular dentition and adjacent soft tissues. The **middle mental** nerve block is applied at the middle mental foramen ventral to the mesial root of the second premolar in the dog or halfway between the canine and third premolar in the cat. This will block the rostral mandibular body, the dentition, and adjacent soft tissues. The **major palatine** nerve block is applied to the palatal mucosa just rostral to the major palatine foramen and will block the palatine shelf of the maxilla. The **maxillary** nerve block is given just caudal to the last molar tooth as the maxillary nerve enters the maxillary foramen. This will bock the incisive bone, maxilla and palatine bone as well as maxillary dentition.

Lip reconstruction

Many smaller upper lip excisions can be performed in an inverted "V" shape and reconstructed directly. The upper lip in most dogs has significant mobility to allow both cosmetic and function reconstruction in this manner. Two to three-layer closure can be used depending on patient size. In cases where larger quantities of tissue require excision a direct uni- pedicle advancement flap may still facilitate closure. For more extensive defects or significant defects of the lower lip an angularis oris axial pattern flap or caudal cervical flap may be used to bring additional tissue into the reconstruction.

Soft tissue reconstruction following mandibulectomy

In most cases mandibulectomy is being performed to remove an oral malignancy. Gingival margins can be assessed visually however infiltration into adjacent bone and soft tissues is best conduted on CT. Margins of at least 1cm (more when feasible) should be achieved when performing excisions. Caudal mandibulectomies can usually be closed by direct intraoral mucosal apposition. In these instances, there is often no need to excise skin, allowing relatively straight forward if slightly awkward closure. Four tissue surfaces must be considered when excising central or cranial portions of the mandible; the oral sublingual mucosal, gingival, labial mucosa and skin. Excision should be performed to remove the desired bone and soft tissue margins. Additional bone often needs to be resected as transaction should be performed between dentition and still leave enough soft tissue to cover the bone ends (ie the bone excision must extend beyond the soft tissue excision). Edges of bone that are excised should be rounded off or filled where possible to avoid focal pressure on the overlying mucosa. Priority should be given to complete neoplastic excision and it is not uncommon that the cutaneous tissues of the skin must be sutured directly to the sublingual mucosa.

Although osseous reconstruction has been described with the use of BMP impregnated scaffolds, most large excisions will be performed without structural replacement. As such mandibular drift should be expected or managed at the time of the original surgery with elastic trainers. Subsequent application of elastic trainers following drift is less likely to be successful.

Maxillectomy

When preforming a maxillectomy the palatine and infraorbital arteries may need to be transected and ligated. A combination approach, both intraoral and through a lateral skin incision can be used to perform these procedures. Closure is typically accomplished by direct apposition of the labial mucosa to the palatine mucosa. A single pedicle advancement flap can be generated from the labial mucosa to allow improved closure. It is not uncommon to need to drill holes in the palate to allow placement of the sutures as the palatine mucosa can be friable. In situations were the palatine resection passes midline, complete closure with a labial flap can be difficult. A superficial cervical axial pattern flap or angluaris oris flap can be used.

Palatine Defects

Many patients with congenital palatine defects are euthanized at birth by the breeder. Those that are candidates for reconstruction need to reach at least 3-7 months of age before surgery. Premature correction can result in tearing of tissues or damage to the periosteum which can hamper further development. Waiting until adult dentition has erupted will also allow extraction, freeing more soft tissues to allow closure. Waiting too long can result in larger defects and often the pet will require a tube feeding until definitive repair which can place a burden on the owner.

The defects are a result of incomplete fusion of the maxillofacial structures;

- Cleft lip, rostral hard palate
- Midline of hard and soft palate

- Lateral area of the soft palate
- Hypoplastic soft palate

Attempts should be made to have the repair supported by underlying bone. This is where a CT is of value in pre-operative assessment. The oral fistula may appear relatively small, however the defect in the supportive bone shelf can be much larger. Flaps should be 1.5x larger than the required defect, electrosurgical equipment or lasers should be avoided, and closure should take place with no significant tension.

Rostral defects

Congenital defects of the primary palate (rostral to the incisive papilla) require flaps of oral and nasal soft tissue. Advancement, rotation, transposition or overlapping flaps are followed by reconstruction of the cutaneous tissues.

Overlapping flap technique for hard palate repair.

An incision is made in the mucoperiosteum 1-2mm away from the dentition on one side of the defect laterally. The second incision is made in the mucoperiosteum at the defect. Elevation of the tissues is performed bilaterally while attempting to preserve the major palatine artery. The first flap is then rotated medially across midline and under the second flap before being secured.

Medially positioned flap technique for the hard palate

Bipedicle flaps are directly elevated from each side of the oral mucosa and centrally apposed. This is used only for minor reconstructions as there is no underlying bone to support the closure. In cases of trauma the bone apposition can sometimes be improved by direct digital pressure on each side of the maxilla or twisting orthopaedic wire between the maxillary canines (and then covering in a self-curing composite).

Medially positioned flap technique for soft palate repair

Incisions should be made along the medial margins of the defect to the level of the caudal end of the tonsils. By cutting the margins of the defect two separate sets of flaps are created, one in the nasopharynx and one in the oropharynx. Separate clsoure of the two flaps creates a double layered closure.

Labial based mucoperiosteal flap for repair of oronasal fistula

Either a single (labial based only) or double (labial based over a reflected palatine mucosal flap) repair can be used to close these defects.

Elongated soft palate

Elongated soft palates are typically resected with laser, blade or vessel sealing device to the level of the caudal tonsils.

WSAVA GLOBAL NUTRITION

WATER, WATER EVERYWHERE: NUTRITION AND HYDRATION IN THE MANAGEMENT OF FELINE CKD

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Introduction

Chronic kidney disease (CKD) is the most common renal disease in the cat. The prevalence of CKD seems to be increasing over time; estimates are that it affects about one-third of all cats over 15 years of age. It is an important cause of mortality, especially in older cats. CKD is typically a progressive disease and can be accompanied by a wide range of clinical and pathological changes. However, the clinical presentation is variable from patient to patient.

The International Renal Interest Society (IRIS) has published guidelines for clinical staging and treatment targets for both canine and feline kidney disease (http://www.iris-kidney.com). As well, the International Society of Feline Medicine published **Consensus Guidelines on the Diagnosis and Management of Feline Chronic Kidney Disease** in 2016 (http://jfm.sagepub.com/site/Guidelines/Guidelines.xhtml). The reader is referred to these documents for a complete discussion of CKD diagnosis and management.

The goals of CKD therapy are to:

- Minimize clinical signs of uremia
- Minimize disturbances of electrolytes, vitamins, and minerals
- Provide adequate nutrition and hydration
- Improve quality of life (QOL), especially in International Renal Interest Society (IRIS) stages 3 and 4
- Modify disease progression

Wherever possible, potential therapies should be evaluated considering a specific treatment goal and based on available evidence. In some patients, multiple treatments may be indicated, but administration of multiple therapies must be balanced with QOL; prioritizing therapies most likely to benefit each patient is important.

This session will focus on keeping CKD cats well hydrated and nourished as it is critical for their survival and well-being.

Importance of dietary management

The use of diets specifically formulated for CKD results in improved survival and quality of life[1].

The key nutritional factors in CKD diets for cats are as

follows[2]: phosphorus restriction, high quality protein moderation, sodium control, B vitamin fortification, and alkalization (the only feline diets to promote so). The potassium content is usually higher than typical maintenance diets, although it varies from brand to brand. Some of them also include long chain omega 3 fatty acids EPA and DHA.

Energy

Cats with CKD have typically waxing and waning appetite, which is why CKD diets are and energy dense (high fat, low fiber). These diets are high in energy density (thus, low in fiber and high in fat) to promote adequate energy intake even in the face of occasional inappetence. Both dry and canned use this strategy, but canned foods, by virtue of their high moisture content, are less energy dense than dry foods.

Phosphorus

The most important modification in CKD diets is phosphorus restriction (below 1 g/1000 Kcal, in general), which is indicated to address renal secondary hyperparathyroidism, and this strategy is believed to slow down progression of CKD. The IRIS society (www.iris-kidney.com) recommends the use of low phosphorus diets initially (stage II onwards), but recommends the addition of phosphate binders at later stages, where dietary restriction is not enough.

Sodium

These diets have moderate sodium concentrations as a precaution due to the likelihood of hypertension in these patients, although the effect of dietary sodium on blood pressure is unclear. Sodium is never truly restricted, because a very low sodium diet can stimulate the renin angiotensin aldosterone axis and result in hypertension[3]. Most diets maintenance diets provide 1 g/1000 Kcal, and renal diets range from 0.5 to 1 g/1000 Kcal.

Potassium

Potassium content varies amongst feline CKD diets (from 1.5 to 3.5 g/1000 Kcal, approximately). In hyperkalemic patients (e.g. some patients treated with ACE inhibitors), choosing the lowest K diet available is indicated. For hypokalemia, high K diets can be chosen (or, alternatively, potassium can be supplemented orally).

Protein

Protein should never be restricted. Providing all nitrogen and essential amino acids is essential and protein deficiency will result in lean mass loss and worse prognostic. Diets for CKD are always above the requirement, but they tend to be lower than typical maintenance diet to minimize nitrogen waste product formation and accumulation, which contribute to uremia. Dietary protein should be of a high biological value. Protein moderation then helps reduce clinical signs but it

is not believed to affect progression (except potentially in proteinuric patients)

The NRC[4] minimum protein requirement is around 16% protein calories, while AAFCO recommends maintenance feline diets provide at least 22%. Thus, protein intake will be adequate provided that the patient meets its energy needs. If the cat does not eat enough calories, muscle mobilization will happen and both body and dietary protein will be used to obtain energy, resulting in protein:calorie malnutrition.

Feline CKD diets range from 22 to 34%, and all of them provide all amino acid requirements, thus there is a wide range to choose from for each specific case and adjusted to the stage of disease.

B vitamins & acid base balance

B vitamin losses can be increased due to polyuria, and inappetence can result in a decreased daily intake.

Kidneys are very important for acid base balance, and cats with CKD are prone to metabolic acidosis, which is why feline CKD diet are alkalinizing.

Omega 3 fatty acids

EPA and DHA have shown positive effects on experimental canine CKD, and one retrospective study in cats suggested that diets rich in these fatty acids could result in longer survival[5].

Managing hydration

Cats with CKD are predisposed to dehydration, especially in IRIS stages 3 and 4. Studies confirming the clinical impact of maintaining hydration are lacking, but it is considered a critical part of management. Maintaining hydration may help maintain QOL, address electrolyte and acid-base disturbances, and preserve renal blood flow by

preventing dehydration (and potentially affecting disease progression). Unstable or decompensated cats with CKD may require hospitalization and intravenous (IV) fluid therapy, along with management of electrolyte and acid-base disturbances. Owners should also be educated about long-term management of hydration, including increasing voluntary water intake and home subcutaneous (SC) fluid therapy (75-150 mL every 1-3 days).[6] Fluid choices include balanced electrolyte solutions or 0.45% saline. Potassium chloride can be added if needed to treat hypokalemia.

Managing nausea and inappetence

Cats with CKD may have nausea, vomiting, and inappetence because of uremic toxins affecting the central chemoreceptor trigger zone. Owners identify poor appetite as an important QOL concern; it could also result in protein and calorie malnutrition. A reduction in appetite should be actively investigated and treated;

nausea should always be considered as a possible cause even if the cat is not vomiting. Maropitant (1 mg/kg, PO, every 24 hours) has been shown to reduce vomiting[7] and mirtazapine (1.88 mg/cat, PO, every 48 hours) has been shown to reduce vomiting, increase appetite and promote weight gain.[8] Other effective anti-emetic drugs include ondansetron (0.5-1 mg/kg, IV, SC or PO, every 8 hours)[9] and dolasetron (0.6-1 mg/kg, IV, SC or PO, every 24 hours). While hyperacidity may occur in some cats with CKD, gastric ulceration is typically not found. Instead, gastric mineralization and fibrosis are the most significant lesions.[10] If therapy for hyperacidity is considered, omeprazole (1 mg/kg, PO, every 12 hours) is superior to famotidine.[11,12] Cats that are not achieving adequate food intake with drug therapy may benefit from placement of an esophagostomy feeding tube to maintain hydration, dminister drugs and provide nutrition.

Nutritional plan

A complete nutritional evaluation (http://www.wsava.org/guidelines/global-nutrition-guidelines) should be carried out before making recommendations. These recommendations should include:

1. When to start dietary management: The recommendation is to change to a CKD diet in stages II to IV. Patients with stage I only require diet change if they present proteinuria, where they will benefit from a moderate protein diet. Earlier change will result in better acceptance.

2. What to feed

There are several commercial feline CKD diets, choice will depend on stage of disease, price, availability, palatability, and nutrient characteristics.

3. How much to feed

The amount of food should be enough to maintain a stable body weight and ideal body condition score (BCS). Label instructions are a good start but they will need twice a month adjustments. Patients with low BCS should be fed 20% more than label instructions/formulas.

4. How to feed

Multiple small feedings or ad libitum feeding is indicated in thin patients. Overweight cats should be fed portion-controlled amounts to at least prevent further weight gain. Feeding tubes can be used in patients that are not eating enough to provide an adequate diet (plus medications and water).

Summary

For each CKD patient, the IRIS stage should be established and an individual treatment plan should be developed, considering what is most appropriate for each patient and owner. The options should be prioritized based on the cat's medical needs and the owner's preferences and abilities. The plan should be reviewed with the owners and their commitment confirmed. A reassessment and monitoring schedule should be established to assess the patient's response, make any necessary changes to the treatment plan, ensure the owner understands the treatments and uncover compliance issues.

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NEPHROLOGY

APPLICATION OF THE IRIS GUIDELINES IN THE MANAGEMENT OF CKD

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Introduction

The prevalence of chronic kidney disease (CKD) among dogs and cats is unknown. Estimations range between 0.5-7% in dogs and up to 30% in geriatric cats. Chronic kidney disease is irreversible and progressive in nature even if the inciting cause can be eliminated. Due to the reduced number of nephrons, each remaining nephron is undergoing hypertrophy (and is becoming a "super nephron"), but this compensatory mechanism eventually leads to additional nephron loss.

The current terminology of the disease, as suggested by the International Renal Interest Society (IRIS), is "chronic kidney disease" and it is classified into four stages (see below) to guide therapeutic guidelines and predict the prognosis. Classification into the stage is determined by plasma creatinine concentration, but is done only **when the animal is in steady state**. The disease is further classified based on renal proteinuria and blood pressure. Both have been incorporated in the disease staging because there is solid evidence to suggest that proteinuria and hypertension influence the survival of dogs and cats with CKD. The degree of **renal** proteinuria is determined by the urine protein to creatinine (UPC) ratio. Blood pressure should be determined using appropriate technique and equipment, as stress and anxiety might dramatically influence the blood pressure ("white coat effect").

Diagnosis

The diagnosis of CKD is based on clinical signs, which are often non-specific, and is confirmed by laboratory tests. Serum creatinine, symmetric dimethylarginine (SDMA), urea concentration, urine concentration ability, presence of proteinuria, imaging techniques, blood pressure, and kidney biopsy can all be used to diagnose and classify the disease. Early diagnosis is critical, as nephron loss is an irreversible process, thus early intervention slows down the disease progression, and delays onset of clinical signs.

Management of chronic kidney disease

Treatment goals are to slow down the progression rate and to control the clinical signs and clinicopathologic abnormalities. These include polyuria and polydipsia, anorexia, vomiting, diarrhea, weight loss, anemia, bleeding, protein loss, hypertension, acid base and electrolytes imbalances. Management should be tailored to the specific patient according to its IRIS Stage. While most of the clinical manifestations are expected when the animal is in CKD Stage III or above, some of the complications (e.g., hypertension, proteinuria) might be present even in Stage I CKD.

Nutrition

Nutritional management is a cornerstone in the management of CKD. Evidence suggests that consuming a therapeutic kidney diet is associated with lower number of uremic crises and decreased mortality rate compared to patients consuming maintenance diets. Yet, anorexia and weight loss are major factors that influence owners of dogs and cats with CKD to elect euthanasia, and thus many owners and veterinarians elect to feed free choice diets, despite the fact that the latter facilitate uremic crisis and progression. Thus before offering a therapeutic kidney diet, clinical signs (especially gastrointestinal signs) must be controlled. A variety of commercial diets should be offered, and any transition to a kidney diet must be done gradually and only when clinical signs are controlled. Any attempt to transition a patient to a kidney diet when the animal is presented to the clinic for the first time, before clinical signs are controlled, is doomed for failure.

Hypertension

Hypertension is common in dogs and cats with CKD and is associated with rapid progression of the disease. When systemic hypertension is documented or suspected, the risk for end organ damage (i.e., kidney, heart, eye, brain) needs to be evaluated. Typically, hypertension is not considered an emergency unless it is extreme or when evidence for end organ damage is apparent. The most commonly used drugs to control hypertension are ACE inhibitors (ACEi) (e.g., enalapril, benazepril), angiotensin receptor blockade (ARBs) (e.g., telmisartan) and calcium channel blockers (e.g., amlodipine). Caution should be used not to administer ACEi to dehydrated animals, as glomerular filtration rate may drop precipitously if these drugs are introduced before the patient is adequately hydrated or in uremic crisis. In dogs, the IRIS recommendation is to combine ACEi and calcium channel blockers, but in cats calcium channel blockers are usually sufficient to control hypertension. The target blood pressure is systolic blood pressure <160 mmHg.

Proteinuria

When present, the initial goal is to identify the origin of the proteinuria and its cause, as anti-proteinuric therapies should be administered only to animals with renal proteinuria. Common causes (e.g., infectious, inflammatory, neoplastic processes) should be looked for and eliminated if possible. If the initial cause can neither be eliminated nor can be identified, the treatment is symptomatic. Kidney biopsy should be considered as a diagnostic aid to identify the underlying disease and

presence of immune complexes. The treatment goals are to decrease the magnitude of proteinuria and to prevent complications that may be associated with it (e.g., edema, decreased antithrombin concentration leading to hypercoagulability). The treatment include dietary modification, ACEi/ARBs, and immunosuppression, if the process is immune mediated. ACEi and ARBs decrease proteinuria by decreasing the resistance of the afferent glomerular arteriole, and consequently decrease the intraglomerular pressure. Thus, serum creatinine concentration should be monitored ~5 days following initiation of treatment to identify any decrease in GFR in a timely manner. Low dose aspirin has also been recommended if albumin concertation is <2.0 g/dL. When histology supports presence of immune complexes, immunosuppression should be considered.

Hyperphosphatemia and Secondary hyperparathyroidism

Hyperphosphatemia and secondary hyperparathyroidism are inevitable consequences of CKD. Hyperphosphatemia and secondary hyperparathyroidism are controlled using phosphorus restricted diets and phosphorus binders. These drugs bind the phosphorus in the food, thus should not be administered without it. The most commonly used phosphate binders are aluminum based or calcium based. In Stage I-III, phosphorous concentration needs to be maintained at the range of 2.7-4.6 mg/dL, and at stage III and IV <5.0 mg/dL and <6.0 mg/dL, respectively. Evidence suggests that judicious use of calcitriol (1.5 to 3.5 ng/kg), prolongs survival in dogs in Stage III when phosphate is controlled and ionized calcium and PTH are monitored.

Anemia

Anemia is evident mostly in animals in Stage III CKD. There are many potential causes for anemia in patients with CKD, including decreased erythropoietin production, bleeding (mostly to the gastrointestinal tract) and bone marrow dysfunction. The timing for treatment initiation is determined by the severity of the anemia, the degree of clinical signs associated with it, and when the clinician is convinced that its origin is erythropoietin deficiency. In most animals anemia is treated when hematocrit is <20%. The two common therapies include erythropoietin and blood transfusions. The latter are used mainly in crisis or when a rapid response in needed. The main complication of erythropoietin treatment is antibody production.

Metabolic acidosis

Metabolic acidosis is a common complication of CKD and is to be expected in late Stage III and Stage IV CKD. The diagnosis of metabolic acidosis is based on total venous CO_2 or bicarbonate concentration. Treatment of metabolic acidosis is to be initiated when bicarbonate is < 18 mmol/L and includes administration of sodium bicarbonate or potassium citrate.

Hydration

The main goal of subcutaneous fluid administration is to prevent dehydration. It is likely that patients that maintain hydration status by drinking enough water to compensate for their fluid losses do not benefit from subcutaneous fluids. Moreover, fluid administration should not be regarded is risk free. Potential side effects of subcutaneous fluids include hypernatremia and worsening pre-existing hypertension, negative effect on the owner-pet interaction and decrease the patient quality of life. In animals that tend to get dehydrated (especially cats), subcutaneous fluid administration is beneficial and thus might be used on a regular basis. As the disease progresses, this treatment becomes more and more common both in dog and cats.

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http://www.iris-kidney.com

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SVA EXOTICS

DEALING WITH THE PEDIATRIC BIRD EMERGENCY

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Abstract

Psittacine chicks have little in the way of compensatory mechanisms to cope with disease and consequently frequently present as acute emergencies. This presentation details the major problems facing a chick with an emergent condition, and their recognition and treatment by the clinician.

Introduction

Most companion birds (e.g. parrots and passerine birds) are altricial (i.e. when hatched they are blind, deaf and not feathered, and therefore totally dependent on their parents or rearer. They go through a rapid growth phase, during which their size and weight increases to a level above that of their adult weight, before falling and then plateauing at their fledging weight. During this time their eyes and ears open, their feathers grow, the bones and internal organs mature, and their social skills and learned behaviours are shaped. This is obviously a period of rapid change, where small changes can have a big impact on their health and development.

Added to these rapidly-developing changes is the lack of awareness by the owner that a problem is present - and then a delay in seeking veterinary assistance - meaning that most chicks presented for health problems are often severely decompensated and are emergent cases.

What are the challenges of a paediatric case?

There are unique challenges the clinician faces when dealing with a paediatric case. These include:

a. Their small body size

1. A large surface area to body mass ratio, sensitising them to temperature extremes (especially hypothermia)

2. Difficulty in accessing blood vessels for the administration of fluids and medications and collecting blood samples

3. Small muscle mass, making intramuscular injections difficult to administer without causing significant bruising pain, and discomfort

b. The lack of body fat makes:

1. Body heat retention difficult, contributing to hypothermia

2. Coelomic radiography difficult to interpret due to the loss of contrast.

c. Immature physiology:

1. The ability to invoke compensatory physiologic responses (e.g. vasoconstriction and vasodilation) is limited

2. Lack of white fat (for insulation) makes them reliant on brown fat (to a small extent) and metabolism to maintain their body temperature. This requires the frequent ingestion of a high energy, easily digested food

3. The kidneys have poorly developed concentration and filtering capacity

4. The digestive tract has a rapid transit time and is easily 'stressed', leading to ileus

5. The respiratory tract is immature, and the air sacs are compressed by the intestinal tract, giving a low respiratory reservoir

d. Haematologic and biochemical differences from adults can make the use of diagnostic testing difficult. Compared to an adult of the same species, chicks generally have:

1. Lower PCV and higher WCC

2. Lower total protein and uric acid

3. Higher CK

Evaluating the sick chick on presentation

On initial presentation of the chick, a rapid but thorough evaluation needs to occur. This evaluation includes a history, physical examination, and finally diagnostic testing (performed after the chick is stabilised).

The history of the patient needs to include the species and age of the chick, whether it was artificially or naturally incubated, whether it is been parent-reared or hand-reared, and what the parents were been fed. If it is been hand-reared, the following information must be ascertained: from what age did the hand-rearing start; what is been fed; how is it been mixed; how often and how much is the chick fed; when it was last fed; and at what temperature the chick is been maintained. It is also important to know what the exposure of the chick to other birds/chicks is, and whether there have been any problem with them. It is useful if there are records accompanying the chick, but these are often not available.

The physical examination needs to include its body

weight, its body condition (best evaluated by examination of the toes and elbows), its posture and conformation, its body colour and temperature, its hydration status, whether its crop has food in it, and an evaluation of its droppings.

If possible, a few drops of blood can be collected for a PCV, blood smear, TPP, and blood glucose.

This evaluation, while not exhaustive, can performed rapidly and assessment made as to the chick's status using the four H's.

The four H's

Regardless of the underlying aetiology, the majority of problems with sick chicks can be associated with the "four H's":

1. Hypovolaemia

2. Hypoglycaemia

3. Hypothermia

4. Hypoxaemia

Other problems, such as beak abnormalities and angular limb deformities, rarely present as emergent conditions and will not be discussed further in this paper.

Hypovolaemia

Severe dehydration is common in chicks, usually associated with GI dysfunction (vomiting, diarrhoea, and ileus) or decreased intake (poor husbandry, refusal to eat). Respiratory loss due to panting when heat stressed can also lead to dehydration. Dehydration and hypovolaemia lead to decreased tissue perfusion and subsequent organ damage and even failure. As chicks appear to lack the ability to compensate for mild hypovolaemia to increase tissue perfusion (increased heart rate and contractility, and increased vascular tone), they are extremely sensitive to the effects of dehydration.

Clinical signs include:

1. Thickened mucoid saliva

2. Wrinkling of the skin

3. Decreased venous return (assessed by 'blanching' the basilic vein)

4. Decreased urine output and thickened urates

5. Crop stasis

If any uncertainty is present, it is usually safe to assume the chick is dehydrated and act accordingly.

Treatment requires the administration of warmed isotonic fluids. These can be given IV, IO or SC, but if the SC route is chosen the chick should be normothermic, as peripheral vasoconstriction will slow fluid absorption. IO catheters can be placed in the tibiotarsus or ulna, while IV catheters can be placed in the jugular, basilic or medial metatarsal veins. An initial fluid bolus of 3% - 4% of the chick's body weight can be given over 15 minutes, and repeated based on the chick's response. Ongoing fluid administration at 3mls/kg/hour can then be initiated. Care must be taken to avoid fluid overload (often seen as a dyspnoea associated with non-cardiogenic pulmonary oedema). Once the chick is well hydrated the oral administration of fluids can be expected to maintain a suitable fluid balance.

Hypoglycaemia

Hypoglycaemia often arises due to either poor husbandry (inappropriate diet, infrequent feeding, diluted food, or insufficient volume of food) or severe systemic illness (particularly sepsis or those conditions leading to GI ileus or other GI dysfunction). When low dietary intake is combined with an immature gluconeogenic response and low body fat, the result can be severe hypoglycaemia. This in turn leads to CNS and cardiac disturbances (weakness, seizures, coma and death). In the early stages of hypoglycaemia, chicks may be constantly hungry, exhibited as excessive begging behaviour.

Chicks with severe hypoglycaemia should be treated with an IV/IO bolus of dextrose (250-500mg/kg (50% dextrose, 0.5-1ml/kg diluted 1:4) administered over 5 minutes. Repeat boluses can be given, based on response to treatment, and then once normoglycaemic the chick's IV/IO fluids can be supplemented with 5% dextrose until the chick is eating.

Less severe hypoglycaemia may be effectively dealt with by an early return to feeding (or by feeding a more appropriate diet).

Hypothermia

Hypothermia in chicks arises because of the effect of the following factors:

1. Low reserves of white (insulating) fat

2. Large surface area to body mass ratio

3. Lack of feathers to insulate the body

4. Reduced ability to vasoconstrict or shiver

5. Reduced activity (chicks are usually sleeping or eating)

When these factors are combined with inappropriate environmental temperatures or reduced food intake (decreased metabolic energy), the result can be profound hypothermia.

Hypothermic chicks are lethargic and poorly responsive to stimulus. Their limbs and bodies are cool to the touch.

Peripheral vasoconstriction can be seen as a pallor to the skin. Left untreated cardiovascular function will be compromised, resulting in organ dysfunction and finally cardiac arrest.

Hypothermic chicks should be warmed before fluid resuscitation, but care should be taken to avoid peripheral vasodilation and possible hypotension. Warming can be achieved with warmed air or heat lamps but the chick must have the ability to move away from the heat source once it is normothermic, to avoid hyperthermia.

Hypoxaemia

Hypoxaemia is common in chicks and can be associated with anaemia, the aspiration of hand rearing formula, infectious respiratory diseases (e.g. bacterial or fungal infections), or compression of the air sacs by distended loops of intestinal tract. Hypoxic chicks will have an increased respiratory rate and effort (mouth breathing, increased sternal lift and tail bobbing). Cyanosis can be seen, but is usually difficult to appreciate. Pulse oximetry may be of benefit but – because of calibration difficulties with nucleated erythrocytes - trends, rather than absolute numbers, should be monitored.

Oxygen supplementation (via an anaesthetic induction chamber, flow-by oxygen or an intranasal oxygen line) should be administered when hypoxaemia is diagnosed or suspected Care must be taken to prevent oxidative tissue damage associated with 100% oxygen administered for prolonged periods of time.

Where to from here?

Once the sick chick has been stabilised the clinician can then move on to determining the underlying reason for the problems that the chick was presented. The most common causes of disease in chicks are infectious diseases (viral e.g. PBFD, APV; fungal e.g. Candida, Aspergillus; and bacterial), malnutrition (stunting syndrome, metabolic bone disease), scissor beak, and crop burns.

But the diagnosis and treatment of these cases can only be performed once the chick is stabilised. Careful evaluation and examination are paramount in diagnosing and treating paediatric problems.

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WSV18-0240

DIAGNOSTIC IMAGING AND GASTROENTEROLOGY
(SIMULTANEOUS TRANSLATION INTO MANDARI

CANINE ESOPHAGITIS

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Etiology:

The esophageal mucosa can be damaged by gastric juices and bile in gastroesophageal reflux disease (GERD). This disease is a common complication of lower esophageal sphincter relaxation during general anesthesia, but often remains subclinical. Esophagitis may also occur in dogs with frequent and severe vomiting. In addition, esophageal foreign bodies and swallowing of caustic substances may also cause esophagitis. In endemic area, **Spirocerca lupi** infections may be at the origin of esophageal diseases. The adult worms generally inhabit the esophageal submucosa and adventitia, and lead to the formation of granulomatous nodules in the caudal esophageal wall.

Strictures share the same etiology as severe esophagitis (foreign bodies, gastro-esophageal reflux, ingestion of caustic substances). They occur when the submucosa and muscularis layers of the esophageal wall are affected, and lead to excessive scarring causing partial or near total obstruction of the esophageal lumen.

Clinical presentation:

Esophagitis: regurgitation, dysphagia, swallowing attempts "on empty", halitosis, odynophagia (pain on swallowing as demonstrated by hesitation in swallowing, tension of neck muscles), excessive salivation, anorexia/inappetence, tachypnea, dyspnea, cough, exercise intolerance, fever (in case of aspiration pneumonia)

Strictures: history often reveals previous esophageal disease (for instance esophageal foreign body) or general anesthesia prior to the development of signs. Same clinical signs as for esophagitis. Regurgitation may only occur with a specific food consistency (e.g. dry food but not a pureed diet).

Diagnosis:

A tentative clinical diagnosis of esophagitis is made on the basis of clinical signs and history. Survey radiography is usually unremarkable. The esophagus may be dilated with gas or fluid but this is a non-specific finding. Confirmation of diagnosis and assessment of the extent and severity of lesions requires endoscopic examination. For **Spirocerca lupi** infections, diagnosis relies on positive fecal floatation and ultimately endoscopic confirmation of esophageal wall nodules (early) or masses (late) in dogs with compatible signs.

A diagnosis of esophageal stricture in a patient with compatible clinical signs is confirmed either by the use of endoscopy or barium contrast radiography. It is essential to differentiate stricture of the esophageal lumen from external compression.

Management:

Symptomatic treatment of esophagitis is focused on the protection of the mucosa against further damage, and facilitation of mucosal healing. Fasting for 24h is recommended, if feasible without compromising the dog's condition. A diet low in fat is recommended because high fat diets may be associated with increased episodes of gastro-esophageal reflux. Sucralfate suspension is used to promote mucosal healing (1 g every 8 h for large dogs, 0.5 g at the same frequency for smaller dogs. Inhibitors of gastric acid secretion such as omeprazole (1-2 mg/kg PO every 12 h) and a prokinetic agent that accelerates gastric emptying (metoclopramide 0.5 mg/kg PO, SC q8h, or constant rate infusion of 1-2 mg/kg over 24h) or promotes gastric emptying and increases the distal esophageal sphincter tone (cisapride 0.5-1 mg/kg PO q8 h) should be administered preferably 30-60 min. before meals.

Treatment of spirocercosis consists in administering high doses of the avermectin drug doramectin (off label). One author recommends a daily dose for 6 weeks followed by a recheck endoscopy.

For strictures, treatment consists of dilating the stricture(s) using balloons of different diameters under endoscopic control, or "bougies" of different sizes under fluoroscopic or endoscopic control. Multiple sessions at intervals of 1-2 weeks are often necessary. Injection of triamcinolone in the esophageal submucosa at the level of the stricture can help reduce the risk of relapse. After stricture dilation, treatment as described for esophagitis is recommended.

In severe cases of esophagitis, placement of a gastrostomy tube via endoscopy or surgery is beneficial to provide enteral nutrition and prevent complications associated with malnutrition while resting the esophageal mucosa.

Prognosis:

Esophagitis of mild and moderate severity generally has a good prognosis. Severe inflammatory mucosal lesions may lead to esophageal stricture development. The prognosis for strictures is guarded. In the best-case scenario, 2-3 procedures for stricture dilation will be required, and the clinical signs will be under control, at least with soft food. In the worst-case scenario, repeated attempts at dilating the stricture will remain unsuccessful.

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MAXIMIZING THE RETURN ON TAKING A BIOPSY

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INTRODUCTION

Taking a biopsy sample of diseased tissue for histopathological assessment is one of the most commonly performed diagnostic procedures in small animal practice. Considerable effort and expense is often expended in obtaining such biopsy specimens, particularly if the procedure involves incision or excision of a lesion under general anaesthesia. For the benefit of the animal and the client, it therefore makes sense to do everything possible to maximize return from the submission of biopsy specimens. This short presentation reviews the key stages in sample submission from the perspective of the diagnostic histopathologist.

WHAT SAMPLE?

The clinician must make the fundamental decision as to the optimum sample to collect from a lesion arising within the individual patient – and this may differ between patients for a variety of reasons (e.g. anaesthetic risk, client budget, desired speed of result). In general, pathologists prefer the largest sample possible – so an excisional sample is preferable to an incisional biopsy, trucut core biopsy or fine needle aspiration (FNA). FNA of a skin mass is a sensible rapid pre-operative procedure when performed in-house, but where cytological samples are sent to a diagnostic laboratory from a mass that is likely to be excised surgically no matter what the outcome, it may make sense to simply undertake the surgical procedure and submit the excised specimen.

FIXING THE SAMPLE

Tissue samples for histopathological assessment should be fixed in 10% neutral buffered formalin. A tenfold excess of formalin to sample volume is required for adequate fixation. Formalin penetrates into tissue at the rate of 5 mm per 24 hours. Where possible, an entire specimen should be submitted (e.g. if splenectomy is undertaken the entire spleen rather than a selected portion), but where size precludes sending the entire sample (e.g. expense of postal costs or safety of postal submission) the most representative area of the lesion to include a junction with normal tissue, should be sent. Dermatologists often deliberately submit a biopsy of normal skin in parallel with lesional tissue. Sending an entire sample will allow the assessment of multiple margins. If possible, you should avoid incising samples or dividing them into multiple portions. It is better that

the pathologist does this and allows further fixation time in the laboratory; however, bisecting a relatively large specimen will enable more effective fixation.

Samples should always be submitted in purpose-designed, wide-mouthed biopsy pots. A fresh sample may well readily be squeezed through the neck of a pill bottle, but once fixed, often the only way to remove the sample is by smashing the container. Portions of tissue that are likely to fold or curl during fixation (e.g. samples of intestine) may be pinned to board so that they fix in an optimum position. Very small endoscopic biopsy samples may benefit from being fixed by being layered within purpose-designed 'sponges' in histocassettes or in purpose-designed mesh bags. This will avoid unnecessary trauma to the delicate samples by being shaken within the formalin pot.

Where margins are to be assessed (i.e. for tumours) it is important that you are able to indicate to the pathologist the orientation of the excisional sample within the animal so that specific margins can be identified. The use of photographs or sutures placed on the specimen can achieve this; alternatively the use of purpose-designed inks can mark particular margins. These surgical inks are resistant to histological processing and should appear on the final stained section.

Postal samples should be packaged according to local regulations – generally surrounded by layers of absorbent material, within a sealed plastic bag and an appropriate outer container. Fixed tissues are generally regarded as being 'Category B' biological samples and the outer envelope should bear the designation UN3373.

SENDING THE CLINICAL HISTORY

One of the greatest frustrations to the diagnostic pathologist is receiving no or minimal clinical history or sometimes not even the signalment data of the patient. Most diagnostic pathology laboratories provide submission forms that detail the required information. It goes without saying that you might provide the most detailed clinical history, but unless the pathologist can read the handwriting this effort is wasted! Quite simply, the more information you provide, the more helpful the pathologist can be. It is wonderful when submission forms are accompanied by digital photographs of the patient – showing the lesions **in situ**. Dermatologists seem particularly adept at using this technology. The clinician should also be aware that some pathologists and some laboratories have particular expertise in dealing with specific sample types (e.g. endoscopic biopsies of gastrointestinal mucosa, skin or bone marrow biopsies), and it may be that on occasion a specialist laboratory might be used for a particular case.

BE PATIENT

Most diagnostic histopathology laboratories offer a 24 hour turn-around for routine biopsy submissions, but the clinician should appreciate that non-standard samples require more complex processing and it will therefore take longer to generate a result. A large (e.g. orange-sized) or very soft tissue sample may require additional fixation in the laboratory before processing or a 'long process' over several days rather than overnight. Tissues including areas of bony metaplasia or samples including bone (e.g. digit removal, bone tumours) will require prolonged decalcification before the tissue is soft enough to process and section. Decalcification of bone may take many weeks.

UNDERSTAND THE LANGUAGE OF THE PATHOLOGIST

Once the pathology report is sent to you, do take the time to read and understand the complete report. Many clinicians will read only the 'bottom line' (i.e. the diagnosis) or the 'comments' section of a report and skip the actual descriptive report. Much information is conveyed in that description, particularly assessment of marginal infiltration or metastatic activity. Pathologists do use a very specific language to convey degrees of certainty (and this has been studied and published) and you should understand the way that your pathologist uses terms such as 'consistent with', 'not inconsistent with', 'probable', 'likely' and others.

COMMUNICATE WITH YOUR PATHOLOGIST

This is the most important of all. If anything is unclear or the pathological report does not match your clinical expectations then do not be afraid to telephone your pathologist to discuss the case. This exercise is of mutual benefit. The pathologist should be able to advise on whether further sectioning of the gross specimen is justified, or whether special histochemical, immunohistochemical or molecular studies might be performed. For some infectious agents it is now possible to take samples from the wax-embedded tissue for PCR diagnosis and molecular tests for cancer characterization are now routinely available. Many pathology laboratories can now readily provide digital images of the histopathology to help your understanding. Finally, no pathologist will be offended if you ask for a second opinion on a slide and should be able to advise on an appropriate colleague to provide that opinion.

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ISFM - FELINE CARDIOLOGY

CARDIAC NEOPLASIA IN CATS: EXPECT THE UNEXPECTED

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Measurement of N-terminal pro-B type natriuretic peptide (NTproBNP) and cardiac troponin I (cTnI) has now become commonplace in general and referral practices. These assays offer a straightforward and accessible test, which often feature in the diagnostic investigation of feline cardiac disease by veterinarians in general and referral practice alike. However, our understanding of the clinical utility of these laboratory tests is ever evolving as more research in the area is published.

Table 1 highlights common clinical scenarios in which cardiac biomarkers may be measured, and briefly summarises the evidence behind the noted utility (table 1 is at the end of this section).

Clinical use of the patient-side NTproBNP SNAP test: can it help to identify which heart murmurs to investigate?

A potentially useful test for assessing which heart murmurs to investigate and which to monitor/reassess in time is the patient-side NTproBNP SNAP test. It may also be useful as part of pre-anaesthetic screening in geriatric cats, where heart disease is common.

This test was validated by comparing a population of cats with moderate to severe cardiomyopathy ("clinically significant") to a population of cats with either normal echos or mild cardiomyopathy ("not clinically significant") (Machen et al, 2014). The SNAP test works well on EDTA plasma and has a positive cut-off value of approximately 120 pmol/L NTproBNP. If the value is negative, the NTproBNP is probably < 100 pmol/L. Occasionally it may read equivocal if between these values.

The test performed well in validation. To get a feel for how clinically useful the test is in asymptomatic cats with heart murmurs, we should consider the test specifics by Machen et al and combine these data with the prevalence data from Payne et al (Table 2).

In summary, a negative test is highly likely to mean that a cat does not have significant heart disease. A positive test is less useful, but it means that echocardiography is indicated (or even just monitoring of respiratory rate, care with intravenous fluids, cautious selection of sedation/ anaesthesia protocols, etc — if echo is prohibited by cost or local access to expertise). In older cats, a positive test is associated with a greater likelihood of clinically significant heart disease being present.

Age group	Pre-test likelihood of clinically significant heart disease (i.e. prevalence of HCM)	Post-test likelihood of clinically significant heart disease	
		Positive SNAP test	Negative SNAP test
6 - 12 months	17.9%	50.8%	3%
1 - 3 years	24.5%	60.6%	4.4%
3 - 9 years	30.1%	67.1%	5.7%
≥ 9 years	42.6%	77.8%	9.4%

Table 2: How using the NTproBNP SNAP test may help you to assess likelihood of significant heart disease being present in cats with heart murmurs

As you can see from Table 2, using the NTproBNP SNAP test allows you to further refine the process of deciding which cats with a heart murmur are likely to require echocardiography. To expand upon an example of a middle aged female cat, with a grade II/VI systolic heart murmur:

- Around 30% of cats like this have disease. If you recommend echo to every cat like this, around 70% of the time there will be no heart disease that is significant to that cat. However, if you were to use the NTproBNP SNAP test, you could be more selective in which owners to recommend echocardiographic assessment.
- If the SNAP test were positive, this individual cat has a 67% chance of having heart disease. This is clearly not a perfect test for heart disease, but it does mean that there is now more than twice the likelihood of heart disease being present in this cat (probability was 30.1%, now is 67.1%).
- If the test were negative you could explain to the owner that there is around a 1/20 chance the cat has significant heart disease (5.7%). Echocardiography may be undertaken, but just under 19 out of 20 times the echo will not show clinically relevant cardiomyopathy.

Clearly, the NTproBNP SNAP test should not be used to make decisions regarding treatment and prognosis, because it does not provide a certain diagnosis. However, it may be helpful in assessing need for, or urgency to, perform echocardiography.

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Clinical context	Cardiac troponin I (cTnl)	NTproBNP	Level of evidence
Respiratory distress: determining cardiac from noncardiac causes	Higher cTnl in cardiac disease, but significant overlap between groups limits clinical use	Higher NTproBNP in cardiac disease. Plasma: cut-off value around 200 pmol/L may be useful, sensitivity >85%, specificity 84-88%. Pleural fluid can also be used; apparently more sensitively than plasma and negating requirement for a blood sample.	High for both, NTproBNP more accurate
Identification of cats with occult cardiomyopathy	No specific studies; reported increase in cTnl for cats with subclinical heart disease	Higher NTproBNP in cats with subclinical cardiomyopathy. Cut-off value around 100 pmol/L appears useful. Higher concentrations associated with more severe disease, but insensitive for determining which cats have mild disease. A patient-side test with a cut-off at this level ("abnormal" result >100pmol/L) has been validated in a screening population to determine cats with moderate to severe heart disease, likely to be clinically significant, but the test has not yet been validated in a general practice population of older, non-pedigree cats.	Higher for NTproBNP
Prognostic use in cardiomyopathy	Higher cTnl in cats that dies because of cardiac disease vs. noncardiac death. cTnl >0.7 ng/ml associated with a shorter survival time in HCM, independent of clinical size or left atrial size, possibly reflecting severe ischaemia or infarction.	Higher single NTproBNP measurement associated with reduced survival, but not useful once presence of clinical signs or left atrial size are considered. Reduced NTproBNP during hospitalisation (serial measurements; admit and discharge) was associated with a longer survival time than cats where NTproBNP did not reduce significantly.	High for both

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Hyperthyroidism	cTnl increased in the hyperthyroid state, to the same levels as cats with heart disease. Normalises once euthyroid state achieved by radioactive iodine therapy.	NTproBNP increased in the hyperthyroid state, to the same levels as cats with heart disease. Normalises once euthyroid state achieved by radioactive iodine therapy.	High for both

Chronic kidney disease	cTnl higher in cats with azotaemic CKD than healthy age-matched controls. No correlation between creatinine and cTnl.	Higher NTproBNP in cats with IRIS stage IV renal disease, but no correlation with creatinine concentration overall.	High
Systemic hypertension	Increased cTnl in cats with hypertension secondary to CKD – higher still in cats with evidence of hypertensive retinopathy. However, no significant reduction with successful antihypertensive treatment and few cats underwent echocardiography.	NTproBNP increased in cats with hypertensive retinopathy than healthy controls. Few echocardiographic results available, so incidental cardiomyopathy cannot be excluded.	Medium
Anaemia	Increased cTnl in a group of anaemic cats than a control group of non-anaemic but systemically unwell cats. No echocardiography performed. Different exclusion criteria for test vs. control groups.	No published studies	Low cTnl No published studies for NTproBNP

WSV18-0296

SVA SOFT TISSUE SURGERY

SURGICAL MANAGEMENT OF BRACHYCEPHALIC SYNDROME

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Key Points

• English bulldogs are significantly over-represented.

• Light general anesthesia is required for accurate evaluation of laryngeal function and defects.

• Limited use of crushing clamps and cautery results in less postoperative swelling. • Overall prognosis for dogs with brachycephalic syndrome is favorable.

If you would like a video of these surgical procedures on DVD go to www.videovet.org or contact the author at videovet@me.com. You may click on the 'Seminar Price' for any DVD you would like to purchase.

Definition: Brachycephalic syndrome is a combination of upper airway disorders commonly seen in brachycephalic breeds (e.g., English bulldog, Boston terrier, Pugs). Disorders associated with this syndrome include stenotic nares, elongated soft palate, and everted laryngeal saccules. Occasionally patients present with laryngeal collapse. Patients may present with any combination of above listed disorders.

Diagnosis

Clinical presentation:

Signalment: Brachycephalic breeds are most commonly affected (i.e., English bulldog, French bulldog, Boston terrier, Pug, Pekingese, Boxer, and Bull Mastiff). The age at presentation ranges from less than one year to 11 years. The majority of patients present between 1 and 4 years with English bulldogs presenting at a younger age than other breeds. There is no apparent sex predisposition.

History: Historical findings are generally related to upper airway obstruction and include noisy respiration, heat intolerance, exercise intolerance, cyanosis, and occasionally syncopal attacks. Gagging, retching, and vomiting may also be reported. Historical findings may vary depending upon the number of abnormalities present (i.e., stenotic nares, elongated soft palate, and/or everted laryngeal saccules). Generally, the more abnormalities present the more severe the historical and clinical findings.

Clinical signs: The most frequently reported clinical signs in patients with brachycephalic syndrome include noisy respirations and exercise and/or heat intolerance.

Moderate to severely affected patients or patients with multiple defects may present with cyanosis and/or syncope.

Physical examination: Physical examination is generally normal except for patients with stenotic nares. In patients with stenotic nares the wings of the nostril (i.e., dorsolateral nasal cartilage) obstruct airflow resulting in turbulent airflow and resultant noise.Examining the patient after exercise may exacerbate clinical signs (i.e., noise and exercise intolerance) making diagnosis of brachycephalic syndrome more likely. Oral examination of the awake patient is generally unrewarding as the laryngeal apparatus and related abnormalities cannot be seen without light general anesthesia.

Radiography: Diagnosis of brachycephalic syndrome is based on signalment, history, physical examination, and direct visualization of the laryngeal apparatus with the patient under light general anesthesia. Thoracic radiographs are generally recommended to rule out lower airway disorders such as tracheal hypoplasia and pulmonary abnormalities.

Differential diagnosis: Any disorder causing noisy respirations, exercise intolerance, cyanosis, and syncope. Included are laryngeal mass, laryngeal collapse and laryngeal paralysis.

Medical management: Medical management is directed at decreasing airway turbulence and subsequent inflammation and edema. Strict confinement, antiinflammatory medications (e.g., steroids, NSAIDS), and a cool environment are recommended. Obese patients should be placed on a weight reduction diet plan. As medical management does nothing to change the anatomic deformity of the disorder, it is considered palliative but not curative.

Surgical treatment: The objective of surgical treatment is to provide an adequate airway by relieving any anatomic obstruction.

Preoperative management: Use of anti-inflammatory medication preoperatively is generally recommended. Patients are given intravenous steriods (dexamethasone 0.5 - 1 mg/kg IV) at the time of anesthetic induction.

Anesthesia: Anesthetic management is somewhat dependent upon the severity of clinical signs at presentation and degree of airway abnormality.

Patients with mild signs may be anesthetized with the clinicians standard anesthetic protocol. Careful evaluation of the laryngeal apparatus is performed prior to intubation and while the patient can still breath on its own (i.e., light general anesthesia). Laryngeal function is carefully evaluated during inspiration and expiration.

Patients with moderate clinical signs may need to be preoxygenated prior to induction. Induction should be performed quickly, the laryngeal anatomy and laryngeal function examined thoroughly, and the patient intubated to establish an open airway.

Patients with severe clinical signs should be preoxygenated 5 to 10 minutes prior to induction. A vagolytic agent (i.e., atropine) should be considered 10 to 15 minutes prior to induction because vagal tone is generally increased and cardioinhibitory reflexes are enhanced. Induction should be quick, examination of the laryngeal anatomy and function performed, and the patient intubated to establish an open airway.

Laryngeal examination: Once the patient is under a light plain of anesthesia laryngeal function is evaluated. Care is taken to observe for evidence of laryngeal collapse, elongated soft palate, and everted laryngeal saccules.

Surgical anatomy: The soft palate in the dog forms a long and broad movable partition between the oral and nasopharynx. The cranial border is attached to the bony palate; the caudal margin forms the dorsal border of the opening from the mouth into the pharynx. This portion of the palate is in contact with the epiglottis during normal inspiration; during deglutition, the epiglottis moves away from the soft palate to protect the opening of the glottis. At the same time the soft palate moves dorsally to close the nasopharynx and prevent regurgitation of material into the nasal cavity. The dorsal nasopharyngeal surface has a mucous membrane lining continuous with that of the nasal cavity and a slightly convex contour. The mucous membrane of the ventral concave surface is a continuation of the lining of the hard palate and is referred to as the oral surface of the soft palate.

Relevant pathophysiology: Protrusion of an elongated soft palate into the laryngeal inlet during respiration significantly obstructs air passage into the glottis. Stenotic nares, when present, contribute to the severity of the occlusion by increasing the inspiratory effort (and subsequent negative pressure) thus drawing the soft palate deeper into the larynx. Edema and inflammation result from friction against the epiglottis during each respiration. The resultant thickening further lessens airflow. As increased inspiratory effort continues, increased negative pressure in the airway encourages laryngeal saccules to evert.

Positioning: Patients may be positioned in ventral or dorsal recumbancy.Stenotic nares: The author prefers ventral recumbancy with the head supported on towels so the head position is normal and functional.

Elongated soft palate and everted saccules: Patients can be operated in either ventral or dorsal recumbancy. In dorsal recumbancy, the maxillary canine teeth are taped securely to the operating table. The mandibular canine teeth are taped to an ether stand situated over the patients head. The mouth is opened wide to enhance visualization. This positioning is critical as oral

cavity exposure is key to adequate visualization and instrumentation.

In ventral recumbancy, the maxillary canine teeth are hooked over the bar of an ether stand. The mandibular canine teeth are then taped to the operating table in such a fashion that the mouth gapes open. The tongue is grasped with tongue forceps and gently pulled from the mouth.

Surgical technique: The surgical technique varies depending upon the defect to be repaired.

Stenotic nares: This technique is illustrated on the Respiratory Surgery I DVD available via www.videovet. org.

Stenosis is decreased by removing a horizontal wedge of alar cartiladge from the wing of the nostril. The flap created is sutured to remaining tissue of the wing of the nostril using 3-0 or 4-0 Dexon or Vicryl in a simple interrupted suture pattern. Two or three sutures is all that is generally required to complete the nasoplasty.

An alternate technique gaining popularity in Shih Tzu and Boston breeds is to completely excise the alar cartilage. Bleeding is controlled by wedging a gauze sponge in the patients nostril for 5 minutes by the clock.

Presurgical temporary tracheostomy?: Use of a presurgical tracheostomy facilitates exposure and visualization of the soft palate and laryngeal saccules. However, it is not necessary in the majority of patients. The author considers use of a tracheostomy in patients that present with severe clinical signs (i.e., cynosis, syncope) and have a combination of defects to repair. Tracheostomy is preferred over exiting the endotracheal tube through a pharyngostomy as the tracheostomy can be used in the postoperative management of the patient if necessary. In our hospital, regardless of the severity of the airway obstruction, the patient is recovered in a critical care environment and instruments necessary to perform an emergency tracheostomy are readily available.

Elongated soft palate: This technique is illustrated on the Respiratory Surgery I DVD available via www.videovet. org.

The patient is placed in ventral or dorsal recumbancy with the mouth opened widely (see positioning). A broad malleable retractor is used to retract the tongue caudally; this greatly facilitates visualization of the soft palate and laryngeal structures. A headlamp facilitates visualization but is not necessary. Since postoperative edema and swelling are of major concern following soft palate surgery, it is important to keep surgical trauma to a minimum. Use of clamps and electrocautery may cause excessive surgical inflammation and should be avoided. The soft palate is evaluated for extent of resection. A Babcock or Allis tissue forceps is used to grasp the

caudal margin of the soft palate. The length of the soft palate in relation to the tonsil and epiglottis is examined. The soft palate should extend no further caudal than the midpoint of the tonsil. Alternately, the incision is made at the point where the soft palate just slightly overlaps the tip of the epiglottis.

Once this line of excision is determined, a 3-0 or 4-0 Dexon, Polysorb or Vicryl stay suture is placed in the soft palate on each lateral margin of the proposed excision. A third stay suture is placed on the margin of the central portion of the soft palate. The incision is begun from the left or right margin and one-third to one-half of the soft palate is incised.

Using the long end of one of the 3-0 or 4-0 Dexon, Polysorb or Vicryl stay sutures, the incised nasal mucosa is sutured to the incised oral mucosa using a simple continuous suture pattern. Dexon, Polysorb or Vicryl is chosen because of its soft supple nature; Maxon, Biosyn or PDS are much to stiff and may cause irritation to the oral cavity. Hemorrhage is controlled by suture pressure. No attempt is made to cauterize or clamp bleeding vessels. When the palate excision and suturing are complete, the stay sutures are cut and the remaining soft palate replaced and evaluated once again for extent of resection.

Everted laryngeal saccule resection: There is some suggestion that if the stenotic nares and elongated soft palate can be successfully treated (see above), the lateral saccules will return to their normal location in the larynx and no longer cause airway obstruction without the need for surgical resection. The author only removes lateral saccules in patients that present with severe respiratory signs (i.e., severe cyanosis, syncope).

When removing laryngeal saccules, the patient is placed in dorsal recumbancy with the mouth opened widely. Everted laryngeal saccules appear as edematous, translucent tissue balls lying in the ventral aspect of the glottis and obscuring the vocal folds.

Surgical removal is performed using a sharp long-handled laryngeal cup biopsy forceps (or similar long handled biopsy instrument) or a long handled Allis tissue forceps and #15 BP scalpel blade. If a laryngeal cup biopsy forceps is used the everted saccule is grasped and amputated with the biopsy forceps. Any remaining tags are grasped with a long-handled DeBakey forceps and trimmed with a #15 BP blade or scissors. If an Allis tissue forceps is used the laryngeal saccule is grasped with the Allis forceps and a long-handled scalpel with a #15 BP blade is used to excise the saccule at its base.

If the patient had a tracheostomy tube placed prior to surgery, the saccules are easily visualized and excised as described above. If the patient has an endotracheal tube exiting the laryngeal apparatus, the tube is temporarily removed while the saccules are excised.

Suture material/special instruments: Malleable retractors, head lamp, long-handled laryngeal cup biopsy forceps (or similar instrument), long-handled Allis tissue forceps, long-handled scalpel handle, long-handled rat tooth forceps, 3-0 or 4-0 Dexon, Polysorb or Vicryl with a cutting or sharp taper needle.

Postoperative care and assessment: Any patient requiring surgery to relieve airway obstruction should be monitored carefully (preferably in a critical care environment) for the first 24 hours postoperatively. The degree of care may vary depending upon the patients presenting signs and surgical manipulations required to correct the airway obstruction. Examples of the authors degree of postoperative care based on patient presentation and surgery performed are listed below:

Stenotic nares only: These patients are generally held for observation 12 24 hrs postoperatively and discharged from the hospital the day following surgery.

Soft palate resection only: Patients that present with mild clinical signs (i.e., noise, mild exercise or heat intolerance) and are bright and alert 24 hours after surgery can be discharged that day. Patients that present with moderate to severe clinical signs (i.e., severe exercise intolerance, episodes of cyanosis, syncopal attacks) are monitored in a critical care environment until signs resolve. Immediate postoperative gagging and coughing are observed in about 13% of patients. Patients requiring a tracheostomy prior to surgery, or an emergency tracheostomy, remain in a critical care environment until the tracheostomy can be removed.

Combined nares, palate, saccule repair: These patients are treated similarly to patients with soft palate resection and are based on presenting clinical signs. Patients with multiple defects tend to present with moderate to severe clinical signs and may require more intensive care. Immediate postoperative gagging and coughing are observed in about 80% of patients.

Patients that present with mild clinical signs (i.e., noise, mild exercise or heat intolerance) and are bright and alert 24 hours after surgery can be discharged that day. Patients that present with moderate to severe clinical signs (i.e., severe exercise intolerance, episodes of cyanosis, syncopal attacks) are monitored in a critical care environment until signs resolve. Patients requiring a tracheostomy prior to surgery, or an emergency tracheostomy, remain in a critical care environment until the tracheostomy can be removed.

Prognosis: Prognosis for patients with brachycephalic syndrome is generally dependant upon the defects found at presentation.

Stenotic nares only: About 96% of dogs with stenotic nares will improve postoperatively.

Soft palate resection only: About 85 90% of dogs with soft palate resection only will improve postoperatively. Young dogs (i.e., less than 2 years of age) are more likely to improve (90%) than dogs greater than 2 years of age (70%).

Stenotic nares and soft palate resection: Dogs having a combination of stenotic nares repair and soft palate resection are more likely to have a favorable outcome (96%) compared to those that did not (70%).

Soft palate and everted saccule resection: Dogs having this combination of defects repaired will have an 80% chance of significant improvement postoperatively.

WSV18-0066

SVA EXOTICS

BEHAVIORAL DEVELOPMENT IN CHICKS, AND MANAGEMENT OF ASSOCIATED PROBLEMS

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Many of these problems are often chronic by the time the bird is presented to veterinarians. Early recognition and treatment of these problems is more likely to result in successful treatment.

Understanding bird behaviour

To understand bird behaviour, it is necessary to go back and look at the behaviour of birds in the wild - after all, most pet birds are only a few generations removed from wild birds. However, to generalise and talk about 'bird behaviour' is akin to talking about 'mammalian behaviour'. There are some 9,000 species of birds; approximately 360 of these are parrots. Parrots have evolved in Australia, South America, Africa and the Asian region. Different environments and environmental pressures have shaped both the physical and psychological development of these birds, and to try and apply 'rules' across all 360 species is fraught with danger. However, there are some generalisations that can be made.

Avian behaviour can be categorised into two functional groups:

- Self-maintenance behaviours, designed to accomplish a specific task to maintain the health of the individual. These include feeding, feather care, locomotion, and concealment; and
- Social behaviours, designed to communicate information to another individual. These behaviours include territoriality, concern or fear, and courtship.

Parrots are altricial – the young are hatched near-naked, blind and perhaps deaf. They live for the first weeks of their life in a quiet, dark hollow, completely dependent on their parents. Social interactions at this stage are limited, confined to their siblings and parents. After fledging, many parrots learn to socialise by contact with their parents, siblings, and other birds (often their own age) in a flock. An example of this is the galah (**Eolophus roseicapillis**). In the wild clutches of 1 - 2 chicks, or more than 5, rarely survive. Once they fledge, the chicks are taken to a nearby 'crèche', a tree or stand of trees where other galah 'families' are interacting. Here they learn early social skills before joining a nomadic juvenile flock where they remain until reaching sexual maturity. This socialisation period teaches the juvenile birds food recognition (and how to locate it); predator recognition; sentinel duties; grooming behaviour; survival skills and early social behaviours.

As they reach sexual maturity, they learn new skills: how to select a mate, exhibit courtship behaviours and develop a pair bond; how to select, prepare and defend a nest site; and how to reproduce and raise their young.

Some of these behaviours are believed to be innate or instinctive; others are learnt. All are reinforced by the reaction the bird receives. Captive parrots, especially those hand-reared as pets, may not have the opportunity to learn these behaviours. Their instinctive behaviours though – particularly as they reach maturity – may bring them into conflict with their human flock.

How do behavioural problems develop?

Problems seen in clinical practice can be attributable to one of two causes.

- The first basic problem is a failure of the socialisation process. This is usually the result of an individual bird being hand reared in isolation and not being taught basic social skills. Once weaned, the bird is often ignored as its novelty value wears off. This process often results in attention-demanding behaviour (begging calls, screaming, feather chewing, etc.), displacement behaviours such as biting; feather damaging behaviour, phobias and sometimes even self-mutilating behaviour.
- The second group of problems result as a failure of the human 'flock' failing to understand normal parrot behaviour, and expecting birds to 'fall into line' with their human expectations. It was once said that there are no abnormal behaviours – just normal behaviours expressed inappropriately. Behaviours such as screaming morning and night, displaying territoriality and certain reproductive behaviours are examples of normal behaviour that are inappropriate in a companion bird scenario.

So how do these problem behaviours develop?

The reinforcement of self-maintenance behaviours benefits companion birds – and abnormal self-maintenance behaviours are the most common behavioural disorders seen in these birds. They still have the same self-maintenance behaviours as their wild counterparts. However, they need less time for foraging and feeding behaviours (after all the food is in a dish in front of them every morning), and therefore feather care, social communication and displays make up more of their daily activities.

Young captive birds need continued mentoring and behavioural moulding, and require guidance for the establishment of a normal bird-human flock relationship. This includes a range of normal social behaviours of flock interaction, with appropriate rules of conflict resolution and appropriate maintenance and social behaviours.

Failure to be taught – or learn – these behaviours means that many young birds are not prepared for a life in captivity, and may develop behavioural problems. In the absence of imposed rules, the bird will make its own rules, based on immediate gratification and revolving

around perceived value; however these rules may not be socially acceptable. They eventually develop into behavioural problems and the bird becomes unable to socially interact with people without fear or social framework, and therefore a series of displacement or defensive behaviours develop – aggression, biting, etc. As these behaviours develop, the bird may become even more isolated, and therefore become more vocal in trying to re-establish contact with their 'flock'.

And this is often reinforced when owners respond: the bird receives a positive response (e.g. talking, feeding, etc.), it may augment the behaviour; if, on the other hand, the bird receives a negative response (e.g. covering cage, time-out, water pistols) that response may augment the feeling of isolation, and the problem may worsen.

Unfortunately, these problems are often chronic by the time the bird is presented to a veterinarian. Early recognition and treatment are much more likely to result in successful treatment; prevention through education of bird owners is a far more preferable approach. Techniques such incorporating behavioural training into annual wellness examinations are important steps in preventing problems, and should be pursued vigorously by all those involved in the wellbeing of companion parrots.

DIAGNOSTIC IMAGING AND GASTROENTEROLOGY (SIMULTANEOUS TRANSLATION INTO MANDARI

CANINE MEGAESOPHAGUS

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Introduction:

Megaesophagus (ME) is characterized by generalized dilatation of the esophagus. It is the most common cause of regurgitation and a common esophageal disease in dogs. Causes of acquired megaesophagus include: idiopathic ME, myasthenia gravis (MG) - generalized or focal, esophagitis, endocrine diseases, toxicosis (e.g. lead). Less common causes include: other neurologic diseases (botulism, tetanus, dysautonomia, rabies), systemic lupus erythematosus

Clinical presentation:

Clinical signs consistent with esophageal disease include: regurgitation, dysphagia, swallowing attempts "on empty", halitosis, ravenous appetite, tachypnea, dyspnea, cough, exercise intolerance, fever (in case of aspiration pneumonia)

Diagnostic approach:

The first step in the diagnostic approach is to rule out esophageal obstruction (foreign body, stricture) by taking a detailed history, and getting good quality thoracic radiographs. A generalized dilatation of the esophagus is usually easily identified on the thoracic films. Generalized dilatation is typically due to functional disease whereas segmental dilation is more commonly associated with foreign body, infiltrative disease (neoplasia or inflammation, hiatal diseases, segmental motor disease, stricture, ring anomaly and redundant esophagus. Radiographically, the dilated esophagus can be gas or fluid opaque depending on the disease process. Radiographic signs include visualization of the esophagus, dilatation with gas, retention of food or fluid, tracheal stripe sign, ventral displacement of the intrathoracic trachea, and ventral displacement of the heart. In addition, it is important to carefully check for aspiration pneumonia in these patients.

The systematic approach is continued by screening animals for primary, underlying diseases associated with secondary megaesophagus. A minimal database consisting of CBC, biochemistry panel (including serum creatine kinase) and urinalysis is recommended. Then, screening for endocrinopathies (resting cortisol or ACTH stimulation test, serum thyroxine and endogenous TSH), and search for opacities in the cranial mediastinum (looking for possible thymoma associated with myasthenia gravis [MG]) are recommended. A sensitive

and specific serum assay documenting the presence of autoantibodies against nicotinic acetylcholine receptors is available for the diagnosis of MG http://vetneuromuscular.ucsd.edu. Endoscopic examination of the esophagus may be useful in unclear cases and to allow visualization of extent and severity of esophagitis, if present. In cases with suspected polymyositis or polyneuropathy electrodiagnostics (EMG, nerve conductions studies) and collection of muscle and nerve biopsies for histopathologic evaluation should be considered. In endemic lesions, infection with the parasite **Spirocerca lupi** should be ruled out (see esophagitis lecture).

Management:

Dietary management is of central importance - it is important to try various options such as dry food kibbles, canned food in meat balls and food blended with water in different consistencies (thick or thin slurry) because each animal may respond differently. Dogs and cats with ME should be fed a caloric-dense diet in a vertical position and be maintained in that position for 10-15 min. after the meal in order to use gravity to facilitate aboral movement of the food bolus. Small dogs can be held on a person's lap for that time. Medium size and large dogs can be fed on stairs and maintained with their front limbs higher than their hind limbs. The Bailey chair is a useful device to keep medium size and large dogs in a vertical position during and after meals. This aspect of treatment can be a challenge for dogs with pre-existing orthopedic diseases such as coxofemoral arthritis. In severe cases, placement of a gastrostomy tube may be beneficial to ensure appropriate nutrition and timely delivery of oral medications to the stomach, and to prevent aspiration pneumonia.

Identified underlying diseases need to be treated. For instance, recommended treatment of MG includes administration of acetylcholinesterase inhibitor pyridostigmine (1-3 mg/kg PO q12h, start with a low dose to minimize risk of cholinergic crisis) and immunosuppressive doses of prednisone or prednisolone (1-2 mg/kg PO q12h). Gastric prokinetic drugs do not significantly influence esophageal motility, while some of them increase LES tone in dogs (cisapride, erythromycin). Bethanechol is a cholinergic agent that has been shown to increase esophageal motility in some dogs and can be used in the management of clinical cases of idiopathic ME (5 to 15 mg/dog PO q8h, start with a low dose to minimize risk of cholinergic crisis). Because of the high prevalence of esophagitis in patients with ME, sucralfate suspension should be administered to facilitate mucosal healing (0.5-1 g/dog q8h). Treatment of aspiration pneumonia is mostly supportive. If secondary bacterial infection is suspected, antibiotic treatment is best based on culture and sensitivity from a tracheal or bronchial wash, however empiric treatment with a broad-spectrum antibiotic may be necessary in some cases (e.g. amoxicillin and clavulanic acid, ampicillin and sulbactam).

Home management of pets with megaesophagus is complex and time-consuming. Owners may feel overwhelmed and discouraged at first. Therefore, optimal communication between the veterinary care team and the animal's owners is essential for successful management of this condition, and web-based (e.g. Facebook) support groups may be very helpful.

Prognosis:

Studies from Scotland and Germany reported a short median survival time of 3 months after diagnosis regardless of the etiology in dogs with ME. In the Glasgow study, 41% dogs survived 1 year, 31% were alive at 2 years, and 22% survived for 5 years. Identified risk factors for shorter survival were dogs older than 13 months of age at the time of diagnosis and presence of aspiration pneumonia at diagnosis. Megaesophagus may be reversible when associated with endocrinopathies, but this represents only a small percentage of dogs with ME.

WSV18-0029

CLINICAL PATHOLOGY

USE AND INTERPRETATION OF COOMBS TEST

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USE AND INTERPRETATION OF THE COOMBS TEST

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This lecture will use a case study to demonstrate the use and interpretation of the Coombs test in canine medicine. The Coombs test might be requested in the following clinical situations:

- To confirm the presence of red cell-bound IgG, IgM or complement C3 in suspected cases of PRIMARY immune-mediated haemolytic anaemia (IMHA).
- To confirm an immune-mediated component in cases of SECONDARY IMHA (e.g. infection, neoplasia, drug or vaccine exposure).
- To determine the presence of red cell-bound antibodies in cases of suspected immune-mediated thrombocytopenia (IMTP; Evans Syndrome) or immune-mediated neutropenia (IMNP).
- As part of the diagnosis of systemic lupus erythematosus (SLE; a very rare disease), when anaemia is one clinical problem.
- To diagnose cold agglutinin disease.

The Coombs test

The Coombs test is used to demonstrate the presence of antibody and/or complement proteins on the surface of red blood cells (RBCs) in order to confirm a diagnosis of immune-mediated haemolytic anaemia (IMHA). In veterinary medicine, a direct Coombs test is performed, using RBCs from the patient. Detecting free serum antibodies specific for RBCs (the indirect Coombs test) is not performed diagnostically.

The sample required for a Coombs test is 2–5 ml EDTA anticoagulated blood (volume determined by the degree of anaemia). There are no specific handling requirements and the sample can be sent as for standard haematological examination. The attachment of antibody to RBCs is relatively robust and the test will still be positive after shipment. The test will also be unaffected if an animal has just started immunosuppressive therapy.

The first stage of the test, which may also be simply performed in practice, is the 'in saline agglutination test'. The tube of EDTA blood should be placed in a refrigerator (4°C) for 30 minutes and then evaluated for agglutination of RBCs. If there is visible evidence of agglutination, a drop of blood is placed on a microscope slide and diluted with a drop of saline. The slide may be gently rocked to mix the blood and saline. If the aggregated RBCs disperse, they are not true agglutinates (and are likely rouleaux formed in hyperproteinaemic plasma), but if they persist, then this is evidence of autoagglutination. This reaction may not be seen at room temperature or 37°C. The observation of autoagglutination is highly indicative of IMHA, but occurs perhaps only for one in every 10 dogs with the disease. The presence of autoagglutination does not necessarily preclude performing the Coombs test.

The Coombs test is a simple agglutination test. RBCs from the patient are 'washed' in phosphate-buffered saline (PBS) to remove the plasma and buffy coat, and the cells are then resuspended in PBS. Washed RBCs are then mixed with an antiserum (or a panel of different antisera) with specificity for the immunoreactants that might be expected to be found on the surface of RBCs in IMHA (i.e. IgG, IgM or complement C3). The antiserum will cross-link molecules of immunoglobulin or complement on the surface of the RBCs providing a visual read-out as agglutination.

Different diagnostic laboratories perform the Coombs test in different ways. Some use only one antiserum (the polyvalent canine Coombs reagent) to detect any immunoreactants present. The most thorough Coombs test uses polyvalent reagent in addition to separate specific antisera for IgG, IgM and complement C3, and fully titrates these antisera, while duplicating the test for incubation at both 4 and 37°C. Such testing provides much more complete information about the nature of the immunological reaction occurring.

In general, there are two broad patterns of Coombs test reactivity and these correlate well with the clinical presentation, severity of disease and prognosis. The most common pattern indicates the presence of a 'warm-reactive IgG antibody' where the test is positive for IgG alone, IgG + IgM (with or without complement) with similar titres at 4 and 37°C. This pattern is generally seen with dogs that have chronic compensated disease caused by extravascular haemolysis that carries a better prognosis. The less common pattern is that of 'cold-reactive IgM antibody' where the test is dominated by IgM antibody (with or without complement) with higher titre at 4°C. This pattern is more often are associated with acute onset disease, autoagglutination, intravascular haemolysis and a more guarded prognosis. Sometimes a mixed pattern of Coombs reactivity occurs in a single dog.

The Coombs test does not distinguish between primary and secondary IMHA, but unusual patterns, low-titre

reactions and cold-reactive IgM are more likely to be associated with secondary disease.

The chances of a false-negative Coombs test are minimized by performing the complete test as described above. However, it is possible for rare cases to have insufficient RBC-associated immunoreactants for detection or for circulating RBCs to be unaffected where the immune attack is directed against erythroid precursors in the bone marrow (non-regenerative IMHA or pure red cell aplasia).

In some laboratories, detection of RBC-bound immunoreactants is performed using fluorochrome-labelled antisera and the RBCs are passed through the laser beam of a flow cytometer. Flow cytometry is reported to be equally or more sensitive than the Coombs test, but the Coombs test is considered to be more specific than flow cytometry.

Some in-house rapid diagnostic tests have also been developed that provide a yes/no answer (i.e. is the dog Coombs positive?) within 30 minutes. Currently, in Europe the most widely available of these tests is that marketed by Alvedia (LabTest DAT). Not all of these tests have been validated against the gold standard full test (as described above), but those that have often show good correlation.

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WSVA8-0009

ALTERNATIVE MEDICINE, HOMEOPATHY AND ACUPUNCTURE

USE OF WU LING SAN TO TREAT KERATOCONJUCTIVA SICCA

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INTRODUCTION

Keratoconjuctivitis Sicca (KCS) or dry eye syndrome is a common condition in dogs and is a challenge to treat. The use of Wu Ling San alone or combine with other formulas provide another alternative to treat KCS. Wu Ling San consist of or five ingredients. In Shang Han Lun, Wu Ling San is used to treat when drinking too much of water but the Spleen can't distribute it causing water accumulation. This lead to disturbance of bladder's qi transformation follows with urine retention. Wu Ling San warm yang and transform Qi, drain water and harmonize surface. Wu Ling San can treat intense thirst with up surge of body fluid at the same time this formula should be able to produce fluid in the eyes.

OBJECTIVES

To evaluate the possibility of Wu Ling San to treat KCS.

METHODS

A KCS case was selected to follow the effectiveness of Wu Ling San treatment

RESULTS

A 10-year old male Cocker Spaniel was presented for thick green-yellow ocular discharge and KCS was diagnosed. The dog previously was treated for glaucoma. His eyes were still inflamed, swollen and having cataract. The tongue was red and pulses were thin and fast. Wu Ling San 1 tablet twice daily was given for 1 month. After 1 month the owner reported the KCS was resolved completely. The owner stopped medication for few months but recurred mush milder. Wu Ling San was prescribed again and ask to maintain on the medicine.

CONCLUSIONS

Wu Ling San can be used to treat KCS.

WSVA8-0010

ALTERNATIVE MEDICINE, HOMEOPATHY AND ACUPUNCTURE

USE OF TDP LAMP AND CHINESE HERBAL MEDICINE FOR TAN-HUAN SYNDROME IN A DOG

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INTRODUCTION

Spondylosis is a condition that affects the vertebral bones of the spinal column characterized by the formation of osteophytes or bony spurs along the edges of the vertebra body. TDP lamp emits a band of electromagnetic waves that are absorbed by the body through selective absorption. These absorbed electromagnetic waves help in generating various beneficial biochemical stimuli that the body lacks and accelerating decomposition of unstable structures such as dead cells Due to the cost and invasiveness of surgical approach TCVM approach was preferred in this case.

OBJECTIVES

To evaluate the effectiveness of 'Special Electromagnetic Health lamp' (TDP Lamp) and TCVM treatment for Tanhuan syndrome.

METHODS

A 5 years old female Pekingese cross Shih Tzu was presented and X-ray revealed spondylosis at C2-C3 and T13-L1. MRI detected compression of C2-C3 on the right side while there is compression of the T13-L1 on the ventral side. The dog was completely paralyzed with the diagnosis of Tanhuan in TCVM. The dog was then treated with the combination of acupuncture, Chinese herbal medicine, B12 tablet and TDP Lamp.

RESULTS

After two months of treatment, the owner decided to stop bringing the dog for acupuncture but bought the TDP lamp and applies it every day for at least half an hour a day at the comfort of home. The two herbal medicines and B12 tablet were continued. Since fourth month of treatment the dog slowly regained its neuromuscular function and recovered completely.

CONCLUSIONS

TDP light provide an alternative treatment besides surgical approach.

WSVA8-0064

ANESTHESIA

EVALUATION OF LIDOCAINE INJECTED INTO THE EPIDURAL SPACE VIA INTERCOCCYGEAL SITE PERCUTANEOUSLY IN RABBITS

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INTRODUCTION

Lumbosacral approach has been used for rabbit epidural anesthesia (Hughes et al. 1993). However, the technique was reported to cause intrathecal injection in all studied rabbits (Otero et al. 2012).

OBJECTIVES

To evaluate the effects of lidocaine injected into the epidural space via intercoccygeal site percutaneously in rabbits.

METHODS

Under general anesthesia with isoflurane following propofol induction, rabbits were placed in ventral recumbency with elevated hip. A 27-gauge spinal needle was inserted percutaneously at dorsal tail base in caudocranial direction. Correct needle placement in the epidural space was confirmed by contrast radiography using iohexol (0.05 – 0.1 ml/kg). Then, either 0.3 ml/kg of lidocaine 2% (n=4) or normal saline (NS) (n=4) was injected randomly in blinded fashion. Pulse rate and blood pressure (non-invasive monitoring) were recorded before and after the injection. One minute after the injection, general anesthesia was discontinued. Neurological examination was performed in blinded fashion before and every 20 minutes after general anesthesia until all findings back to normal; and reassessed one week later.

RESULTS

Lidocaine group has significant longer recovery time to normal anal tone, gait, conscious proprioception (CP), and deep pain perception (DPP) of hindlimbs, compared to NS group. No difference was observed for the recovery time of mentation and forelimbs' CP and DPP between two groups. Blood pressure significantly decreased after lidocaine injection, but not changed after NS injection. No complications were observed during one week follow-up period.

CONCLUSIONS

Percutaneous lidocaine injection technique into the epidural space via intercoccygeal site may be used for rabbit hindlimb anesthesia.

WSVA8-0023

ANESTHESIA

EFFECTS OF TRAMADOL AND TRAMADOL-LIDOCINE INFUSIONS ON THE MINIMUM ALVEOLAR CONCENTRATION OF SEVOFLURANE AND ENTROPY INDICES IN DOGS

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INTRODUCTION

Previous studies indicate anti-nociceptive effects of tramadol and lidocaine infusions. However, the effects of intravenous infusion of both tramadol and lidocaine on minimum alveolar concentration (MAC) sparing effects of sevoflurane in dogs remain unclear.

OBJECTIVES

To compare the effects of continuous rate infusion (CRI) of tramadol and tramadol-lidocaine on MAC of sevoflurane and entropy indices in dogs.

METHODS

Eight, healthy German shepherds were induced and maintained with sevoflurane. A standard tail-clamp technique was used for MAC determination. The MAC was determined with only sevoflurane for MAC baseline (MAC_B), during infusions of tramadol (intravenous loading dose of 1.5 mg/kg and CRI of 2.6 mg/kg/hr; MAC_T), and tramadol-lidocaine (tramadol CRI of 2.6 mg/kg/hr and intravenous loading dose of 1.0 mg/kg lidocaine and CRI of 6 mg/kg/ hr; MAC_{TL}). Stated entropy (SE), response entropy (RE) and RE-SE difference were recorded at five minutes prior to and during tail-clamping.

RESULTS

The MAC_B was 2.4 ± 0.2%. Tramadol and tramadol-lidocaine decreased MAC to 2.2 ± 0.3% and 1.7 ± 0.3%, respectively. The MAC-sparing effect of tramadol-lidocaine was greater than tramadol alone (8.2 ± 8.9% vs. 30.1 ± 10.7%; *p*<0.01). All of SE, RE and most RE-SE difference were increased (all *p*<0.05) when subjects responded purposefully to the noxious stimulation. While no response occurred, all of entropy indices did not change compared to pre-stimulation data.

CONCLUSIONS

In dogs, a combination of tramadol-lidocaine infusion can reduce anesthetic requirements in higher degree than tramadol alone. Entropy indices may change associated with nociceptive responses in anesthetized animals.

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WSVA8-0179

ANIMAL WELFARE

A SURVEY OF HUMAN AND ANIMAL CASUALTIES RESULTING FROM BITES OF STRAY DOGS IN THE MUNICIPAL AREA IN PALAKKAD DISTRICT, KERALA

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INTRODUCTION

There are nearly 1,197 stray dogs in the municipal area of Palakkad district in Kerala. It is also estimated that when no stray dog control measures are undertaken by the various civic bodies, there could be up to 25 per cent yearly increase in the numbers of stray dogs. Increasing numbers of stray dogs pose significant safety threat to both humans and domestic livestock and hence, mass killings of stray dogs by the public happen at times

OBJECTIVES

We intended to examine the magnitude of threat to public safety from the bites of stray dogs in the l area.

METHODS

For the same, we undertook surveys and collected data from print and visual media on all reported cases of stray dog bites from January 2015 till date.

RESULTS

Over the last ten month period, nearly 3800 humans and 459 domestic animals were reported to have suffered stray dog bites in the municipal area. In humans, males (60%) suffered more bites than females (40%). Compared to humans, in animals, the number of reported bite cases is severely underestimated, mainly because of the poor surveillance systems for recording animal casualities. Most of the reported animal cases were in domestic goats and only a lesser number were in domestic cows and dogs

CONCLUSIONS

Unless the local civic bodies undertake adequate measures to control the numbers of stray dogs, changing the public perception as well as opinion against the mass killings will remain an uphill task.

WSVA8-0015

ANIMAL WELFARE

RABIES OUTBREAK PREVENTION SYSTEM BASED ON CUSTOMARY VILLAGE SOCIETY

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INTRODUCTION

Rabies outbreak prevention system based on customary Village Society is one of the options according to the Balinese people's condition that still strong on the culture social life A disease prevention effort with approaching of the social culture of the society is very important, where is in this system the government and rural communities and indigenous peoples work each other to implement the Communication, Information and Education program (IEC). This is aimed to keep the public informed so they can participate to implement the basic principles of animal welfare, rabies vaccination program, and perform sterilization

OBJECTIVES

The objectives of this activities were to: understand the human-dog relationship in Bali about animal welfare, explore local knowledge, attitudes, and practices (KAPs) relating to animal welfare and assess potential community-driven activities to optimize rabies control and surveillance

METHODS

These activities carried out by the Indonesian Veterinary Medicine Association Bali Region are: communication, information and education about animal welfare, rabies vaccination program and mass sterilization (Spay and Neuter)

RESULTS

The activities carried out by the Indonesian Veterinary Medicine Association Bali Region, has successfully campaigned on the importance of animal welfare, conduct mass sterilization programs, rabies vaccination program, and educational activities for villagers.

CONCLUSIONS

Rabies disease prevention approach to animal welfare community-based customary village society is a way that can be done in tackling the problem of rabies in Bali province.

WSVA8-0013

ANIMAL WELFARE

PREVALENCE OF HEALTH PROBLEMS IN SHELTER DOGS IN THE CZECH REPUBLIC

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INTRODUCTION

Health and injury status are considered major determinants of adoption in dogs. They influence the likelihood of adoption as well as the length of stay at the shelter.

OBJECTIVES

The aim of this study was to analyze a prevalence of health problems in shelter dogs.

METHODS

The subject of this retrospective study were dogs impounded at a Czech municipal shelter over a ten-year period (2007 to 2016). The results were analyzed using the statistical package Unistat 5.6. Statistical comparisons between frequencies of the categorical variables of interest were performed with the chi-square test within the contingency table procedures.

RESULTS

A significantly (P < 0.001) larger proportion of shelter dogs was clinically healthy. Whereas 63.4% of dogs at the monitored shelter were clinically healthy, 36.6% of dogs showed single or multiple signs of disease. Sex, size, age and purebred vs crossbred categories differed (P < 0.05) in terms of the frequency of clinical signs of disease. A greater prevalence of clinical signs of disease was found in males, in large dogs, in senior dogs (aged 9 years and more) and in purebred dogs. Most diseased dogs (41%) showed multiple clinical signs. Gastrointestinal disease was the most frequent single diagnosis, followed by respiratory diseases, skin diseases and injuries.

CONCLUSIONS

A positive finding is that the population of shelter dogs is healthier than expected. More than 60% of dogs available for adoption over a 10-year period at the monitored shelter had no clinical signs of disease.

WSVA8-0014

ANIMAL WELFARE

MORTALITY AND EUTHANASIA RATES OF DOGS AT A NO-KILL SHELTER

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INTRODUCTION

Even if dogs admitted to no-kill shelters are not in immediate danger of being killed, those that are not adopted may meet their ultimate fate there eventually.

OBJECTIVES

The aim of this study was to analyze the records on dogs impounded at a Czech municipal shelter in the period from 2007 to 2016.

METHODS

The results were analyzed using the statistical package Unistat 5.6. Statistical comparisons between frequencies of the categorical variables of interest were performed with the chi-square test within the contingency table procedures.

RESULTS

Over 90% of shelter dogs were adopted, whereas death was the ultimate outcome for only 6.4% (2.9% died and 3.5% had to be euthanized for health reasons). Significantly (P < 0.01) more males than females died or were euthanized at the shelter. Different age categories of dogs differed (P < 0.001) in terms of euthanasia and death rates. Unassisted death was most common in dogs younger than six months (44% of all deaths at the shelter) and was preceded by a short stay at the shelter. The major reasons for the unassisted death of dogs at the shelter were gastrointestinal diseases (42%), in particular infections. 60% of euthanasia was performed on senior dogs (aged 9 years and more) after prolonged care provided at the shelter. The most common reason for euthanasia in shelter dogs was multisystem organ failure (31%).

CONCLUSIONS

Despite the shelter context being different from that at breeding kennels, our results suggest that mortality and euthanasia rates in shelter dogs can be reasonably low.

ANIMAL WELFARE

FACTORS AFFECTING LENGTH OF STAY OF DOGS AT A NO-KILL SHELTER IN THE CZECH REPUBLIC

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INTRODUCTION

The physical features of the dog were reported to play a significant role in the choice of a dog to adopt.

OBJECTIVES

The aim of this study was to identify factors affecting length of stay (LOS) of dogs at a no-kill shelter in the Czech Republic.

METHODS

Records on characteristics of shelters dogs were collected from a Czech municipal shelter in the period from 2007 to 2016. The results were analyzed using the statistical package Unistat 5.6 (a two-tailed Mann-Whitney U test or a Kruskal-Wallis ANOVA).

RESULTS

The median LOS was 34.5 days. Males stayed at the shelter longer (P < 0.001) than females (median LOS 40 and 29 days, respectively). Medium and large dogs stayed at the shelter longer (P < 0.001) than small dogs (median LOS 42, 48 and 26 days, respectively). The median LOS of dogs increased significantly (P < 0.001) with increasing age from 18 days in the youngest category (less than six months) to 123 days in the oldest (aged nine years and more). Crossbreds stayed at the shelter slightly longer than purebreds (median LOS 35 and 32 days, respectively), however the difference in median LOS was not statistically significant (P > 0.05). Dogs with impaired health stayed at the shelter longer (P < 0.001) than dogs with no clinical signs of disease (median LOS 67 and 26 days, respectively).

CONCLUSIONS

Our findings are in agreement with the results of many foreign shelter studies showing an almost uniform perception of these traits across different communities.

BEHAVIOR

IMPACT OF ENVIRONMENTAL ENRICHMENT ON THE BEHAVIOR OF DOGS IN LABORATORY KENNEL

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INTRODUCTION

Laboratory dogs spend at least 10 consecutive days confined individually in restricted environment to measure diet digestibility. This can compromise well-being and lead to abnormal behaviors, such as stereotypies or undesirable behavior (coprophagy). Regarding this, environmental enrichment in laboratory dogs can reduce stress and contribute to their activities, decreasing leisure time and improving their well-being.

OBJECTIVES

We aimed to evaluate the behavior of experimental kennel dogs with and without environmental enrichment during the period of twenty days.

METHODS

Eight 6-year-old Beagle dogs were housed individually in concrete kennel with 4.7 meters in length and 2.22 meters in width. The kennels contained a water *ad libitum* and rubber mat for the dogs' rest. The experiment was carried out over a period of 20 days: the first 10 days without enrichment, in which the dogs were fed in conventional stainless steel pots. In the following 10 days, environmental enrichment was used with PetBall® (PetGames - São Paulo, Brazil) for food. The dogs were evaluated by cameras (AXIS, 3004-v) installed in points of the kennel where dogs could be fully visualized and monitored for 15 hours a day, totaling 60 hours of monitoring. The behaviors recorded were: walking, eating, coprophagy, exploratory, lying, standing, sitting, sleeping and social interaction.

Figure 1 - PetBall®.

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RESULTS

There was an increase in feeding time and exploratory behavior (P<0.05), on the other hand, the behavior of coprophagy decreased (P<0.05) in dogs that had environmental enrichment included.

CONCLUSIONS

Feeding dogs with PetBall® increases exploratory behavior and decreases coprophagy in laboratory dogs.

WSVA8-0012

BEHAVIOR

PREVALENCE OF BEHAVIOUR PROBLEMS IN DOGS ADOPTED FROM SHELTERS

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INTRODUCTION

Adoption success is influenced by many factors. The current research underlines specifically the importance of good behaviour to dog adopters.

OBJECTIVES

The aim of this study was to assess behaviour of dogs adopted from shelters as perceived by their adopters.

METHODS

In order to collect information on behaviour of adopted dogs, a questionnare was handed in to people adopting a dog from Czech shelters. The results were analyzed using the statistical package Unistat 5.6. Statistical comparisons between frequencies of the categorical variables of interest were performed with the chi-square test within the contingency table procedures.

RESULTS

According to the respondents of our survey, 72% of dogs exhibited behavioural problems in the first week after adoption. The most frequent behaviour problems in adopted dogs were aggression (24%), fearfulness (21%), destructiveness (17%), excessive vocalisation (15%) and separation anxiety (13%). 30% of adopted dogs exhibited more than one type of problem behaviours. No effect ($P > 0.05$) of sex, age, size or health status was found. However, a significant ($P < 0.05$) impact of abuse on occurrence of problem behaviours was found. Shelter dogs with documented history of abuse exhibited problem behaviours after adoption more frequently than non-abused dogs.

CONCLUSIONS

Given the fact how common the presence of problem behaviour in shelter dogs is and how often it was cited as the primary reason for relinquishment or returning dogs to animal shelter providing help to adopters to remedy common shelter dog behaviour problems could significantly increase the rate of successfully adopted dogs.

WSVA8-0060

CARDIO-RESPIRATORY MEDICINE AND SURGERY

DIAPHRAGMATIC HERNIA REPAIR WITH THE ASSISTANCE OF ANESTHESIA REBREATHING BAG

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INTRODUCTION

Diaphragmatic hernias presented to the hospital were due to trauma. A total of 5 procedures were done successfully using an anesthesia machine rebreathing bag for ventilation.

OBJECTIVES

To clear the thoracic region from herniated abdominal organs and return the normal respiration of the patient

METHODS

Complete blood count, serum chemistry, urinalysis and radiographs were performed prior to surgery of patients. Two teams of doctors were utilized; one to perform the surgery and other to do the anesthesia and life support. A ventral midline incision is made towards the cranial part of the abdomen from xyphoid to umbilicus.

Abdominal organs are then retracted into place. The Thoracic cavity is washed with saline solution and suctioned well while patient is on an inclined plane with the head higher than the rest of the body. Diaphragm closure is done dorsally to ventrally with a continuous suture pattern. Either an absorbable or non absorbable monofilament suture can be used to close the diaphragm.

The important part is 4breaths/minute or 1puff/15sec using the right bag for the patient size. Before total closure of the tear, a Fr.7 tube is inserted and negative pressure is applied. Timing is essential. Tighten the last knot while removing the tube to allow negative pressure to remain inside the thorax. Stop the assisted breathing and monitor the patient's normal respiration. The abdomen is then closed routinely.

RESULTS

All five cases were operated on successfully, with only one experiencing respiratory complications.

CONCLUSIONS

Diaphragmatic hernias may be successfully repaired without the use of a ventilator

WSVA8-0049

CARDIO-RESPIRATORY MEDICINE AND SURGERY

PULMONARY HYPERTENSION IN HONG KONGESE DOGS

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INTRODUCTION

Pulmonary hypertension (PH) is defined as persistent increase in pulmonary vasculature pressure and occurs as a primary or secondary disease.

OBJECTIVES

To evaluate retrospectively the prevalence and characteristics of PH in a random sample of 100 dogs from a database.

METHODS

The database included 2123 dogs scanned in Hong Kong over a period of 2.5 years. PH was diagnosed from velocity of tricuspid regurgitant jet > 2.8 m/s. Dogs were classified into different groups on the basis of their diagnoses. Age, breed, sex and clinical signs have been compared among different groups. In dogs where follow up echocardiograms were available, success of PH therapy was evaluated.

RESULTS

In the sample of 100 dogs there were 54 dogs diagnosed with PH; there were 40.7% of dogs with mild PH, 38.9% with moderate PH and 20.4% with severe PH. In the random sample 60% of all mitral valve disease cases had PH (89% of Class B2 or C). PH was more common in male dogs. Among the dogs with PH there were 92.6% of small breed dogs. Most common breeds of dogs with PH were Shih Tzu and Pekingese. Most common clinical signs of dogs with PH were exercise intolerance, syncope, cough and dyspnea/tachypnea. Dogs were treated for primary disease and pimobendan and/or sildenafil. Improvement of clinical signs and a decrease in pulmonary arterial pressure was observed in 76.2% of dogs with average decrease in pressure of 27%.

CONCLUSIONS

PH is common in Hong Kongese dogs.

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WSVA8-0028

CARDIO-RESPIRATORY MEDICINE AND SURGERY

COMPARISON OF DOPPLER-DETERMINED ELEVATED PULMONARY ARTERIAL PRESSURE BETWEEN SHIH-TZU, MALTESE TERRIERS AND OTHER SMALL BREED DOGS WITH DEGENERATIVE MITRAL VALVULAR DISEASE

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INTRODUCTION

Shih-Tzu is a brachycephalic breed and frequently suffers from respiratory distress. Chronic respiratory distress may cause of elevated pulmonary arterial pressure. Pulmonary hypertension (PH) is known as a negative prognostic factor for degenerative mitral valvular disease (DMVD) in small breed dogs.

OBJECTIVES

The aim of this study was to compare the incidence and severity of PH among Shih-Tzu and other small breed dogs with DMVD.

METHODS

Sixty-three client-owned small breed dogs (BW < 10Kg) affected with ACVIM stage B2 DMVD were included. A full physical examination, noninvasive blood pressure measurement, blood checkups, electrocardiographic and echocardiographic examination were carried out in all dogs. Echocardiographically, high tricuspid regurgitation (TR) velocity > 2.5m/s considered as PH. Dogs, except Shih-Tzus, with brachycephalic obstructive airway syndrome were excluded from this investigation. Dogs with right bundle branch block, tracheal collapse and primary/secondary lung tumors, positive reaction for circulating antigens of *Dirofilaria immitis*, and pulmonary stenosis were also excluded from this investigation.

RESULTS

The incidences of PH (TR > 2.5m/s) in Shih-Tzu, Maltese terriers and other small breed dogs were 72% (13/18), 30% (6/20) and 28% (7/25), respectively (*P* = 0.007). No significant differences in fractional shortening, LA/Ao (1.39 ± 0.17, 1.45 ± 0.16, 1.41 ± 0.15) were found. However, TR peak velocity (2.73 ± 0.98, 2.04 ± 0.73, 2.23 ± 0.87 m/s) was significantly higher in Shih-Tzus (*P* < 0.04).

CONCLUSIONS

Higher incidence PH was found in Shih-Tzu, compared with other small breed dogs with Stage B2 DMVD. Further studies of skull pattern in longterm management of DMVD are warranted.

WSVA8-0027

CRITICAL CARE AND EMERGENCY MEDICINE

SUCCESSFUL THERAPEUTIC USE OF SINGLE DONOR CANINE PLATELET TRANSFUSIONS SEVERELY THROMBOCYTOPENIC DOGS- A REVIEW OF THIRTY EIGHT CASES (2016-17) IN CHENNAI, INDIA

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INTRODUCTION

Ehrlichiosis, Babesiosis induced thrombocytopenia (TCP) are commonly encountered in small animal practice in India. Canine platelet transfusions are slowly gaining momentum in our country to counter bleeding diathesis.

OBJECTIVES

To prepare Platelet-Rich Plasma(PRP) from separate dog donors and transfuse PRP to the affected dogs with severe TCP with bleeding diathesis.

METHODS

Canine Platelet-Rich Plasma (PRP) was prepared using Triple blood bags. 350 mls fresh whole blood collected from separate donor dogs *(n=38)* and were centrifuged on a soft spin of 2000 x g for 10 minutes (braking time = 2 minutes, 30 seconds), the PRP was expressed into satellite bags and stored as 100ml single units at 24 °C for five days in the platelet agitator.

RESULTS

The mean Platelet Count was 5.0 (+/- 3.0) x 10 (10) platelets/PC (range, 3.3 to 6.4x 10(10) platelets/PC) and the mean (+/- SD) platelet yield from WB to PRP was 78 (+/- 13) % Hematocrit was 1 to 5% and PRP was negative for fungal and bacterial culture. Severe Thrombocytopenic dogs with Platelet Counts (PC) below 25,000 cells were transfused and after 24 hours good increase in the Platelet Counts (PC)above 1,25,000 cells / cmm were recorded.

CONCLUSIONS

Thrombocytopenic dogs with PC below 25,000 cells/cmm with petechiation, ecchymosis, purpura, epistaxis, haematuria, haemoglobinuria, melena were transfused with PRP to increase the platelet counts. During 2016-2017, 28 severely thrombocytopenic dogs referred to the TANUVAS Animal Blood Bank, Madras Veterinary College, were transfused with stored or fresh PRP from separate canine donors provided immediate short term local hemostatic effects.

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WSVA8-0148

CRITICAL CARE AND EMERGENCY MEDICINE

THERAPEUTIC OUTCOMES OF INJECTABLE FILGASTRIM IN EIGHT DOGS AFFECTED WITH PERSISTENT FEVER, ACUTE LEUCOPENIA AND NEUTROPENIA.

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INTRODUCTION

Filgrastim is a recombinant Methionyl Human Granulocyte Colony- Stimulating Factor (r-metHuG-CSF) analog used to stimulate the proliferation and differentiation of granulocytes in humans undergoing chemotherapy. Recently, dogs with persistent fever (T-105° F), severe acute leucopenia and neutropenia (below 600 cells/mm) have been presented with high mortality & were taken for study. Leucopenia from reduced production of white blood cells or increased utilization and destruction, or both are fatal. Apart from identification of the cause and effective antimicrobial therapy, recently the treatments are aimed in increasing the leucocyte counts to fight infections and related sepsis.

OBJECTIVES

To evaluate leucopenic dogs with persistent fever, post treatment with single dose subcutaneous Fligastrim injection (NEUPOGEN ®)

METHODS

Routine diagnostic tests including ultrasonography, radiography, lymph node aspirations, blood smears were done on affected dogs for confirmation. These severely leucopenic dogs were treated with Injection Filgrastim @ 10 μg / kg b.wt subcutaneously single dose along with CRI crystalloid therapy. Clinico pathogical studies were done pre and post treatments on day 1, day 2, day3 and day 5 following Filgastrim administration

RESULTS

Seven dogs with severe leucopenia (<600 cells/cmm) responded clinically within 72 hours and their leucocyte counts increased considerably (4,200- 5800 cells/cmm). whereas one dog succumbed without clinical and clinico pathological response. No adverse reactions were encountered within 24 hours post Filgastrim injections subcutaneously.

CONCLUSIONS

Filgrastim increased the leucocyte counts within 72 hours thereby improving the survival rates in affected dogs showing early critical SIRS. Further clinical trials need to be emphasised based on these findings.

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WSVA8-0128

DIAGNOSTIC IMAGING

AN ECHOCARDIOGRAPHIC STUDY OF A DOG WITH TRICUSPID VALVE DYSPLASIA

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INTRODUCTION

A 9 years old female mix breed (Dachshund X Pomeranian) dog weighed 9 kg was having difficulty to breath, exercise intolerance, anorexia, vomit, and a decrease in body weight. Then the dog was referred to Animal Teaching Hospital, Faculty of Veterinary Medicine - Bogor Agricultural University to get an echocardiography examination.

OBJECTIVES

This study aimed to investigate the cause of the clinical signs and the possibility of heart problems through an echocardiography examination.

METHODS

The anamnesis was recorded, and the physical examination was done to evaluate further health problems, and an echocardiography was performed as supporting diagnostic procedure.

RESULTS

Physical examination has shown that the dog was lethargic, arrhythmic, murmur heart sounds (on the right side), and also exhibit an abdominal pain. Further examination, the Bright-Mode (B-Mode), Motion-Mode (M-Mode), and Color Flow Doppler-Mode (CFD-Mode) echocardiography, were then performed. The echocardiography showed that the tricuspid valve was thickening and was not perfectly closing which caused a turbulence of blood flow. Moreover, there was also an enlargement of the left atrium even though the value of fractional shortening (FS) and ejection fraction (EF) were still in normal limit.

CONCLUSIONS

From these results, the diagnose for the dog was concluded as tricuspid valve dysplasia (TVD) and mild left atrial enlargement. Despite the rare number of TVD case in animals, but an immediate echocardiography examination should be performed in a patient with impaired cardiac function, to detect and identify potential heart problems as quickly as possible.

WSVA8-0084

DIAGNOSTIC IMAGING

COMPUTED TOMOGRAPHIC APPEARANCE OF CAUDAL ABDOMINAL LYMPHOCENTER IN HEALTHY CATS

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INTRODUCTION

In cats, diseases involving caudal area such as at lower urinary tract, perineum or hind limb can cause lymphadenopathy at intra-abdominal caudal abdomen. Due to less sensitivity of radiographs and lymph node location that may cause difficulty to detect by ultrasonogram, computed tomography (CT) can provide much more information. However, there has no information of normal appearance of feline caudal abdominal lymph node.

OBJECTIVES

Therefore, the aim of this study was to investigate the appearance of caudal abdominal lymph nodes in healthy cats.

METHODS

Abdominal pre- and post-contrast enhanced CT images were performed in 15 healthy cats, which divided into 3 groups; kitten, mature and senile cats. Then, the appearance, location and size (width, length and height) derived from multiplanar reconstruction were evaluated.

RESULTS

Among 15 cats, internal iliac lymph node was detected only in 5 cats with average size of 2.4 x 4.2 x 2.3 mm. In contrast, medial iliac and sacral lymph nodes were distinct on post-contrast CT images with average size of 4.1 x 11.8 x 3.2 and 3.5 x 6.9 x 3.6 mm, respectively. In addition, age of cats has effect on lymph node size. The result showed that kitten has significantly larger size of medial iliac lymph node than other groups (P = 0.0013).

CONCLUSIONS

In summary, in healthy cats, internal iliac lymph node is hard to be detected and age of the cat would effect to the size of lymph nodes. This information would be applied as reference values prior detecting lymph node abnormalities in clinical practice.

WSVA8-0144

DIAGNOSTIC IMAGING

ULTRASONOGRAPHIC BIOMETRY OF MEDIAL ILIAC AND JEJUNAL LYMPH NODES IN APPARENTLY HEALTHY DOGS

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INTRODUCTION

The medial iliac lymph node (MILN) and jejunal lymph node (JJLN) generally become hypoechoic, enlarged and detectable when inflamed or infiltrated secondary to reactive hyperplasia in animals.

OBJECTIVES

To standardise the dimensions of MILN and JJLN in apparently healthy dogs, to record the ultrasonographic features of these nodes and to correlate the dimensions of lymph nodes with age, body weight and body condition score (BCS).

METHODS

Ultrasonographic features and biometry were recorded in thirty apparently healthy dogs using LogiQ F8 machine.The observed data was statistically analysed to obtain means, 95% confidence intervals of the mean, standard error, minimum and maximum, correlations and ANOVA to compare the means based on age, body weight and BCS. The Pearson correlation coefficient, Beta coefficients and SEs obtained from univariate linear regression were also calculated.

RESULTS

The length and height of MILN ranged from 0.63-3.75 cm and 0.23-1.27 cm for left node and 0.78-3.98 cm and 0.21-1.03 cm for right node respectively. The length and height of JJLN was 0.21-4.12 cm and 0.26-1.24 cm respectively. The dimensions of the MILN showed significant correlations with age and body weight of the dog. However, BCS had no correlation with the dimensions of MILN. The dimensions of JJLN had no correlation with age, body weight and BCS.

CONCLUSIONS

Ultrasonographic biometry of MILN & JJLN for apparently healthy dogs was standardised. This can be used as a baseline data for evaluation of these nodes in dogs.The alterations in the sonographic features can be used to differentiate between normal and diseased nodes

WSVA8-0113

DIAGNOSTIC IMAGING

MORPHOMETRIC MEASUREMENTS OF THE FOURTH VENTRICLE IN CANINE BRAIN: IN VIVO MRI STUDY

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INTRODUCTION

Although the abnormalities of the 4th ventricle are highly described using MRI technique in dogs, the normal dimensions are still not well defined.

OBJECTIVES

Defining the normal shape and area of the 4th ventricle, establish a new angle in normal canine brains and finding out the effect of head phenotype and body-weight on these parameters.

METHODS

This work was achieved in the Small Animal Hospital/ Glasgow University in 2014. 32 dogs of different breeds were included had age range (1.1- 11.1) years and body-weight (7 -42) kg. The area of the 4th ventricle defined on midline sagittal plane dorsally by the base of the cerebellum, ventrally by the brainstem, anteriorly by the mesencephalic aqueduct and by the central canal of the spinal cord caudally; its length was defined as the longest line of that area. The area of the cranial cavity was also measured. An angle was defined for the first time and named as the 4th ventricle angle.

RESULTS

The 4th ventricle was divided into three parts: rostral, middle and caudal parts in which the rostral and caudal parts had a narrow path like while, the middle has wider area. Furthermore, the rostral and caudal parts were characterized by lower signal intensity comparing to the middle part.

CONCLUSIONS

Normal shape and dimensions of the 4th ventricle can be well delineated using midline sagittal plane of T2 weighted MRI and the signal intensity of its parts is well demarcated. Finally, the created angle appeared to be in correlation to the head phenotype.

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WSVA8-0033

DIAGNOSTIC IMAGING

TRANSTHORACIC ULTRASOUND ELASTOGRAPHY IN SUBPLEURAL PULMONARY LESIONS: A PRELIMINARY STUDY IN CADAVER MODELS

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INTRODUCTION

Elastography is currently used in various organs including the thyroid, breast, liver, prostate and lymph nodes. Nonetheless, few studies have investigated the application of ultrasound elastography in lungs in human and it is not yet applied in dogs.

OBJECTIVES

The purpose of this study was to investigate the feasibility and application of transthoracic ultrasound elastography in pulmonary lesions in dogs.

METHODS

Thirteen cadavers were prospectively obtained from dogs scheduled to undergo euthanasia. A radiofrequency (RF) ablation was utilized to create thermal lesions in cadaver lung. Before and after RF ablation, strain ratios were obtained. The strain index, defined as the muscle to lesion strain ratio (B/A ratio), was calculated automatically by the software program in the ultrasound unit.

RESULTS

The average strain values of dogs with latissimus dorsi muscle(LDM), intercostal muscle (ICM), and pulmonary lesion were 1.80 ± 0.17, 0.52 ± 0.1, and 0.43 ± 0.09, respectively, whereas the strain value of the lung was 0.01 ± 0.001. There were statistically significant differences in tissue hardness. The strain ratio of LDM/lung was significantly different from that of LDM/lesion (180.27 ± 16.83 vs. 4.17 ± 0.94, $p < 0.001$). The strain ratio of ICM/lung was significantly different from that of ICM/lesion (51.88 ± 9.52 vs. 1.15 ± 0.25, $p < 0.001$).

CONCLUSIONS

The elasticity of lung tissue affected by RF ablation and normal lung is quantitatively reflected by strain ratio obtained with ultrasound. This study provides basic information for strain values and strain ratios for the pulmonary lesion in dogs.

WSVA8-0133

DIAGNOSTIC IMAGING

RADIOGRAPHIC ASSESSMENT AND TREATMENT OF PERIODONTAL DISEASE IN A YORKSHIRE TERRIER DOG

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INTRODUCTION

Periodontal disease is a threatening for dog's life. It can be treated by applying certain surgery technique along with radiography diagnostic assistance.

OBJECTIVES

This case study is aimed to understand the radiographic changes and periodontal disease treatment that was occured in a Yorkshire Terrier dog.

METHODS

As a first step, periodontal disease treatment was done by physical examination of the dog's teeth condition which then recorded into the teeth diagram. The radiography was perfomed by using parallel technique to premolar and molar mandibula teeth of the dog.

RESULTS

As a result, the image has shown some decrease in density of the area around molar number 308 whose plaque index, gingivitis, and furcation higher than number 306 that has lower index. Besides that, there is 0.3 mm depth sulcus gingiva in tooth number 308. Dog's teeth extraction was applied to all incisor mandibula teeth.

CONCLUSIONS

The case study showed that radiography can be used as diagnostic tool in periodontal disease treatment. Teeth mobility is also a factor when determining which teeth to extract instead of plaque, gingivitis, and furcation index.

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WSVA8-0044

DIAGNOSTIC IMAGING

ULTRASONOGRAPHIC ELASTOGRAPHY OF RENAL PARENCHYMA IN CANINE WITH CHRONIC KIDNEY DISEASE

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INTRODUCTION

Chronic kidney disease (CKD) is a common disease in dogs that normally progresses to end-stage renal disease (ESRD). During disease progression, the decrease in renal functions is concurrently presented with the fibrotic processes. As a result, losing of renal parenchymal elasticity would be found. At present, ultrasonographic elastography is an advance, advisable and noninvasive technique for tissue elasticity or stiffness evaluation. Assessment through the Young's modulus E (YM) within an interested tissue will provide the tissue stiffness-quantitative measurement

OBJECTIVES

According to the less information of canine CKD elasticity observed through ultrasonographic elastography, the aims of this study were to compare the tissue stiffness between the normal and CKD dogs. In addition, the correlation between the YM value and plasma creatinine (Cr) was elucidated

METHODS

Among 12 canine patients (6 dogs for either normal and CKD group), the mean age of both groups was matched ($P = 0.584$) and the mean body weight of both groups was not significantly difference ($P = 0.372$)

RESULTS

The result showed that the location of kidney was not effected to the YM value in either normal ($P = 0.24$) and CKD group ($P = 0.42$). Interestingly, the CKD dogs had significantly higher YM value in renal parenchyma than those of the normal group ($P < 0.01$). Furthermore, YM value was significantly correlated with the plasma Cr ($r^2 = 0.734$, $P < 0.01$).

CONCLUSIONS

In conclusion, ultrasonographic elastography acts as a promising, noninvasive method to evaluate the renal parenchymal tissue stiffness in canine CKD.

WSVA8-0071

DERMATOLOGY

THE PREVELANCE OF THE FELINE FUR MITE, LYNXACARUS RADOVSKYI ON SINGAPORE CATS

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INTRODUCTION

Lynxacarus radovskyi is a relatively uncommon ectoparasite of cats compared to *Otodectes cynotis*, *Ctenocephalides felis felis* and *Felicola subrotratus*. Despite being first identified on Hawaiian cats in the mid 70's, and its subsequent incident report from Brazil, Malaysia, St. Kitts and Nevis and Australia, very little is known about this mite. (IMAGE 1)

OBJECTIVES

To estimate the prevalence of *Lynxacarus radovskyi* in Singapore and to determine if it is also the most common ectoparasite on Singapore cats

METHODS

Owners' written consent was obtained before a general physical examination, two ear swabs and four separate hair plucks were obtained from the patient. Samples were evaluated microscopically for the presence of flea dirt, fleas, lice, fur mites and ear mites. Result were then tabulated according to age specific groups. The study began from the 25th January 2018 till the 22nd February 2018

RESULTS

A total of 75 cats of which 39 of them males, 36 females were included in this study where 23 of 75 (30.7%) were infested with *Lynxacarus radovskyi*. The highest prevalence was recorded from the mature (7-10 years) age group at 54.5% followed by seniors (11-14 years) at 45.5% and kitten (birth to 6 months) at 31.3%. *Otodectes cynotis* was found in 6 (8%) and *Ctenocephalides felis* in 1 (1.3%), mainly from the kitten age group

CONCLUSIONS

Lynxacarus radovskyii is the most common ectoparasite on cats in Singapore with an estimated prevalence of 30.7%, followed by *Otodectes cynotis* (8%) and *Ctenocephalides felis felis* (1.3%)

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WSVA8-0135

DERMATOLOGY

VETERINARY SATISFACTION TRIAL WITH INJECTABLE CEFOVECIN SODIUM IN DOGS AS TREATMENT FOR SKIN INFECTIONS INCLUDING SUPERFICIAL PYODERMA, WOUNDS, AND ABSCESSES.

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INTRODUCTION

Cefovecin sodium is an injectable cephalosporin antibiotic, indicated for use as a treatment for susceptible skin infections including superficial pyoderma, wounds, and abscesses in dogs, provides clinical efficacy for 14 days, can be repeated up to one time, and is newly available for clinical practice in India.

OBJECTIVES

The objective of this study was to evaluate veterinary satisfaction based on clinical assessment of response to treatment with cefovecin sodium in client-owned dogs.

METHODS

107 dogs of various breeds, ranging in age from 2 months to 11 years, in 25 different veterinary practices in India representing all regions of the country were administered 8 mg/kg cefovecin sodium subcutaneously for skin infections The date of examination/treatment, signalment and appearance of the dog, location of lesion(s), tentative diagnosis, and clinical reassessment/outcome were recorded The veterinarian was also asked to provide visualization of the outcome through high image resolution digital photographs.

RESULTS

Outcomes were as follows: 90 cases classified as either excellent (5), good (6), or satisfying (79) and 8 classified as either indifferent (5) or unsatisfied (3); an outcome was not received for 9 cases. 10 dogs (9.35%) received a second injection.

CONCLUSIONS

The overall satisfaction rate in this sample of dogs from India was 84.11% (90/107), calculated by pooling results from the outcomes of excellent, good, and satisfying. This study demonstrates the usefulness of cefovecin sodium as a treatment for skin infections including superficial pyoderma, wounds, and abscesses in dogs, as assessed by clinical response through the attending veterinarian.
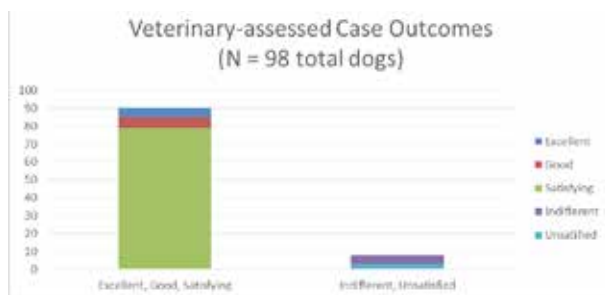
DERMATOLOGY

VETERINARY SATISFACTION TRIAL WITH INJECTABLE CEFOVECIN SODIUM IN CATS AS TREATMENT FOR SKIN INFECTIONS INCLUDING WOUNDS AND ABSCESSES.

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INTRODUCTION

Cefovecin sodium is an injectable cephalosporin antibiotic, indicated for use as a treatment for susceptible skin infections including wounds and abscesses in cats, provides clinical efficacy for 14 days in a single, one-time administration, and is newly available for clinical practice in India.

OBJECTIVES

The objective of this study was to evaluate veterinary satisfaction based on clinical assessment of response to treatment with cefovecin sodium in client-owned cats.

METHODS

22 cats of various breeds, ranging in age from 5 months to 4 years, in 10 different veterinary practices in India representing all regions of the country were administered 8 mg/kg cefovecin sodium subcutaneously as therapy for skin infections including wounds and abscesses resulting from dog, cat, and snake bites, amongst other causes The date of examination/treatment, signalment and appearance of the cat, location of lesion(s), tentative diagnosis, and clinical reassessment/outcome were recorded The veterinarian was also asked to provide visualization of the outcome through high image resolution digital photographs.

RESULTS

Outcomes were as follows: 22 cases classified as either excellent (2) or satisfying (20).

Veterinary-assessed Case Outcomes
(N = 22 total cats)

CONCLUSIONS

The overall satisfaction rate in this sample of cats from India was 100.00% (22/22), calculated by pooling results from the outcomes of excellent and satisfying; no case was assessed as unsatisfying or indifferent. This study demonstrates the usefulness of cefovecin sodium as a treatment for skin infections including wounds and abscesses in cats, as assessed by clinical response through the attending veterinarian.
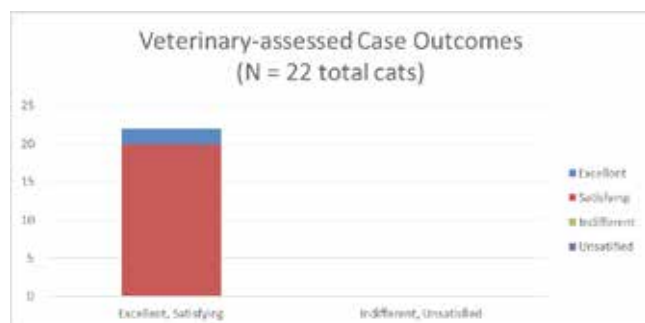
WSVA8-0086

ENDOCRINOLOGY

NON-FUNCTIONAL ADRENOMEDULLARY TUMOR ASSOCIATED THROMBOEMBOLISM

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INTRODUCTION

Malignant adrenal gland tumour can be associated with the life threatening thromboembolism.

OBJECTIVES

To demonstrate the cause of non-cardiogenic pulmonary edema.

METHODS

A 12 year-old, Siberian Husky was referred to further diagnosis according to abdominal organ enlargement and the abdominal breathing Colour Doppler ultrasonography was carried out.

RESULTS

Both sides adrenal gland enlargement was revealed with incident of the posterior vena cava invasion by the right. Chest radiographic finding demonstrated alveolar pattern without fluid accumulation inside pleural space at the left lung lobe and enhancement of radiographic opacity at peri-hilar area. The IRIS stage II azotemia with polyuria polydipsia was displayed on physical examination. No evident of hypernatremia, hypokalemia, hypertension and tachycardia was detected. ACTH stimulation test and low dose dexamethasone suppression test rejected to the disease. Oxygen therapy, antiplatelet, diuretic were administered to correct pulmonary edema and thromboembolism. Unfortunately, the dog was not very well responded and necropsy was carried out. The architecture of the right adrenal medulla was devastated by the tumour mass. Histopathological description of caudal vena cava was composed of neoplastic polygonal-shaped with slightly indistinct cells borders arranged in cords, small packets and nested patterns. A thin ribbon of adrenal cortices was seen and compressed by neoplastic cells. The left adrenal gland was intact.

CONCLUSIONS

In summary, the cause of death is thromboembolism and regional angioinvasion induced by malignant pheochromocytoma.

WSVA8-0041

EXOTICS

IVERMECTIN TOXICOSIS IN CHELONIANS

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INTRODUCTION

Ivermectin has been known to cause serious toxicosis in chelonians. Although the use of this drug is strictly contraindicated, there is always risk of accidental administration.

OBJECTIVES

The objective of this case report is to recognize the symptom and to provide the necessary medical care.

METHODS

A group of 19 tortoises and turtles were brought to the clinic suffering from side effects following ivermectin injections. The tortoise includes aldabras, sulcatas, emys, red foot and Indian star. The turtle includes common snapping, fly rivers, red ear sliders, biukus, and snake head. They were injected with ivermectin in a previous week with the dose of 0.1 cc SC (Ivomect ®). All tortoises and turtles did not eat and showed symptom of weakness. Some of them with sunken eyes, and some were very lethargic and even paralized. All of them were immediately treated with ICe fluids (10 ml/kg bw). Activated charcoal were used to neutralize the ivermectin effect and was given peroral with a dose of 1 g/kg bw. Nutritional support was given by soft tubes and vitamin B12 with the dose of 0.05 mg/kg bw IM was added to stimulate appetite. They were kept warm using UVA lamps. The aquatic turtles were kept in shallow water.

RESULTS

The recovery time varied as turtles needed longer time to recover. Only one fly river turtle died during treatment, while the other 18 recovered fully.

CONCLUSIONS

Despite guarded prognosis related to ivermectin toxicosis in chelonians, good supportive care can still yield positive results.

WSVA8-0186

EXOTICS

ELECTROCARDIOGRAPHICS PARAMETERS OF CAPTIVE CHELONOIDIS SP.

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P.T. Chalfun Guimarães-Okamoto[1],
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INTRODUCTION

The electrocardiogram (ECG) can be very useful for evaluation and monitoring in wild animals. The literature is scarce about electrocardiographic data in *Chelonoidis*.

OBJECTIVES

This study aimed to evaluate the EKG of 27 healthy *Chelonoidis* sp., kept in captivity, without the use of pharmacological restraint.

METHODS

Eight females and 19 males were evaluated. The data was assessed according to gender (males and females) and body weight (animals up to 5 kg - G1 and animals above 5 kg - G2). The EKG was performed with the electrodes positioned similarly to the mammalian EKGs. The variables evaluated were: heart rate (HR); heart rhythm; duration and amplitude of the EKG waves and intervals.

RESULTS

Alligator-type electrodes, connected to the thoracic and pelvic limbs, were effective in capturing cardiac electrical impulses. Morphology of the recorded waves was similar to mammals. In G1 and G2 the sinus rhythm was predominant. HR in G1 was higher than G2. In wave morphology, SV wave was observed in two animals from G2. The P-wave showed positive deflection in 78.57% and negative in 21.43% of G1. All of the animals in G2 presented positive P-wave. QRS complex in G1 and G2 showed a positive R-wave and the absence of Q and S waves were observed in 100% of the animals, and the duration was superior in G2.

CONCLUSIONS

Body weight influenced heart rate, duration of the QRS complex, and QT and RR intervals. It is very important to consider the weight of the animal in EKC interpretation in *Chelonoidis* sp.

WSVA8-0103

EXOTICS

EVALUATION OF HIP JOINT LAXITY IN CRAB-EATING FOXES

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INTRODUCTION

Like canids, crab-eating foxes may probably be predisposed to similar orthopedic diseases of domestic dogs, such as hip dysplasia. However, for the adequate hip dysplasia diagnosis in wild animals, the normality characteristics of each species must be determined.

OBJECTIVES

This study aimed to assess radiographic and computed tomographic (CT) values of hip joint laxity in healthy crab-eating foxes.

METHODS

Fifteen intact crab-eating foxes, eight males and seven females, ages 1 to 5 and mean body mass of 6.66 kg were used. Norberg angle (NA) was calculated from ventrodorsal hip-extended radiographs. To calculate the dorsolateral subluxation (DLS) score, the center distance (CD) index, the lateral center edge angle (LCEA), and the dorsal acetabular rim angle (DARA), measurements obtained from transverse CT images were used.

RESULTS

No statistically significant differences were observed between the right and left sides in the radiographic and tomographic parameters. The mean NA was 107.57°. The mean DLS score, the CD index, the LCEA, and the DARA were 60.79%, 0.16, 98.25° and 13.47°, respectively.

CONCLUSIONS

The data obtained are helpful in characterizing mean values of the hip joint in healthy crab-eating foxes, and can contribute to the knowledge of the species.

WSVA8-0183

EXOTICS

MEDICAL AND SURGICAL DISORDERS IN RABBITS: A RETROSPECTIVE STUDY

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INTRODUCTION

In several countries, the rabbits have been considered the third most popular companion animal compared to dogs and cats, which requires a greater attention to the particularities of the species. These animals are subject to a number of diseases, which need to be properly identified and treated.

OBJECTIVES

The aim of this study was to determine medical and surgical disorders in rabbits examined in a reference center for a period of 6 years.

METHODS

Data concerning the animal identification (sex, age, body weight), reason to bring the rabbit to the clinic, and characterization of the disorder (medical or surgical) according to the system were evaluated.

RESULTS

A total of 249 rabbits was examined, comprised of 139 males, 83 females, and 27 without identifying information. The age varied from 3 days to 9.7 months. Males weighed from 360 grams to 4.19 kg (mean 1.67 kg), and females weighed from 486 grams to 4.36 kg (mean 1.68 kg). The major occurrences involved the integumentary system (25%), digestive (22%) and musculoskeletal (11%) systems. Parasitism by mites and abscesses were the most common skin problems. The disorders more frequent in digestive system were tooth overgrowth and gastrointestinal stasis. Hind limb fractures were the most prevalent of musculoskeletal conditions. Spaying and castration were the most common surgical procedures, followed by osteosynthesis.

CONCLUSIONS

In conclusion, the rabbits admitted had parasitism by mites and dental problems as more frequent disorders, and among the surgical procedures realized the most common was the neutering.

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EXOTICS

A NEW TECHNIQUE BY A SMALL VERTICAL INCISION FOR TREATING ABERRANT OVERGROWTH OF CONJUNCTIVA IN RABBIT

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INTRODUCTION

The aberrant overgrowth of conjunctiva is a rare condition in rabbits The conjunctiva plica gradually extend and occasionally covers the central cornea.

When surgically resection of overgrown conjunctiva is performed, the condition is often recurs easily.

Therefore, some methods of fixing the overgrown conjunctiva to the sclera have been reported. Then topical corticoid or cyclosporine is used after surgical fixation. On the other hand, our method is very simple and has rare recurrence.

OBJECTIVES

We hypothesized that abnormal repair function caused the aberrant overgrowth of conjunctiva by inflammatory stimulation. The blocking and suppression for this phenomenon was applied to 2 rabbits

METHODS

1. Lidcaine solution was administered to the ocular surface as a surface anesthesia.

2. Some incisions were made to frap-like elongated conjunctiva. They were performed radially from the margin of the frap-like conjunctiva to the corneal limbus. Then the elongated conjunctiva was pull back close to the corneal limbus depending on the forth that the flap attempts to contract.

3. After the treatment, prednisolone and antibiotics were administered to control the inflammation and to prevent infection.
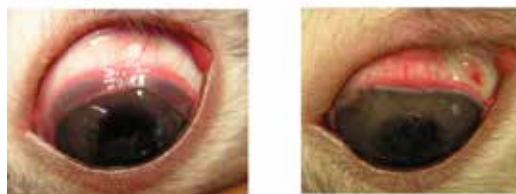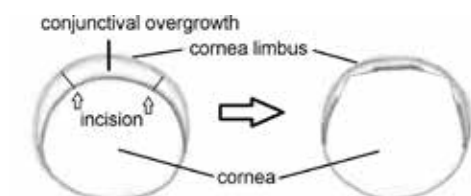
RESULTS

Case1: One small incision and medication was performed to the conjunctiva on day 5 of the disease. Rabbit recovered almost normally on day 12 of the disease.

Case2: Two small incision and medication were performed. There is no recurrence after 2 months.

CONCLUSIONS

After this treatment, a slight residual was observed but no recurrence was noted.This treatment can be done with a simple technique, and general anesthesia is not necessary. It is considered to be a valuable method to be done first.

WSVA8-0151

FELINE MEDICINE

DETECTION OF LEPTOSPIRAL INFECTION IN SHELTERED CATS IN MALAYSIA.

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INTRODUCTION

Leptospirosis is one of the most widespread zoonotic diseases in both human and animals. Extensive studies have been conducted to investigate *Leptospira* in human and different animals' species, including cats. Although prevalence of leptospiral infection in cats had been reported in many countries, but reliable information about cat's leptospirosis in Malaysia seems to be insufficient.

OBJECTIVES

The aim of this study was to detect the leptospiral infection in sheltered cats in Malaysia and to determine the predominant serovars among cats.

METHODS

Seventy sheltered cats from four shelters were recruited in this study. Upon physical examination, cats were apparently healthy, but some cats (around 20 cats) showed mild signs of feline upper respiratory disease. Serological test for antibodies detection by using Microscopic Agglutination Test (MAT) was performed. Sera were tested against 20 pathogenic serovars, namely; Australis, Autumnalis, Canicola, Icterohaemorrhagiae, Grippotyphosa, Pomona, Ballum, Copenhageni, Javanica, Bataviae, Hebdomadis, Hardjobovis, Hardjo-prajitno, Lai, Tarassovi, Pyrogenes, Celledoni, Cynopteri, Djasiman, Malaysia Bejo-Iso9 and one non-pathogenic *L. biflexa* Patoc 1 strain.

RESULTS

Based on the cut-off point of 1:100, 14.29% of the cats (*n*=10/70) were tested seropositive to at least one serovar. Only one sera had co-agglutinations to both Javanica (1:400) and Bataviae (1:100). The predominant serovars found were Bataviae (*n*=4/70), Ballum (*n*=4/70) and Javanica (*n*=3/70), with titres ranged between 1:100 to 1:400.

CONCLUSIONS

This result shows that cats might remain clinically healthy despite seropositive against leptospiral infection. More studies are warranted in order to investigate the role of cats in the transmission of the leptospirosis.

WSVA8-0082

FELINE MEDICINE

RESULTS OF IN-CLINIC RAPID TESTS FOR FELV ANTIGEN VARY SIGNIFICANTLY

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INTRODUCTION

Feline leukemia virus (FeLV) is a highly contagious virus that can cause life-threatening diseases in cats if infected. In practice, rapid antigen tests are commonly used, and often it is the only mechanism to identify infected cats. The accuracy of rapid tests is of paramount importance.

OBJECTIVES

To compare the performance of three in-clinic rapid tests for FeLV antigen: SNAP® Feline Triple® (IDEXX), Anigen® FIV/FeLV test (BioNote), Speed Duo® FeLV/FIV test (Virbac).

METHODS

Serum or plasma samples were collected from those submitted for feline retroviral testing at IDEXX Reference Laboratories. All samples were screened using an independent laboratory ELISA assay, ViraCHEK® FeLV (Zoetis), for the presence or absence of FeLV antigen. A total of 84 positive and 101 negative samples were included in this study. Samples were blinded and randomized for testing with in-clinic rapid tests, according to manufacturers' instructions.

RESULTS

Compared to ViraCHEK, percent agreements for positive and negative samples were 97.6%/100% (SNAP), 66.7%/97.0% (Anigen), and 51.2%/99.0% (Speed Duo).

CONCLUSIONS

These findings suggest a lower sensitivity for the Anigen and Speed Duo tests, based on the ViraCHEK results Consistent with a recently published study, this study also found a lower specificity for the Anigen test, which would indicate a proportion of the Anigen test positive results could be false positive given very low prevalence for FeLV Both false negative and false positive test results for FeLV could lead to significant clinical issues.

FELINE MEDICINE

SUITABILITY OF COMMERCIAL FELV NUCLEIC ACID DETECTION KITS FOR SMALL LAB ENVIRONMENTS

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INTRODUCTION

Feline leukemia retrovirus (FeLV) causes a chronic infectious disease manifested by profound anemia, malignancies, and immunosuppression. If present in multi-cat environments, FeLV has an impact on survival and quality-of-life, therefore accurate and early detection is imperative to prevent the virus from spreading to other cats.

OBJECTIVES

Comparison of two commercially available molecular kits for the detection of FeLV RNA and DNA using several well-known nucleic acid extraction kits.

METHODS

Molecular kits, Biogal PCRun® and PrimerDesign were tested and compared to each other to define the sensitivity, specificity, and suitability for use in small laboratory settings. DNA and/or RNA (N=121) were purified from three different retrospective sample archives (buffy coat and plasma) collected in Switzerland (Group 1), Portugal (Group 2) and USA (Group 3) employing extraction kits from 2 different companies (Qiagen and Zymo). Final results were compared to the Cq values or SNAP FeLV/FIV generated by the laboratory source.

RESULTS

Group 1: PCRun results correlated better with TaqMan (sensitivity 100% / specificity 84%) than with SNAP.

Group 2 Extraction methods affected final results. Zymo preps generated DNA with lower inhibition of the PCRun® reactions (Sensitivity: Zymo 92-97%; Qiagen 74%). Zymo produced poorer quality RNA extracts compared to Qiagen (Sensitivity 89.5-91.5%).

Group 3: Results obtained with PCRun® were 100% complementary to the TaqMan and matched closely to the viremic stages of the subjects.

CONCLUSIONS

PCRun® is a point-of-care test, while PrimerDesign is a Taqman RealTime PCR The hands-on application and accuracy of PCRun® makes it highly suitable for screening of FeLV in laboratories with minimal equipment.

FELINE MEDICINE

CAUSES OF DEATH OF DOMESTIC CATS IN A VETERINARY HOSPITAL OF MINAS GERAIS STATE, BRAZIL: A RETROSPECTIVE STUDY

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INTRODUCTION

Cats play an important role as pets in the present society. The knowledge about their main diseases and causes of death is fundamental to prevent various pathologies. However, when dealing with domestic cats there are very few researches in Veterinary Medicine.

OBJECTIVES

We identified the causes of death in cats through a retrospective study in the Animal Pathology Laboratory in the Federal University of Uberlândia (UFU) (Brazil), in a period of 36 years, in which 350 cat necropsies were conducted.

METHODS

Necropsy medical records of cats were selected from the archives of Animal Pathology Laboratory (UFU) (Brazil, MinasGerais State). Epidemiological data as gender, age, and breed were collected. The necroscopic reports were evaluated and the cause of death recorded. The diagnostic method used was anatomopathological examination, considering all the complementary postmortem examinations performed for definitive diagnosis (histopathological, toxicological, and immunohistochemical). Only definitive diagnosis was considered, and when elucidating the cause of death was not possible, the diagnosis was considered inconclusive.

RESULTS

The most frequent causes of death were digestive system diseases (13.71%), and among them, feline hepatic lipidosis was the most frequent etiology (18.75%). The second most affected system was the respiratory (10.86%) and acute pneumonia, pulmonary edema, and diaphragmatic hernia were the main diseases in this system. Other decease causes were rabies, squamous cell carcinoma, fibrous osteodystrophy, fractures and traumatism, hypovolemic shock, and feline lower urinary tract disease.

CONCLUSIONS

In conclusion, the digestive system was the system most frequently related to the death of cats, and feline hepatic lipidosis was the most frequent disease.

WSVA8-0100

FELINE MEDICINE

PREVALENCE OF GASTROINTESTINAL DISEASES IN CATS FROM BOTUCATU CITY, SÃO PAULO STATE, BRAZIL.

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INTRODUCTION

Gastrointestinal (GI) diseases involving the alimentary tract and hepatobiliary system are common in cats. Domestic cats present specific anatomical, physiological, nutritional and behavioral characteristics related to the gastrointestinal tract, which may influence the prevalence of diseases of the digestive system. Fundamental efforts to diagnose GI disorders should always be directed toward localizing disease to a particular segment and determining a cause.

OBJECTIVES

The aim of this study was to evaluate the prevalence of digestive system diseases in domestic cats from Botucatu city, São Paulo (SP) state, Brazil.

METHODS

Were used the medical records of cats with digestive disorders presented at Veterinary Teaching Hospital (FMVZ-UNESP, Botucatu, SP, Brazil) between 2013 to 2016.

RESULTS

The total number of animals evaluated in this period was 1047 cats. Of these, 259 cats with clinical signs consistent gastrointestinal diseases were included in this study (24.7%). Diagnoses were gastrointestinal parasites (27.8%); hepatic lipidosis (22%); chronic *gingivostomatitis* (8.9%); acute gastroenteritis (7.7%); acute gastritis (6.2 %); inflamatory bowel disease (3.9 %); dietary indiscretion (3.1 %); Pancreatitis (2.70%); constipation (2.7%); chronic liver disease (1.2%); Megacolon (0.8%); intestinal foreign bodies (0.8%); chronic gastritis (0.8%); Colitis = 2 (0.8%); hepatic neoplasms of unknown etiology (0.8%); feline triad (0.4%); acute hepatitis (toxic) (0.4%); portosystemic shunt (0.4%); *Hepatic cysts* (0.4%); *alimentary lymphoma* (0.4%); and *intestinal adenocarcinoma* (0.4%).

CONCLUSIONS

The most prevalent GI diseases in cats were gastrointestinal parasites and hepatic lipidosis. To the best of author´s knowledge, this is the first survey study on feline digestive disorders in Botucatu city, SP, Brazil.

WSVA8-0112

FELINE MEDICINE

PLASMA LIPID PROFILE IN OBESE CATS.

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INTRODUCTION

Obesity is the most common nutritional disorder in companion animals. It is related to several comorbidities and reduced lifespan. The literature on the effects of obesity on the lipid profile in cats is still limited.

OBJECTIVES

The aim of this study was to evaluate and compare the lipid profile in obese and control (lean) cats.

METHODS

Obesity was determined based on the body condition score (BCS), with the nine-point scale. Were evaluated 40 cats, 20 obese (BCS 8-9) and 20 control cats (BCS 5). Blood was collected after a 12 hour fast, via jugular venipuncture. Serum samples were analysed for total cholesterol (TC), triglycerides (TG), high density lipoprotein cholesterol (HDLc) and low density lipoprotein cholesterol (LDLc) levels. Automated enzymatic colorimetric methods were used for serum TC, triglyceride and HDLc measurements. LDLc levels were calculated using the formula: LDLc = TC − (HDLc + TG/5) (Friedewald et al., 1972).

RESULTS

In the obese group, 50% and 25% showed an increase in TC and triglycerides, respectively. In the control group, elevations in TC and triglycerides occurred in 50% and 5% of the dogs. There were no statistical differences between the two groups for any of the parameters evaluated. Mean cholesterol, HDLc, LDLc, and triglycerides were: 138.9±37.3, 94.6±16.8, 28.0±28.8 and 117.8±118.8 for obese cats; 132.9±35.0, 93.7±17.4, 25.3±26.8 and 75.7±28.2 for lean cats.

CONCLUSIONS

In obese cats, dyslipidemia was mild but frequent and it should always be evaluated, with emphasis in monitoring of serum cholesterol levels. More studies are needed to evaluate the lipid profile in obese cats.

WSVA8-0122

FELINE MEDICINE

PREVALENCE OF DISORDERS RECORDED IN CATS (FELIS SILVESTRIS CATUS) ATTENDED IN VETERINARY SCHOOL HOSPITAL (FMVZ – UNESP, BOTUCATU), SÃO PAULO, BRAZIL

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INTRODUCTION

The knowledge of prevalence of cat´s disorders is essential to improve understanding and supporting clinicians when listing differential diagnoses and health control strategies in specific geographical location. Information concerning to demographics of cats disorders in Brazil is lack in the literature.

OBJECTIVES

Assess the prevalence of the most common disorders recorded in cats attended in veterinary school hospital (HV), School of Veterinary Medicine and Animal Science (FMVZ, Botucatu), University Estadual Paulista (UNESP), São Paulo, Brazil.

METHODS

Were used clinical records between 2011 and 2014, and the cats came from Botucatu city, São Paulo and their regions.

RESULTS

One thousand and forty-eight (1,048) cats were diagnosed with clinical disorders by two veterinarian, and the most prevalent disorders were urinary diseases (n = 441; prevalence, 42.08%), digestive diseases (n = 302; prevalence, 28.82%), respiratory diseases (n=106; prevalence 10.11%), dermatological disorders (n=72; prevalence 6.87%), intoxications (n=42; prevalence 4.01%), cardiovascular diseases (n=35; prevalence 3.34%), endocrine disorders (n=15; prevalence 1.43%), and neurological disorders (n=9; prevalence 0,86%). The most prevalent disorder groups recorded was mixed disorders (respiratory/digestive) (n=16; prevalence 1.53%), digestive/dermatological disorder and digestive/urinary disorders (n=4; prevalence 0.38%), and cardiovascular/urinary disorders (n=2; prevalence 0.19%).

CONCLUSIONS

Each of these disorders can be associated with another disease and the most of the diagnosis was performed by clinical signs and laboratory examination as hemogram, blood serum biochemistry and image diagnoses. Veterinarians could use these results to focus their differential diagnoses, health control strategies, prophylactic efforts and demographic and clinical feline studies towards the most prevalent feline disorders in São Paulo, Brazil.

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WSVA8-0185

FELINE MEDICINE

BIOCHEMICAL AND ELECTROCARDIOGRAPHIC PROFILE IN CATS WITH OBSTRUCTIVE FELINE LOWER URINARY TRACT DISEASE

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INTRODUCTION

Urethral obstruction is a common condition in domestic male cats. Many problems can occur due to the interruption of the urinary flow, such as acid-base and electrolytic changes that results in abnormalities in the myocardial cells potential action and, therefore, in electrocardiogram.

OBJECTIVES

The aim of this study was to characterize the electrocardiographic, biochemical and hematological changes of cats with urethral obstruction.

METHODS

Eighteen male cats with first urethral obstruction were evaluated in the clinical routine of Veterinary Medicine and Animal Science School – UNESP, Botucatu, SP.

RESULTS

The most significant electrolytic change was hyperkalemia, in 76.5% of the animals. 17.7% was hypokalemic and 5.9% with normal potassium levels (5.4±1.7). The cats were azotemic, with serum urea (267.8±181.9) and creatinine (7.9±6.0) above reference range in 94.7% and 79% of the cats, respectively. It was observed as well hypoalbuminemia in 57.9 % of cats (2.5±0.3). There was a marked leukocytosis in 79% of the cats (32147±16782) characterized by neutrophilia (29700±16444). In electrocardiogram 81.3% showed sinus rhythm, however 6.25% presented sinus bradycardia and 12.5% sinus tachycardia. Other relevant findings were widened QRS complex (87.5% of the animals) and prolonged P-wave duration (62.3%) and Q-T interval (50%). There were also abnormalities such as ventricular extrasystole (6.3%), atrial still, right bundle branch block and fascicular block (12.5%). The cats which presented serum potassium levels above 7.0 (22.2%) had as the most important irregularities fascicular block and atrial still.

CONCLUSIONS

It is important to monitor the cardiac rhythm in cats with urethral obstruction, since electrocardiographic changes are relatively frequent.

WSVA8-0163

FELINE MEDICINE

PHYLOGENETIC CHARACTERIZATION OF FELINE RETROVIRUSES IN HUNGARY

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INTRODUCTION

Feline retroviral infections are common in domestic cats causing AIDS-like diseases like Feline Immunodeficiency Virus (FIV), or neoplastic diseases like Feline Leukemia Virus (FeLV).

OBJECTIVES

Our aim was to characterize these retroviral infections among domestic cats, examine the prevalence in Hungary, and to make a phylogenetic analysis from the discovered strains.

METHODS

Prevalence data were counted after examination of EDTA-anticoagulated whole blood samples of client-owned cats in Hungary. We carried out ELISA snap tests and polymerase chain reaction (PCR) on each specimen. Sequence analysis was done only on PCR-positive strains. In case of both viruses, amplification of *pol* and *env* sequences was performed. Individual sequence text files in FASTA-format were created after alignement of both unidirectional electropherograms. Multiple sequence alignement were performed with ClustalW software, accepting the default parameters. Phylogenetic trees were created with BioEdit software with maximum likelihood-based phylogenetic analysis.

RESULTS

In this survey we collected more than 300 blood specimens and phylogenetic analyses were carried out on those contained FIV and/or FeLV. Data of *pol* and *env* sequences showed, that Hungary has mostly subtype A strains of FIV, which correlates to data provided by surrounding countries.

CONCLUSIONS

This study filled a gap of European feline retroviral surveys, providing information of Hungarian distribution of FIV and FeLV strains among domestic cats. The data correlates with phylogenetics of surrounding countries, showing mainly subtype A FIV infections.

WSVA8-0051

PREVALENCE OF GASTROINTESTINAL PROTOZOA IN PET AND STRAY CATS IN THE KLANG VALLEY, MALAYSIA

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INTRODUCTION

The gastrointestinal tract of the feline species can harbour various protozoa. Most of these gastrointestinal protozoa cause diarrhea in cats and some are considered zoonotic.

OBJECTIVES

This study was conducted to determine the prevalence of gastrointestinal protozoa in the pet and stray cat population in Klang Valley, Malaysia. In addition, this study aimed to investigate the risk factors associated with gastrointestinal protozoal infection.

METHODS

201 fecal samples were collected using rectal swabs and were kept in lysis buffer for storage. 192 blood samples were collected and placed in plain tubes to obtain serum and stored at -20°C. Fecal samples collected were subjected to nested-PCR to detect *Giardia spp., Tritrichomonas foetus,* and *Toxoplasma gondii.* The simple floatation method was used to identify *Cystoisospora spp.* for 44 samples. Indirect-ELISA was used to detect antibodies against *Toxoplasma gondii* in the blood samples collected.

RESULTS

The prevalence of gastrointestinal protozoa in stray and pet cats respectively were 11% and 5% *(Giardia spp.)*, 56% and 14% *(Tritrichomonas foetus)*, 15% and 6% *(Toxoplasma gondii)*. 52% (23/44) of *Cystoisospora spp.* were found in stray cats. Seroprevalence of *Toxoplasma gondii* was 7.2% (stray cats) and 4.2% (pet cats). Statistical analysis showed no significant association with protozoan infection with sex, breed and management of the cats. Age showed significance where older cats (≥3years) showed higher risk for *Tritrichomonas foetus*.

CONCLUSIONS

Overall, there was a higher prevalence of gastrointestinal protozoa detected in stray cats compared to pet cats. This study also identified that cats older than 3 years of age were at a higher risk for *Tritrichomoans foetus* infection.

WSVA8-0160

CASE SERIES OF CATS WITH SUSPECTED FELINE INFECTIOUS PERITONITIS (FIP) WET FORM IN JAKARTA

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INTRODUCTION

Feline Infectious Peritonitis (FIP) remain as significant and lethal disease in cats. Incidence of this disease in Indonesia is believed increasing nowadays. There were no FIP retrospective studies conducted in Indonesia yet.

OBJECTIVES

The aim of this study was to provide retrospective data of suspected Feline Infectious Peritonitis wet form in Jakarta.

METHODS

between January 2017 and January 2018, 37 cats were presented with peritoneal and/or pleural effusion in Veterinary Clinic Drh. Cucu K. Sajuthi and Assosiate, Jakarta, Indonesia. Diagnosis ofsuspected FIP wet form was established from Rivalta test positive result of the cat's effusion fluidsamples. Medical records of suspected FIP wet form cats were evaluated retrospectively.

RESULTS

Most of the cats with peritoneal and/or pleural effusion, 91.9%, were diagnosed as suspected FIP wetform (34/37). Other 3 cats were diagnosed as cardiopulmonary disease (2 cats) and bacterial infectiondisease (1 cat). Of the suspected FIP wet form, 85.3% were live in multi cat household (29/34) and 64.7% were male cats (22/34). The type of effusion varied between the cats, 73.5% cats had peritonealeffusion only (25/34), 14.7% cats had pleural effusion only (5/34), and 11.8% cats had both peritonealand pleural effusion (4/34). The data also showed that 41.2% suspected FIP wet form cats had otherdisease (14/34).

CONCLUSIONS

There were high number of suspected FIP wet form incidence in Jakarta, most of the cats were live in multi cat household

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WSVA8-0038

GASTROENTEROLOGY

PREVALENCE OF CANINE HEPATIC LESIONS FROM PERCUTANEOUS ULTRASOUND-GUIDE TRU-CUT LIVER BIOPSY IN THAILAND

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INTRODUCTION

The definitive diagnosis of canine hepatobiliary diseases required histopathology. To date, percutaneous ultrasound-guided tru-cut biopsy remains the most common technique to obtain the samples.

OBJECTIVES

The objective of the present study was to survey the prevalence of hepatic lesions of dogs in Thailand.

METHODS

A survey of hepatic lesion in dogs with chronic elevated liver enzyme was conducted in 76 dogs (median age [range]: 10.8 years [1.7 – 16.5 years]; 42 (55.3%) females and 34 (44.7%) males) in Thailand during January 2016- January 2018 using ultrasound-guided Tru-cut liver biopsy. The liver samples were processed for histopathology analysis. The associations between anemia (PCV<35) or thrombocytopenia (platelet< 200,000/cumm) with liver lesions were also evaluated.

RESULTS

Of these 76 dogs, there were 46 (60.5%) small, 21 (27.6%) medium and 9 (11.8%) large breed dogs. The histological diagnosis revealed 41 (53.9%) with hepatitis, 9 (11.8%), 25 (32.9%) with fibrosis, 21 (27.6%) with vacuolar hepatopathy and 8 (10.5%) with steroid hepatopathy. There were 14 (18.4%) dogs with anemia, 14 (18.4%) dogs with thrombocytopenia and 3 (3.9%) dogs with both anemia and thrombocytopenia. Dogs with liver tumors were associated with the presence of anemia (p=0.009). Older dogs (age >8 years) were associated with the presence of thrombocytopenia (p=0.003). There was only one (1.3%) dog with bleeding complication after the liver biopsy.

CONCLUSIONS

In conclusion, percutaneous ultrasound-guided Tru-cut liver biopsy is a safe and non-invasive method to diagnose canine hepatobiliary diseases. Caution should be taken in dogs with anemia and thrombocytopenia due to a possible bleeding complication after the biopsy.

WSVA8-0016

GASTROENTEROLOGY

A SYSTEMIC CANINE PROTOTHECOSIS IN CENTRAL REGION OF THAILAND

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INTRODUCTION

Prototluecosis is a disease caused by achlorophyllic algae. The most common clinical presentation is protracted hemorrhagic enteritis. Colon is the most commonly affected portion but the organisms can affect many organs.

OBJECTIVES

To demonstrate an effectiveness treatment protocol for canine protothecosis

METHODS

A 4-years old, neutered female, Thai Bangkaew dog was referred to Kasetsart University Veterinary Teaching Hospital (KUVTH) with the history of 7 months hematochezia and azotemia. The dog did not response to the previous treatment with long-termed antibiotics. Colonoscopy demonstrated the generalized inflammation and focal hemorrhage from colon to rectum. The cytology showed granulomatous inflammation with possible Prototheca organisms. The treatment protocols were symptomatic treatment and Itraconazole 5 mg/kg orally once daily. However, the dog developed acute blindness, retinal detachment with pyogranulomatous retinitis and neurological signs with disoriented after 3 months of the treatment. Repeated cytology from rectal scraping revealed a large number of Prototheca spp. The treatment regimen then was changed to amphotericin B intravenously 1 mg/kg 2-3 times a week together with fluconazole 5mg/kg orally twice daily. Subcutaneous fluid was also provided and renal profile was monitored regularly.

RESULTS

After 4 months of this treatment protocol, the clinical signs were improved and the number of Prototheca spp. derived from rectal scraping dramatically decreased.

CONCLUSIONS

Systemic Protothecosis can affect many organs and results in death. However, with early diagnosis and appropriate management, the dog may has favorable outcome and increase survival time. Rectal scraping is a valuable tool for early diagnosis of Protothecosis as it is non-invasive technique and inexpensive.

GASTROENTEROLOGY

HEPATOPULMONARY SYNDROME SECONDARY TO PORTAL VEIN THROMBOSIS WITH CHRONIC HEPATITIS IN A DOG

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INTRODUCTION

Hepatopulmonary syndrome (HPS) is a respiratory complication of hepatic disease, which was well recognized in humans and defined by the presence of 1) liver disease, 2) hypoxemia and/or high alveolar-arterial oxygen gradient (AaDO$_2$) and 3) intrapulmonary vasodilatation. To the author's knowledge, there is only one suspected HPS case that was previously documented in a living dog (Kaneko et al. 2016).

OBJECTIVES

This case report describes a case of HPS in a dog, and its post-mortem findings.

METHODS

A nine-year-old Doberman was diagnosed with portal vein thrombosis with chronic active hepatitis and showed hypoxia (PaO$_2$: 77 mmHg) and increased AaDO$_2$(38.8 mmHg; reference range:<15 mmHg) under room air condition. The presence of intrapulmonary vasodilatation was confirmed by agitated saline contrast transthoracic echocardiography (Figure 1). Besides, the absence of congenital cardiac defect was confirmed by transthoracic echocardiography. Based on the findings, we suspected that this dog had HPS. The dog died two years after her first visit, and we were able to performed autopsy.

Figure 1: Agitated saline contrast transthoracic echocardiography Bubbles were founded in the left ventricle.

RESULTS

Pathological examination showed dilation of pulmonary capillaries. Real time-PCR analysis showed 19.3 times higher level of lung endothelial Nitric Oxide Synthase (eNOS) and 2.5 times higher level of heme oxygenase (HMOX), compared to three healthy dogs. eNOS and HMOX are related to the production of nitric oxide and carbon monoxide, which was reported to be involved in the mechanism of pulmonary vasodilatation in human HPS cases and rat's model.

CONCLUSIONS

This is a description of the first confirmed canine HPS case.

WSVA8-0057

GASTROENTEROLOGY

PARTIAL ANALYTICAL VALIDATION OF A NEW IN-CLINIC CPLI TEST (VCHECK CPL®)

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INTRODUCTION

Serum concentration of pancreatic lipase immunoreactivity (cPLI, measured by Spec cPL®, IDEXX Laboratories) is the most reliable diagnostic tool currently available for the diagnosis of pancreatitis in dogs. A new in-clinic cPLI test (VCheck cPL®, BioNote), claims similar performance to Spec cPL. However, no analytical validation data are available for this assay.

OBJECTIVES

To partially analytically validate the VCheck cPL assay.

METHODS

Leftover clinical serum samples were used. VCheck cPL testing (with a V200 analyzer) was performed according to manufacturer's instructions. Linearity was evaluated by diluting 2 samples 3 times. Precision was evaluated by determining intra-assay variability for two samples analyzed 12 times on the same day. Reproducibility was evaluated by calculating inter-assay variability for 6 samples measured 10 times on different days. Samples (n=42) throughout the working range were assessed for correlation with Spec cPL.

RESULTS

Mean ±SD observed/expected ratio for dilutions were 47.6% ±16.1%. Intra-assay variability for the two samples were 23% and 36%. Inter-assay variability for the 6 samples were 27, 32, 37, 44, 46, and 56%. Furthermore, repeated measurements often changed diagnostic bins for the samples tested. Of 42 clinical samples, 34 (81%) returned lower cPL values by VCheck compared to Spec cPL (Figure 1).
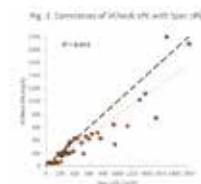
CONCLUSIONS

The new VCheck cPL assay lacked linearity, precision, and reproducibility, suggesting that this assay is not reliable for clinical use. It also frequently provided lower results than Spec cPL, suggesting that reference intervals and cut-off values established for Spec cPL cannot be utilized for the VCheck cPL assay.

WSVA8-0058

GASTROENTEROLOGY

THE EFFECT OF PANCREATIC LIPASE-RELATED PROTEINS ON SERUM LIPASE ACTIVITY AS MEASURED BY DGGR-BASED ASSAYS

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INTRODUCTION

Serum lipase activity can be measured using a wide variety of assays that utilize different substrates. In the early nineties a new synthetic substrate, 1,2-O-dilauryl-rac-glycero-3-glutaric acid-(6-methylresorufin) ester (DGGR) was introduced and was initially believed to be specifically hydrolyzed by pancreatic lipase. Previous studies in humans and dogs have shown however that DGGR-based assays are not specific for the measurement of pancreatic lipase. The exact sources of lipase activity measured by DGGR-based assays in dog sera remain unknown.

OBJECTIVES

To determine the impact of pancreatic lipase-related proteins 1 and 2 (PLRP1 and PLRP2) on serum lipase activity as measured by DGGR-based assays.

METHODS

Serum lipase activity was measured using DGGR-based assays (Diazyme on a Sirrus analyzer and EUROLyser) in 3 different canine serum samples before and after addition of various concentrations of recombinant human PLRP1 or PLRP2.

RESULTS

Addition of up to 500 mg/L rhPLRP1 or 5 mg/L rhPLRP2 to canine serum samples had no impact on serum lipase activity using either the Diazyme or the EUROLyser assays. However, addition of 50 or 500 mg/L rhPLRP2 led to up to 10-fold increases of serum lipase activity using either assay.

CONCLUSIONS

This study confirms that DGGR is not specifically hydrolyzed by pancreatic lipase. Furthermore, DGGR is a viable substrate for PLRP2. As PLPR2 can be synthesized by extrapancreatic sources this finding could be clinically relevant. Further studies are needed to determine the clinical impact of PLRP2 on serum lipase activity as measured by DGGR-based assays in canine patients.

WSVA8-0079

GASTROENTEROLOGY

GLYCOGEN STORAGE DISEASE ASSOCIATED HYPOGLYCEMIA IN SHIH TZU

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INTRODUCTION

Severe and prolong hypoglycemia can be life-threatening. Therapy and prognosis depend on the underlying causes.

OBJECTIVES

To approach hypoglycemic episodes and diagnose glycogen storage disease (GSD).

METHODS

A 13-year-old, neutered female, Shih Tzu dog was presented with anorexia, weakness and diarrhea. Physical examination found abdominal pain and no detectable of neurologic deficit. Pancreatitis was confirmed with abnormal level of SNAP cPL. Although pancreatitis was resolved, the dog remained chronic, marked fasting hypoglycemia (<25 mg/dl).

RESULTS

To elucidate the cause of hypoglycemia, blood work and imaging techniques were conducted. Only mild hypoalbuminemia and moderate increased in liver enzymes were found. Cortisol level was normal. Serum insulin concentration was appropriate for the blood glucose concentration. Pre and postprandial bile acids were markly increased. Ultrasonography and CT scan demonstrated focal thickening of gastric wall and heterogenous parenchyma of liver, and heterogenous parenchyma of enlarged pancreas without mass effect in brain respectively. Histology of gastric, pancreatic and liver samples obtained from exploratory laparotomy revealed gastric amyloidosis, lymphoplasmocytic gastritis, pancreatic hyperplasia and significant glycogen accumulation in hepatocytes without evidence of other hepatic lesions. Special staining of liver confirmed GSD. The treatments were symptomatic treatment and frequent meals. The dog died after three years of diagnosis. Postmortem examination was not allowed

CONCLUSIONS

The case showed GSD is the cause of repeated hypoglycemic episodes that definitive diagnosed by histopathology. The disorder is clinically heterogeneous and progressive, and there is no effective treatment. Further study is required to improve our understanding of the disease progression and allow opportunities to investigate treatment interventions.

WSVA8-0090

INFECTIOUS AND EMERGING DISEASES

ASIA-PACIFIC SEROLOGICAL SURVEY OF CANINE HEARTWORM DISEASE BASED ON IN-CLINIC SNAP® HEARTWORM RT AND SNAP® 4DX® PLUS TEST RESULTS, 2013-2017

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INTRODUCTION

Canine heartworm, caused by infection with mosquito-borne *Dirofilaria immitis*, poses a significant health risk for dogs throughout the world. Although chemoprophylactic medications can reduce the incidence of this serious disease, heartworm continues to be prevalent in many parts of the Asia-Pacific region.

OBJECTIVES

The goal of this observational study was to evaluate Asia-Pacific regional geographic and temporal variation in canine heartworm antigen test results over a five-year period.

METHODS

Canine heartworm antigen test results spanning a period of five years (2013-2017) were obtained from an international database of SNAP® Heartworm RT and SNAP® 4Dx® Plus Test results from veterinary clinics. Repeat test results for a single patient and data from countries with less than one hundred available results were excluded. Data was included from at least 520 locations across twelve Asia-Pacific countries.

RESULTS

Table 1. Heartworm (HW) positive results by country.

Country	Test Count	HW Positive	HW Pos,% (95% CI)
Australia	9,469	53	0.6 (0.4-0.7)
China	432	4	0.9 (0.3-2.4)
Hong Kong	27,952	1,514	5.4 (5.2-5.7)
India	2,369	25	1.1 (0.7-1.6)
Japan	253,229	3,573	1.4 (1.4-1.5)
Malaysia	6,192	568	9.2 (8.5-9.9)
Myanmar	515	76	14.8 (11.8-18.1)
Philippines	950	67	7.1 (5.5-8.9)
Singapore	18,528	488	2.6 (2.4-2.9)
South Korea	9,810	903	9.2 (8.6-9.8)
Taiwan	131,729	13,526	10.3 (10.1-10.4)
Thailand	18,563	561	3.0 (2.8-3.3)
TOTAL	**479,738**	**21,358**	**4.5 (4.4-4.5)**

CONCLUSIONS

Results from this study indicate that the proportion of positive heartworm antigen test results varies greatly by country. These findings underscore that dogs within the Asia-Pacific region are at risk for heartworm infection and the importance of regular screening and use of preventatives to mitigate this risk.

WSVA8-0091

ASIA SEROLOGICAL SURVEY OF CANINE TICK-BORNE DISEASE BASED ON IN-CLINIC SNAP® 4DX® PLUS TEST RESULTS, 2013-2017

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INTRODUCTION

Tick-borne diseases (TBD) are known to occur in dogs throughout Asia, and multi-analyte rapid tests are well suited to identify canine patients with antibodies to TBD-causing pathogens. Patients may be infected with multiple pathogens, and patterns of co-infection can be useful indicators of vector distribution and subsequent exposure risk for both canines and humans.

OBJECTIVES

The aim of this study was to assess Asian geographic variation in the proportion of canine patients exposed to *Ehrlichia* spp. (EC), *Anaplasma* spp. (AP), and *Borrelia burgdorferi* (BB) and examine co-infection patterns between 2013 and 2017.

METHODS

TBD antibody test results were obtained from an international database of in-clinic SNAP® 4Dx® Plus Test results. Eleven countries representing at least 122 reporting locations were included.

RESULTS

Table 1. TBD seropositive results by country.

Country	Test Count	AP Pos,% (95% CI)	EC Pos,% (95% CI)	BB Pos,% (95% CI)	Co Pos,% (95% CI)
China	390	6.4(4.2-9.3)	6.9(4.6-9.9)	1.0(0.3-2.6)	3.1(1.6-5.3)
Hong Kong	25,466	3.5(3.3-3.7)	7.2(6.9-7.5)	0.4(0.3-0.4)	1.7(1.6-1.9)
India	1,645	23.0(21.0-25.1)	51.9(49.5-54.4)	0.6(0.3-1.1)	11.8(10.3-13.5)
Japan	122	1.6(0.2-5.8)	0.8(0.0-4.5)	0.0(0.0-3.0)	0.0(0.0-3.0)
Malaysia	4,733	17.2(16.2-18.3)	32.6(31.3-34.0)	0.5(0.3-0.8)	10.3(9.5-11.2)
Myanmar	433	4.6(2.8-7.0)	12.2(9.3-15.7)	0.2(0.0-1.3)	0.7(0.1-2.0)
Philippines	525	25.7(22.0-29.7)	53.5(49.2-57.9)	0.8(0.2-1.9)	16.0(13.0-19.4)
Singapore	15,885	3.5(3.2-3.8)	9.5(9.1-10.0)	0.3(0.2-0.4)	1.6(1.4-1.8)
South Korea	6,447	3.0(2.6-3.4)	2.9(2.5-3.3)	0.8(0.6-1.0)	0.7(0.5-0.9)
Taiwan	120,212	3.2(3.1-3.3)	7.4(7.3-7.6)	0.2(0.2-0.3)	1.1(1.1-1.2)
Thailand	14,647	29.9(29.2-30.7)	61.2(60.4-62.0)	0.3(0.2-0.4)	22.2(21.6-22.9)
TOTAL	**190,505**	**5.9(5.8-6.0)**	**12.7(12.5-12.8)**	**0.3(0.3-0.3)**	**3.2(3.1-3.3)**

CONCLUSIONS

This study shows that exposure to TBD is common in dogs throughout Asia. In cases of co-infection, patients most often had antibodies to both AP and EC.

WSVA8-0059

EVALUATION OF RAPID SEROLOGICAL TESTS FOR EHRLICHIA CANIS

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INTRODUCTION

Rhipicephalus sanguineus ticks are prevalent in South East Asia, transmit protozoal and bacterial pathogens in dogs. Of these pathogens, *Ehrlichia canis*, the causative agent for canine monocytic ehrlichiosis (CME), is most frequently associated with marked clinical manifestations in dogs. Chronic infections are also common. Diagnosis of CME is generally accomplished by hematologic and serologic findings. Conventional serological tests include immunofluorescence assay (IFA) and ELISA. Rapid in-clinic tests are also commonly used in Asia, including SNAP® 4Dx® Plus (IDEXX) and Anigen CaniV-4 (BioNote). Their relative performance has not been validated.

OBJECTIVES

To evaluate SNAP 4Dx Plus and Anigen CaniV-4 for detection of antibodies to *E. canis* in dogs from Bangkok, Thailand.

METHODS

Client-owned dogs were brought for a spay-neuter program and blood parasite diagnosis through mobile veterinary academic services. The remaining plasma samples from the program stored frozen were used. Majority of the dogs had access to outdoors and had evidence for tick or flea infestation. *E. canis* IFA testing was performed by a commercial reference laboratory. A total of 57 positive (IFA titer ≥ 1:100) and 41 negative samples were included in this study. Samples were blinded and randomized for testing with rapid in-clinic tests, according to manufacturers' instructions.

RESULTS

Compared to *E. canis* IFA, sensitivity/specificity was 94.7%/100% for 4Dx Plus, 63.2%/100% for CaniV-4.

CONCLUSIONS

This study revealed significant performance differences between the two in-clinic tests. The 4Dx Plus test may be better suited for rapid and accurate diagnosis of *E. canis* infection in this region.

WSVA8-0061

GENETIC CHARACTERIZATION OF CANINE RESPIRATORY CORONAVIRUSES IN THAILAND, DURING 2016-2017

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INTRODUCTION

Emerging canine respiratory coronavirus (CRCoV) causes mild to severe respiratory disease in dogs worldwide The genetic analysis reveals that CRCoV is similar to human (HCoV) and bovine coronavirus (BoV) and posed ability to interspecies transmission In Thailand, the genetic information of CRCoV is limited.

OBJECTIVES

This study aimed to characterize CRCoVs in dogs in Thailand during January 2016 - December 2017

METHODS

From January 2016 to December 2017, 375 nasal swab samples were collected from dogs with respiratory signs. The samples were examined for CRCoV by using RT-PCR assay. Selected RNA samples were subjected to spike (S) gene sequencing based on age, breed, severity of clinical signs, collection date and location.

RESULTS

In this study, occurrence of CRCoV was 8.8 % (33/375) Five Thai CRCoVs were selected for S gene sequencing Our results show that nucleotide identities of Thai CRCoVs (THA1-CRCoV) and CRCoV-BJ232, HCoV-OC43 and BoV-Quebec were 99.8%, 97.1% and 97%, respectively (Table 1). Phylogenetic analysis showed that all Thai CRCoVs belong to canine Asian lineage of betacoronavirus (subgroup A coronavirus) which were grouped in the same cluster with human coronaviruses and bovine coronaviruses (Figure 1).

Figure 1: Phylogenetic analysis of spike gene of coronaviruses (betacoronavirus genus). The circle symbols present Thai CRCoVs in this study.

CONCLUSIONS

CRCoV is circulating in dogs in Thailand and Thai CRCoVs were closely related to CRCoV Asian strains, HCoVs and BCoVs. Furthermore, whole genome sequencing is in process for better understanding of the viral diversity and evolution.

Table 1. Nucleotide similarities of spike gene of THA1-CRCoV to other CoVs from GenBank database.

Strain	Species	Country	Collection date	% Nucleotide similarity	Accession number
THA1-CRCoV	Canine	Thailand	2016	100	This study
THA2-CRCoV	Canine	Thailand	2016	99.7	This study
THA3-CRCoV	Canine	Thailand	2017	99.8	This study
THA4-CRCoV	Canine	Thailand	2017	99.8	This study
THA5-CRCoV	Canine	Thailand	2017	99.8	This study
BJ232	Canine	China	2014	99.8	KX432213
4182	Canine	UK	2003	98.9	DQ682406
HCOV-0C43	Human	Germany	1996	97.1	Z32768
BoV-Quebec	Bovine	Canada	1999	97.0	AF220295

WSVA8-0063

INFECTIOUS AND EMERGING DISEASES

A COMPARATIVE STUDY OF TOXOPLASMA GONDII SEROPREVALENCE IN STRAY CATS VERSUS HOUSE CATS USING IN-HOUSE AND COMMERCIAL INDIRECT FLUORESCENCE ANTIBODY TEST (IFAT)

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INTRODUCTION

Toxoplasma gondii is an important zoonotic protozoan parasite with can be encountered around the world. It is capable of infecting all warm blooded animals including humans. Cats play an important role for *T. gondii* as definitive hosts and, remarkably, are known as the only final hosts that produce oocysts in their faeces, contaminating environment (soil, food and water).

OBJECTIVES

The objective of the present study is to compare the prevalence of *T. gondii* infection in house cats versus stray cats with in-house and commercial indirect fluorescence antibody test (IFAT).

METHODS

An investigation of *T. gondii* infection was conducted in 260 cats (130 house cats and 130 stray cats) within Bangkok metropolitan and its vicinity between the year 2015-2016 using in-house and commercial IFAT. IgG antibody to *Toxoplasma* antigen of 1:100 was consider positive for *Toxoplasma* infection.

RESULTS

The overall prevalence of *T. gondii* infection in cats was 6.54% (17/260) by in-house IFAT and 23.85% (62/260) by commercial IFAT, respectively. Interestingly, the prevalence of toxoplasmosis in stray cats was significantly higher than in household cats by both tests. Furthermore, IgM antibody to *Toxoplasma* will be screened to detect acute infection in these samples.

CONCLUSIONS

In conclusion, the results demonstrated that commercial IFAT had a higher sensitivity and specificity compared to in house IFAT and indicated that commercial IFAT can be used as the routine diagnostic test for the detection of *T. gondii* infection. Cats in the Bangkok metropolitan area and vicinity could serve as a zoonotic reservoir for toxoplasmosis.

WSVA8-0119

THE DISTRIBUTION OF FELINE PANLEUKOPENIA REPORTED BY AUSTRALIAN VETERINARY CLINICS AND SHELTERS

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INTRODUCTION

Feline Panleukopenia (FP) is a highly contagious, often fatal disease of cats (especially kittens) that has recently re-emerged in Australia, predominantly associated with shelter-housed cats Limited information exists on the prevalence, distribution or risk factors for infections in Australia or worldwide.

OBJECTIVES

To describe the distribution of FP in the Australian cat population. To determine the role of shelters in disease transmission.

METHODS

A national online survey (January-July 2017) of companion animal veterinarians was conducted to estimate the number of FP cases diagnosed by laboratory tests (e.g. faecal antigen, PCR) or clinical presentation, during 2015 to 2016. Information on the clinic's extent of involvement in shelter work was also collected. Maps were generated to display FP cases, outbreaks and trends.

RESULTS

A total of 534 unique veterinary clinics responded (23.5% of Australian clinics); 154 and 174 FPV cases were reported by 23 (mean 3.3 per clinic; 95% CI 0.3-6.8%) and 47 (mean 3.7; 95% CI 1.2-6.2%) clinics in 2015 and 2016, respectively. Overall, 9% of clinics (47/534) reported cases during the study period.

In 2015, 72% of FPV cases were reported by shelter clinics (P=0.30) compared to only 51% in 2016 (P=0.07). Size of clinic and number of FPV cases reported in 2016 were correlated (r=0.42, P<0.01). The most cases were reported from New South Wales and Victoria. However, this is the first report to document FP in Western Australia, Queensland and South Australia.

CONCLUSIONS

FP, previously only reported in NSW and VIC since 2014, is occurring over a wider geographic area than previously thought.

WSVA8-0070

INFECTIOUS AND EMERGING DISEASES

IS FECAL SAMPLING THE IDEAL SPECIMEN FOR CANINE PARVOVIRUS PCR DETECTION? COMPARISON OF A NOVEL POINT-OF-CARE DIAGNOSTIC WITH REAL TIME PCR

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INTRODUCTION

Canine parvovirus (CPV) is one of the most common causes of acute hemorrhagic enteritis in young dogs, while clinical diagnosis is often indecisive. Infection leads to a rapid loss of condition of the animal and if not treated at an early stage will eventually lead to the death of the animal. Thus, this disease, as well as its diagnosis is of great concern.

OBJECTIVES

The aim of this study was to compare the efficiency of a point of care in clinic test, PCRun® DNA Detection Kit with an in-house probe-based TaqMan Real Time PCR and to determine the optimal sample to be used for PCR analysis – fecal (anal) swabs, oral swabs, or whole blood.

METHODS

Whole blood, fecal and oral swabs samples were collected from 44 unvaccinated healthy puppies and 60 clinical cases of diarrhea or hemorrhagic gastroenteritis from CPV vaccinated or non-vaccinated dogs. DNA purification was performed with a commercial kit and samples were tested using each of the two PCR methods targeting the VP2 gene.

RESULTS

The PCRun® assay, when compared to the TaqMan Real Time PCR assay, had a specificity of 97.9%, 97.7% and 98.1% and sensitivity of 100%, 96.7% and 98.1% when using samples from blood, oral swabs or fecal swabs respectively

CONCLUSIONS

The in-clinic PCR assay was found to be highly specific and sensitive in all samples. Blood and fecal samples has a slight advantage over oral samples. The PCRun® DNA Detection Kit is a suitable test for a simple initial in clinic screening in a short time.

WSVA8-0159

INFECTIOUS AND EMERGING DISEASES

MOLECULAR DETECTION OF HEMOTROPIC MYCOPLASMA SPP. AND BARTONELLA SPP. IN CATS AND FLEAS IN CHIANG MAI, THAILAND

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INTRODUCTION

Hemotropic *Mycoplasma* spp. and *Bartonella* spp. are important vector-borne pathogens in cats.

OBJECTIVES

The study aim was to use molecular techniques to investigate the presence of hemotropic *Mycoplasma* spp. and *Bartonella* spp. DNA in cats and fleas in Chiang Mai, Thailand.

METHODS

A total of 98 blood samples and 22 pools (1-5 fleas per pool) of fleas were collected from cats visiting the Small Animal Veterinary Teaching Hospital, Chiang Mai University and from cats residing in a temple in Chiang Mai Province between June and November 2016. Polymerase chain reaction assays (PCR) targeting each pathogen were performed.

RESULTS

The overall prevalence rates for at least one pathogen in cats and fleas were 73.5% (72/98) and 68.2% (15/22), respectively. Hemotropic *Mycoplasma* spp. DNA was amplified from 34.7% of cats and 9.1% of fleas. *Bartonella* spp. DNA was amplified from 62.24% of cats and 63.6% of fleas, respectively. Of 72 positive cat samples, 38 contained only *Bartonella* spp. DNA, 11 contained only *Mycoplasma* spp. DNA and 23 contained DNA of both pathogens Of 15 positive flea pools, 13 contained only *Bartonella* spp. DNA, 1 contained only *Mycoplasma* spp. DNA and 1 contained DNA of both pathogens.

CONCLUSIONS

The prevalence of hemotropic *Mycoplasma* spp. and *Bartonella* spp. in cats in Chiang Mai is high. Cats can be a potential reservoir for a zoonotic infection of *Bartonella* spp. in humans. Effective flea control in this population is suggested.

WSVA8-0140

INFECTIOUS AND EMERGING DISEASES

CASE REPORT: DIFFUSE GRANULOMATOUS LYMPHADENITIS IN A MINIATURE SCHNAUZER ASSOCIATED WITH ACID-ALCOHOL RESISTANT BACILLUS (MYCOBACTERIUM AVIUM) FROM PARAGUAY

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INTRODUCTION

M. avium is considered an emerging and opportunistic pathogen that can infect animal and human species. The dog has been seen as an animal resistant to infections by M. avium, cases have been reported in Miniature Schnauzer and Basset Hound. It is believed that the cause would be a defect in the immune response of genetic origin with a pattern of autosomal recessive inheritance in miniature Schnauzer bloodlines.

OBJECTIVES

describes a case of diffuse granulomatous lymphadenitis in a miniature Schnauzer associated with BAAR

METHODS

Miniature Schnauzer, male, 2 years old, for gradual weight loss, superficial adenopathy and intermittent lameness. All cytological lymph nodes samples were stained with 10% Giemsa and ZN; and evaluated by a 100X light optical microscope. The owner authorizes the necropsy, taking samples and corresponding studies of the pet.

RESULTS
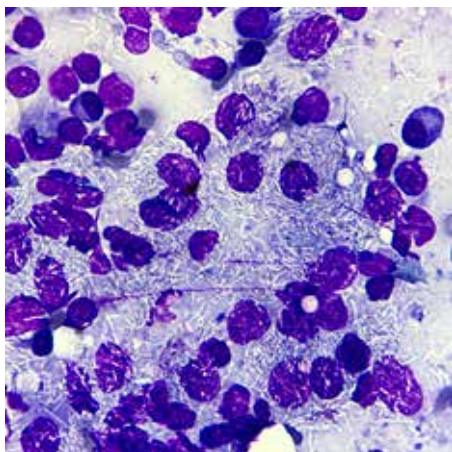
Cytological samples from lymph nodes showed a poorly defined intracytoplasmic presence of refractive linear structures to pale basophils. ZN staining was positive by revealing the presence of BAAR in the macrophages.

The PCR technique using primers (Telenti 1993), where a pattern corresponding to Mycobacterium intracellulare type 1 / Mycobacterium chimera type 1 is obtained, both correspond to the Mycobacterium avium complex. Both samples tested positive for this test.

CONCLUSIONS

This disease can present as a generalized lymphadenopathy, so a differential diagnosis should be made with lymphoma. This is the first report of a systemic infection of M. avium in a canine in the Paraguay and should be included as a differential diagnosis and zoonotic potential in immunocompromised persons

WSVA8-0039

DETECTION OF BLOOD TRANSMITTED PATHOGENS FROM RECRUITED CANINE DONOR CANDIDATES IN TAIWAN

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INTRODUCTION

As rapid progress has been achieved in companion animal medical care in recent years, transfusion techniques were much more broadly and frequently utilized in veterinary medicine. Besides the extensive clinical application of veterinary blood transfusion, some other important related risks of transfusion-associated blood pathogen transmission should also be alerted. The whole selecting process, other than the first step of selection (inquiry of age, weight and husbandry management), includes the health condition monitoring of the donor candidates as well as potential pathogen detection.

OBJECTIVES

After comparing transfusion medical and blood transmitted pathogenic literatures internationally and domestically, this study aimed to set up a standard of blood pathogen screening for canine donor candidates in Taiwan.

METHODS

From June 2016 to January 2018, 138 dogs were recruited into the program. After the steps of screening, 56 candidates donated blood and were tested for blood pathogen infections by using nested PCR with the respective primer sets designed for different target genes (18S rRNA, 16S rRNA and *glt*A gene).

RESULTS

The results revealed positive on *Rickettsia felis* 2.2% (1/56) and negative on *Anaplasma platys*, *Babesia canis*, *Babesia gibsoni* and *Ehrlichia canis*.

CONCLUSIONS

In accordance with the results, even for those donor candidates under good husbandry management and current on heartworm, ectoparasitic preventives as well as up-to-date vaccinations, there are still underlying infectious risks of those blood transmitted pathogens mentioned above.

WSVA8-0053

INFECTIOUS AND EMERGING DISEASES

CASE STUDIES AND MOLECULAR CHARACTERISATION OF THE TRYPANOSOMES RESPONSIBLE FOR CANINE AFRICAN TRYPANOSOMOSIS (CAT) IN NSUKKA AREA, ENUGU STATE, NIGERIA

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INTRODUCTION

Trypanosomosis is one of the major diseases of dogs in Nsukka area, Enugu state of Nigeria. Microscopy is the widely used method of diagnosis and the most reported pathogens are the human infective trypanosomes *Trypanosoma brucei gambiense* and *T.b. rhodesiense*, in addition to *T.b. brucei* and *T. congolense*. Microscopy alone cannot distinguish within the subspecies of trypanozoon group,

OBJECTIVES

To investigate the most prevalent subspecies of *T. brucei* infecting dogs in the study area.

METHODS

We conducted physical examination on the trypanosomes-infected dogs and collected appropriate history from the dog owners. We used ITS 1 primer and sequence analysis to study the sub species of *T. brucei*.

RESULTS

Sequence analysis detected *T.b. gambiense, T. evansi* and *T. congolense* in the infected dogs. Anemia evident in low PCV, Hb, RBCs and fever were common in the infected dogs. The chief complaint were change in eye colour and gradual loss or complete loss of appetite. The common observations during physical examination were corneal opacity and enlargement of superficial lymph nodes. Some serum biochemical changes were noticed in the infected sampled dogs.

CONCLUSIONS

T.b. gambiense, *T. evansi* and *T. congolense* were responsible for canine trypanosomosis in the study area, which suggest that dogs could be reservoirs of infections in humans and animals in the study area. The disease caused decrease in PCV, RBC, Hb, serum albumin and increase in serum BUN in most of the infected dogs.

WSVA8-0075

INTERNAL MEDICINE (OTHER)

PRIMARY CENTRAL NERVOUS SYSTEM LYMPHOMA IN A DOG: A CASE REPORT

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INTRODUCTION

Primary CNS lymphoma was extremely rare and that accounted for only 4% of all primary intracranial tumor. In canine, differentiation of lymphoma subtypes can be performed by histopathological evaluation and immunophenotyping. Subtyping is crucial for prognosis and therapeutic plan.

OBJECTIVES

We describe clinical findings, management and pathological diagnosis of a dog with CNS lymphoma. Subtyping of the tumor was done by immunohistochemistry for CD3 and CD79a.

METHODS

T2 W	T1 W- post contrast
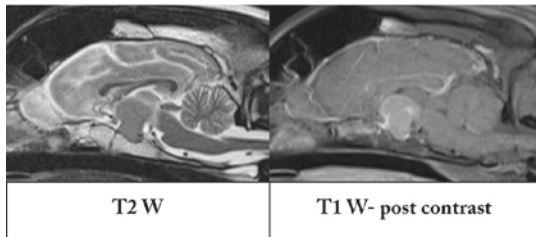
Figure 1 :

Sagittal MR imaging identified mass effect at extraparenchymal structures on sellar region which were hyperintense on T2-weighted images and contrast enhancement on T1-weighted images.

T2 W	T2 FLAIR	T1 W	T1 W- post contrast
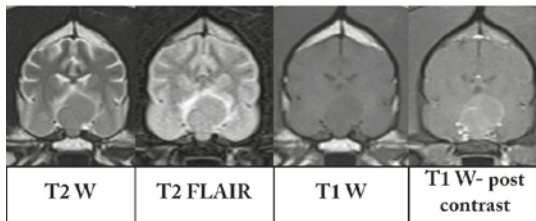
Figure 2 :

Transverse MR imaging of brain showed hyperintense on T2-weighted images, perilesional hyperintensity on FLAIR images, hypointense on T1-weighted and contrast enhancement on T1-weighted images.

An eight-year-old, intact male crossbred dog was presented neurological deficits with disoriented, left circling, nystagmus and left cranial nerve deficits. Blood profile was within normal limits. Superficial lymph nodes were appeared normal at palpation. Magnetic resonance imaging identified mass effect at extraparenchymal structures on sellar region nearby the pituitary gland which were hyperintense on T2-weighted images, contrast enhancement on T1-weighted images and had perilesional hyperintensity on FLAIR images. Prednisolone was used as an anti-inflammatory drug and after that clinical sign was improved to conscious and circling was not detected. Thirty days later neurological signs progressively worsened, obtunded, inappetence, respiratory distress and the dog was died from respiratory arrest.

RESULTS

Gross examination revealed that the mass was located at sellar region of the brain. Histopathological evaluation reveal aggregated round cells and lymphocytes in this region. This round cells had moderate amount of cytoplasm, finely stripped nucleus and some of these cells were bi-nucleated with nuclear molding. Immunohistochemical analysis was performed for CD3 and CD79a, and the tumor was characterized as T-cell-rich B-cell lymphoma.

CONCLUSIONS

We reported a rare case of primary CNS lymphoma, subtyping T-cell-rich B-cell lymphoma.

WSVA8-0076

INTERNAL MEDICINE (OTHER)

METASTASIS OF ORAL MALIGNANT MELANOMA IN A DOG: A CASE REPORT

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INTRODUCTION

Malignant melanoma is a life-threatening disease characterized by highly aggressive biological behavior and often metastases to regional lymph nodes, lungs and also spread distantly to other organs.

OBJECTIVES

We report an advances clinical case of malignant melanoma that metastasized to brain and other organs and describe diagnostic tools that aid ante-mortem diagnosis.

METHODS

A fourteen-year-old, male, crossbred had been removed malignant melanoma from lip and neck that distant metastasis was not detected. Thirty days later the dog came with acute neurological deficits, disoriented and compulsive walking. The clinical sign had progressed rapidly to tetraparesis, head turn and cranial nerve deficits. Chest radiograph revealed diffuse pulmonary metastases. CT scanning showed brain edema with multiple masses and multiple nodules throughout lung field. Abdominal ultrasonography revealed a small hypoechoic nodule presented at spleen. The dog was treated with prednisolone and mannitol to relieve brain edema. Clinical sign was improved for a short period and progressively worsened and died from respiratory arrest.

RESULTS

Multiple black nodules were noted at omental fat, kidney, intercostal muscle, myocardium, lung, eye and brain. A microscopic examination revealed a spindle to polyhedral shape of neoplastic cell that had prominent vesicular nuclei located at the center. Some of these cells contained brown-pigmented granules in their cytoplasm. Histopathologic findings were diagnosed as metastatic malignant melanoma.

CONCLUSIONS

We demonstrated a rare case of disseminated metastatic of oral malignant melanoma that metastasized to brain, uvea and other organs. An investigation of the metastatic site of malignant melanoma could provide information for a further diagnosis and monitoring
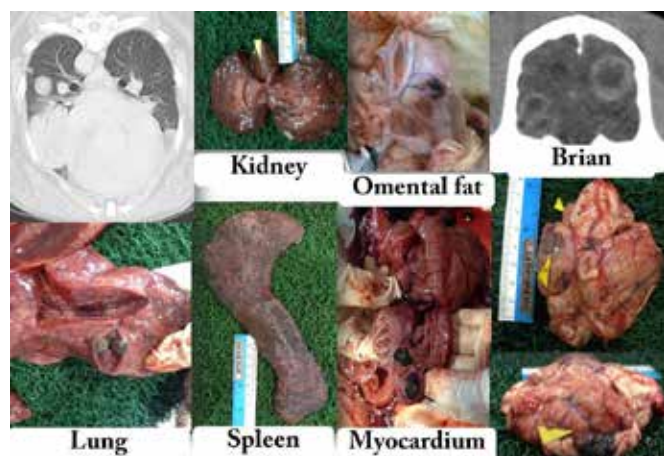

Figure1: showed multiple black nodules were noted at omental fat, kidney, spleen, myocardium, lung and brain.

WSVA8-0173

INTERNAL MEDICINE (OTHER)

IDIOPATHIC ANEMIA WITH SEVERE THROMBOCYTOPENIA IN 5 DOGS: CASE REPORT IN JAKARTA ANIMAL CLINIC AND HOSPITAL [2016-2017]

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INTRODUCTION

Immune-mediated hemolytic anemia (IMHA) and immune-mediated thrombocytopenia (ITP) are one of the most common types of diseases in dogs. Both IMHA and ITP can be classified as either "primary (idiopathic)" or "secondary." In primary disease, an underlying cause of the immune destruction cannot be identified. In comparison, secondary IMHA or ITP occurs when the immune system inadvertently destroys its own red blood cells or platelets. IMHA and ITP frequently occur in dogs, and most affected dogs are middle-aged females.

OBJECTIVES

To identify idiopathic anemia with severe thrombocytopenia (<or=15.0 x 10^3 platelets/ µL), and to evaluate factors, erythrocytes and thrombocytes profile, treatment, and survival of dogs.

METHODS

Retrospective and observational study in 5 dogs with idiopathic anemia and severe thrombocytopenia in Jakarta Animal Clinic (ACJ) and Jakarta Animal Hospital, Indonesia.

RESULTS

Five dogs were identified with the diagnosis of idiopathic anemia and severe thrombocytopenia (Table 1). There were 1 dog (< 1 years old), 3 dogs (1-3 years old) and 1 dog (> 4 years old). There were 3 dogs male and 2 dogs female. Breed of dogs were 3 shepherd dogs (German and Belgian), 1 Yorkshire dog and 1 Samoyed dog. There were 2 dogs that given blood transfusion therapy. The therapies associated with survival, 80% (4 dogs) survived and 20% (1 dog) died after more than twice blood tranfussion therapy.

Table 1 Erythrocytes and thrombocytes profile in 5 dogs of cases in idiopathic anemia with severe thrombocytopenia in Jakarta Animal Clinic and Hospital in 2016-2017

Parameters	Unit	Normal ranges	Case 1	Case 2	Case 3	Case 4	Case 5	Average
Erithrocytes	10^6 /µL	5.50-8.50	1,16	1,15	4,93	2,83	1,46	2,31
Hemoglobin	g/dL	12.0-18.0	2,3	2,7	9,8	5,5	3,7	4,8
Hematocrit/Packed Cell Volume (PCV)	%	37.0-55.0	8,3	8,1	31,3	16,24	11,31	15,05
Mean Corpuscular Volume (MCV)	fL	60.0-77.0	65	70,4	64	57	77	66,68
Mean Corpuscular Hemoglobin Concentration (MCHC)	g/dL	32.0-36.0	27,7	33,33	31,8	34,1	32,4	31,866
Trombocytes/Platelets	10^3 /µL	200-500	8	6	15	2	4	7

CONCLUSIONS

Dogs in this study had a diagnosis of idiopathic anemia and severe thrombocytopenia. The therapies associated with survival, 80% survived and 20% died.

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INTERNAL MEDICINE (OTHER)

TIME-COURSE EVALUATION OF 2,3-DIPHOSPHOGLYCERATE LEVELS IN CANINE PACKED RED BLOOD CELL STORED IN TWO DIFFERENT ADDITIVE SOLUTIONS

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INTRODUCTION

The 2,3-diphosphoglycerate (2,3-DPG) is an allosteric modulator of hemoglobin which facilitates the release of oxygen to tissues. Depletion of 2,3 DPG during RBCs storage is associated with a decreased release of oxygen from the transfused RBCs. Previous studies demonstrated benefits of using additive solutions in order to improve maintenance of 2,3-DPG in human RBCs during storage.

OBJECTIVES

To evaluate the changes of 2,3-DPG levels in canine RBCs stored in two different additive solutions including SAGM and AS-3.

METHODS

Six healthy dogs were included in this study. A unit of whole blood-derived pRBC was prepared from each dog and divided equally into three portions. The first portion was resuspended with SAGM *(SAGM group)*, the second portion was resuspended with AS-3 *(AS-3 group)*, and the third portion was not resuspended with additive solution *(control group)*. The characteristics of SAGM and AS-3 are shown in Table 1.

All pRBC groups were stored in 4°C and samples were obtained at day 1, 14, 21, 28, 35 and 42 of storage. The 2,3-DPG levels from each sample were determined by enzymatic assay kit.

Table 1. Composition and characteristics of SAGM and AS-3 additive solutions used in this study.

	SAGM[§]	AS-3[‡]
Each 100 mL contains		
Adenine (g)	0.0169	0.030
Dextrose (g)	0.900	1.000
Mannitol (g)	0.525	-
Sodium Chloride (g)	0.877	0.410
Monobasic Sodium Phosphate (Monohydrate) (g)	-	0.276
Sodium Citrate (Dihydrate) (g)	-	0.588
Citric acid (Monohydrate) (g)	-	0.042
pH	6.2	5.8
Osmolarity (mOsm/kg)	376	291

[§]SAGM: saline-adenine-glucose-mannitol additive solution
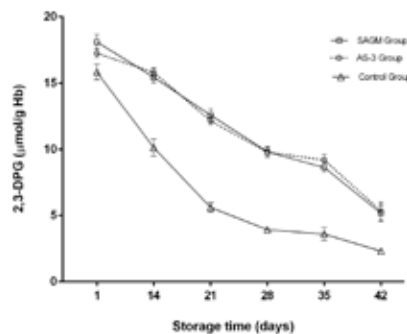[‡]AS-3: additive solution formula as 3

RESULTS

The 2,3-DPG levels were significantly decreased ($p < .001$) overtime in all groups (Table 2). The overall 2,3-DPG decline rates were significantly slower in SAGM and AS-3 groups compared to control ($p < .05$). There were no significant differences in the 2,3-DPG levels and decline rates between SAGM and AS-3 groups (Figure 1).

Table 2. Level of 2,3-DPG in canine pRBC form different groups determined on different days of storage[a].

	SAGM group	AS-3 group	Control group
2,3-DPG (µmol/g Hb)			
Day 1	18.117 (0.53)	17.250 (0.59)	15.833 (0.58)
Day 14	15.450 (0.41)	15.817 (0.33)	10.133 (0.63)
Day 21	12.583 (0.48)	12.150 (0.38)	5.617 (0.36)
Day 28	9.817 (0.38)	9.800 (0.34)	3.933 (0.18)
Day 35	8.633 (0.34)	9.200 (0.40)	3.60 (0.48)
Day 42	5.183 (0.59)	5.333 (0.69)	2.317 (0.18)

[a]Data shown represent mean ± SD (N=6).

Figure 1. Level of 2,3-DPG levels in canine pRBC from SAGM group (□), AS-3 group (O), and congrol group (Δ) during storage. Results shown represent mean±SD (n=6).

CONCLUSIONS

These results indicate that SAGM and AS-3 additive solutions provide better maintenance of 2,3-DPG levels in canine pRBCs during storage.

WSVA8-0083

INTERNAL MEDICINE (OTHER)

BABESIOSIS INDUCED EVAN'S SYNDROME (IMHA AND ITP) IN TWO COCKER SPANIEL PUPPIES.

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INTRODUCTION

Immune-mediated haemolytic anaemia (IMHA) are the most common cause of haemolytic anaemia and immune mediated thrombocytopenia (ITP) is the most common cause of severe thrombocytopenia in dogs. In combinations they are known as Evans syndrome, an autoimmune disease in which dogs antibodies attack their own red blood cells and platelets and often fatal.

OBJECTIVES

To successfully management. Evan's syndrome secondary to canine babesiosis.

METHODS

Two male Cocker spaniel puppies of age 6 months of the same litter were presented in our practice with profound anemia and thrombocytopenia with recent clinical signs of hematuria, fever, lymphadenopathy, petechiae, echymosis and purpura. Routine clinical findings included blanched mucous membranes, pounding pulses and lethargy. Blood routine revealed marked leukocytosis(W.B.C > 23,000 cells/cmm, severe anemia (Hb<2.9 g/dl) and thrombocytopenia (platelets <14,000 cells/cmm. Coombs in both the dogs showed negative results, the saline agglutination tests showed autoagglutination and the presence of spherocytosis was evident. Flow cytometrical analysis confirmed IMHA. PCR and blood smears confirmed *Babesia gibsoni*. Treatment with Diaminazene, doxycycline and prednisone were initiated.

RESULTS

One of the pups showed recurrence of anemic hypoxia and thrombocytopenia within 48 hours and was transfused again with DEA1.1 negative whole blood. Both the pups responded well to combination of Doxycycline @ 10 mg per kg sid, prednisone@ 1m mg per kg tapered to 0.25 mg per kg for 2 and Clindaymicin 15 mg per kg bid for 2 weeks.

CONCLUSIONS

This abstract reports the uncommon Evan's syndrome secondary to canine babesiosis and its successful management with blood transfusion and combination therapy.

WSVA8-0114

INTERNAL MEDICINE (OTHER)

ANTIMICROBIAL EFFICIENT OF CHLORHEXIDINE/ CHITOSAN ANTISEPTIC GEL IN VITRO

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INTRODUCTION

Increasing bacterial resistance to antibiotics is a worldwide problem, which is why external-use antiseptics are gaining attention. Chlorhexidine is one such antiseptic, it reacts with the microbial cell surface. Chitosan increases the permeability of the membranes.

OBJECTIVES

Aim of this study was to assess antimicrobial efficacy of different concentration of chlorhexidine/chitosan gel *in vitro.*

METHODS

Chlorhexidine/chitosan gel was carried *in vitro* using the diffusion agar method. Reference and wild strains of Gram-positive microorganisms: *Staphylococcus aureus* (ATCC 25923), *Enterococcus faecalis* (ATCC 29212); as well as reference and wild strains of Gram-negative microorganisms: *Escherichia coli* (ATCC 25922), *Pseudomonas aeruginosa* (ATCC 27853) were sown to Mueller-Hinton agar ((CM 0337) Oxoid LTD, Basingstoke, Hampshire, England).Three different concentration chlorhexidine/chitosan gel (0.25 %; 0.5 % and 1.0 %) were tested against each strain of organism.

RESULTS

The gel was active against all bacteria, but effectiveness was dependent on specie and strain. The greatest activity was noticed against the reference strain of *S. aureus* (diameter of sterile zone from 26,90±0,61 mm to 29,65±0,30 mm). (c.f. Figures 1 – 2). No significant difference between 0.5 % and 1.0 % concentrations.

CONCLUSIONS

Different concentrations of chlorhexidine/chitosan gel appeared to be effective against all bacteria strains. Whilst the increase in concentration from 0,25% to 1% increased the sterile zone in almost all cases.

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WSVA8-0149

NEPHROLOGY

OUTCOMES OF HEMODIALYSIS IN CHRONIC KIDNEY DISEASE DOGS WITH UREMIC CRISIS SITUATIONS

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INTRODUCTION

Uremic situations commonly develop in chronic kidney disease dogs. The complications associated with uremia including seizure, pneumonitis, cardiac arrest are situations shortened CKD patient life. The hemodialysis used to eliminate the uremic toxin and balances the electrolyte.

OBJECTIVES

The study was to determine the short-term outcome for chronic renal disease dogs with uremic crisis situations treated with hemodialysis compared to conservative treatment.

METHODS

The study was retrospectively reviewed from September 2017 to October 2015 at Kasetsart University Veterinary Teaching Hospital (KUVTH), Faculty of Veterinary Medicine, Kasetsart University. Medical records of 21 uremic dogs were searched and divided into 2 groups of treatment. Hemodialysis treatment (HD) group was consisted of 8 dogs. Conservative treatment (CV) group was consisted of 13 dogs admitted to KUVTH. Two sample t-test and Chi-square test were performed to determine the differences of age, body weight, gender and hematologic values between two groups of treatment. Method of survival analysis with Kaplan meier survival curve was used. The log-rank test was analyzed to determine the difference of median survival time between two groups of treatment.

RESULTS

Between two groups of treatment, body weight and serum creatinine of HD group were significantly higher (P<0.05) than CV group. The median survival time of uremic dogs treated with hemodialysis, 17 days (95%CI = 12-19 days), was significantly different (P<0.05) from conservative treatment group, 3 days (95%CI = 2-6 days).

CONCLUSIONS

Hemodialysis is tools for prolong life and improving quality of life in CKD patient with acute uremic situation.

WSVA8-0078

NEPHROLOGY

CORRELATION BETWEEN RENAL DAMAGE AND CLINICOPATHOLOGICAL CHANGES IN IMMUNE-COMPLEX GLOMERULONEPHRITIS AND NON-IMMUNE-COMPLEX GLOMERULONEPHRITIS IN DOGS

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INTRODUCTION

Immune-complex glomerulonephritis (ICGN) and non-ICGN are major forms of chronic kidney disease (CKD) in dogs. However, the difference between the progression of CKD in ICGN and non-ICGN, is not fully understood.

OBJECTIVES

We aimed to clarify the association between pathological changes in the kidneys and clinicopathological changes in ICGN and non-ICGN, in dogs.

METHODS

Renal biopsy specimens (n=24) were obtained from dogs with protein-losing nephropathies, and these were examined using light microscopy, electron microscopy, and immunofluorescence for the presence of immunoglobulins and complement 3. Severity of glomerular sclerosis (GS) and interstitial fibrosis (IF) were quantitatively evaluated, and correlations between these scores and clinicopathological parameters were statistically analyzed.

RESULTS

Eighteen and six cases were diagnosed with ICGN and non-ICGN, respectively. The GS score in ICGN showed no significant correlation with any clinicopathological parameter, including plasma concentrations of urea, creatinine (pCre), total protein and albumin, and urinary protein/creatinine ratio; however, the IF score showed a significant correlation with pCre. The GS score in non-ICGN showed a significant correlation with pCre, but IF showed no correlation with any clinicopathological parameter.

CONCLUSIONS

Progression of CKD in ICGN was associated with IF severity, whereas in non-ICGN, it was associated with GS severity. This difference between ICGN and non-ICGN might be associated with the amount of proteinuria, which is severe in ICGN; the proteinuria is a major inducing factor for IF, which is in turn associated with decreased renal function.

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WSVA8-0035

BACTERIAL ISOLATION FROM URINARY TRACT INFECTIONS AND URINARY TRACT INFECTION ASSOCIATED WITH UROLITHS FORMATION IN DOGS: A RETROSPECTIVE STUDY

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INTRODUCTION

Urinary tract infections (UTIs) are common in small animal practice. *Escherichia coli* is the most common bacterial isolated from UTIs in dogs. Previous studies have indicated that *Staphylococcus* spp and/or others bacteria may lead to cause stone formation in dogs, especially struvite stones

OBJECTIVES

The objectives of the present study were to study the most common bacterial isolation in dogs diagnosed with UTIs and those with UTIs associated with uroliths formation in dogs

METHODS

The study conducted a retrospective study. All urine samples were collected by cystocentesis and later submitted to Veterinary Teaching Animal Hospital to identify bacterial infection

RESULTS

Of 137 dogs diagnosed with bacterial UTIs, 74 females and 63 males, were enrolled in the present study. *Proteus mirabilis* and *Staphylococcus spp* were the most common bacterial isolated from UTIs (55 samples (40.14 %)). Meanwhile, 82 dogs (59.8%) diagnosed with urolithiasis associated with UTIs, the most bacterial isolation were *Staphylococcus spp* (30.76%) and *Escherichia coli spp* (19.23%), respectively

CONCLUSIONS

Staphylococcus spp and E. coli are the most bacterial UTIs associated with urolithiasis. In contrast, several studies have indicated that the most bacterial isolated from those with urolithiasis are *Staphylococcus* and *Proteus*. Moreover, the most bacterial UTIs in our study were *Proteus* spp., while other studies *E. coli* have indicated. Further investigation on bacteria isolated from stone cultures could give more information on correlation between bacteria and urine stone formation

WSVA8-0067

NEUROLOGY - NEUROSURGERY

THORACAIC VERTEBRAL OSTEOCHONDROMA IN A FELINE LEUKEMIA VIRUS NEGATIVE DOMESTIC SHORT HAIR CAT: SURGICAL MANAGEMENT AND OUTCOME

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INTRODUCTION

1 year old intact male cat was presented with slow progressive pelvic limbs paresis grade IV over 2 months and hyperesthesia at thoracic area. The MRI showed extradural mass at the left dorsolateral of 2nd thoracic- 3rd thoracic (T2-T3) and compress to the spinal cord.
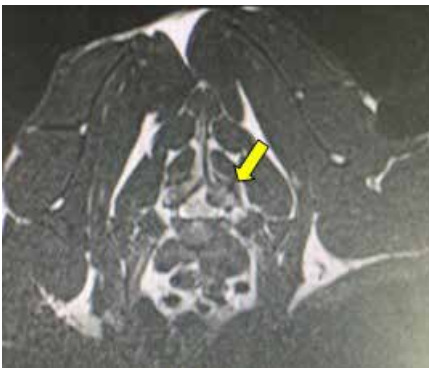
OBJECTIVES

To report surgical treatment and outcome in a case of thoracic vertebral osteochondroma in feline leukemia virus negative Domestic shorthair cat.

METHODS

A dorsolateral approach to left-sided of T_2-T_3 was performed, the proliferative bony mass was found and removed. Bone samples were collected for histological examination. Left-sided hemilaminectomy of T_2-T_3 was performed until spinal cord was seen. After flushing with sterile saline, the defect was covered with an autologous fat graft and the incision was closed routinely.
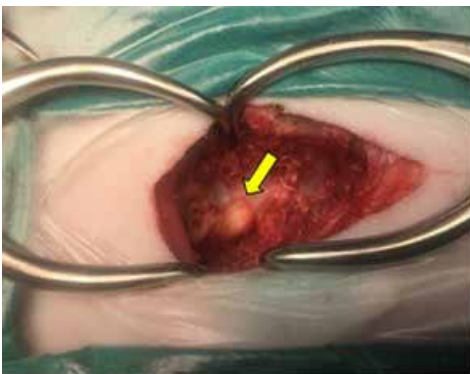
RESULTS

After operation for 8 weeks, the cat showed improvement in the motoric function of the pelvic limbs with the ability to walk but proprioceptive responses deficit. No hyperesthesia was detected. Six months after surgery, only pelvic limbs ataxia was detected. The biopsy showed cartilaginous matrix with uniform sized and shaped, vertebral osteochondroma was diagnosed.

CONCLUSIONS

Solitary osteochondroma as a cause of neurological deficits in the pelvic limb is rarely observed in cats and usually associated with feline leukemia virus, but contrast in this case. There are only 1 reported feline case described in the literature. The surgical treatment was succeeded, the cat did not show any neurological deficits or signs of recurrence until now. Normally, prognosis of solitary osteochondroma is good after surgical excision.

WSVA8-0099

NEUROLOGY - NEUROSURGERY

UNILATERAL VERTEBRAL BODY PLATE FIXATION IN MIDDLE BREED CANINE VERTEBRAL FRACTURE

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INTRODUCTION

Traumatic vertebral fractures are common serious emergencies in small animal practice. Surgical techniques are described for fixation of thoracolumbar vertebral fractures and luxations in dogs including vertebral body plating, cross pinning, spinal stapling, polymethylmethacrylate (PMMA)-pin fixation. In this case, we used unilateral vertebral body plate fixation to stabilize the traumatic vertebral body fracture.

OBJECTIVES

To evaluate the efficacy and stabilization of Unilateral vertebral body plate fixation in middle breed canine vertebral fracture.

METHODS

A 5-years-old intact male, 23kg, mixed dog was presented to the Animal Medical Center Chonbuk National University with a history of fall from the cliff in hunting.

In Neurologic examinations, patient was paraparetic and had loss of bilateral conscious proprioception, hopping reflex. Deep pain perception was weak in left hind limb and absent in right hind limb. Radiographs demonstrated a Vertebral fracture at L2-L3 with fractures of spinous process

Based on these results, it was diagnosed lumbar vertebral fracture. Because of rapid loss of function in the hind limbs, Hemilamincetomy and unilateral 2.7 locking plate fixation was performed the same day.

RESULTS

Two days after surgery, deep pain was recovered and the superficial pain recovered the next day. After cage rest for a week, rehabilitation and acupuncture therapy was performed. Postoperative radiographs taken to evaluate plate placement two weeks after surgery showed no migration and infections.

CONCLUSIONS

From this study, it is known that Unilateral vertebral fixation is one of the good options to stabilize vertebral fracture in middle breed dog.

WSVA8-0092

NEUROLOGY - NEUROSURGERY

THE USE OF CANINE VERTEBRAL SCREW AND ROD FIXATION SYSTEM FOR VERTEBRAL STABILIZATION IN 48 DOGS

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INTRODUCTION

Canine vertebral screw and rod fixation system (CVSRF) was specially developed for vertebral column stabilization in dog. It made from titanium alloy to decrease postoperative complications. All outcomes of mechanical testing of the CVSRF during bending were better than Screw-PMMA technique.

OBJECTIVES

To report the success of vertebral stabilization using canine vertebral screw and rod fixation system in dog.

METHODS

Forty-eight dogs with body weight range from 5-25 kg were refer to the Neurological center of Kasetsart University Veterinary Teaching Hospital. They were presented with clinical signs of paraparesis from vertebral fracture and luxation between the 6th thoracic vertebrae to the 1st sacrum. The operation with CVSRF system was performed to stabilization at bilateral of the vertebrae. Screw with diameter 3.5 mm and rod with diameter 4.0 mm was used as an implant and adjusted the length of screw and rod according to their body weight. The spinal construction and postoperative wound complication were recorded in each appointment at 0, 7 and 30 days after operation.

RESULTS

Postoperative radiography demonstrated the vertebral columns were in alignment with spinal implant in all patients. All postoperative wounds were completely healed without complication after operation for 7 and 30 days. However, a week later after surgery, 6.25% of them (n = 3) found a loosening of the screw from the vertebral body.

CONCLUSIONS

The CVSRF system device could be a benefit instrument for the vertebral column fixation in small to medium size dogs with less complication wound after surgery.

WSVA8-0141

ONCOLOGY - ONCOSURGERY

ELECTROMAGNETIC THERMOABLATION FOR ORAL TUMORS IN DOGS

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INTRODUCTION

Thermal ablation by using electromagnetic thermotherapy system has been a promising cancer treatment modality in recent years. This system applies a high frequency alternating electromagnetic field to heat up apace the alloy needles which are inserted into and beneath the target tumor to cause denaturation of surrounding tissues or vessels and eventually coagulative necrosis.

OBJECTIVES

Use the newly developed system called electromagnetic thermoablation (EMTA) to treat oral cancers of dogs and to investigate clinical outcome after EMTA.

METHODS

Seven client-owned dogs with oral tumors were treated with EMTA. These tumors included 3 malignant melanomas at buccal mucosa, tongue, and hart palatine, respectively; 2 amelanotic melanomas at tongues; and 2 squamous cell carcinomas at buccal mucosa and tongue, respectively. The heat of needle reached to 90-100°C in 30 seconds and then was maintained this temperature for 270 seconds to complete a 300-second procedure. The effective area of each needle after heating was 5 mm in radius from center of needle.

RESULTS

Six tumors showed no recurrence after EMTA. The mean relapse-free interval was 327.5 days (median, 305.5 days) to the date of this abstract submitted. Only one dog with large-sized malignant melanoma at root of tongue showed recurrence at 25th day after EMTA. Side effects were self-limited and included regional pain, salivation or discharge. These dogs were well tolerated and showed good appetite after EMTA.

CONCLUSIONS

This EMTA might be used as an option of the local disease control in oral tumors of dogs, similar with the wide-margin surgery.

WSVA8-0143

ONCOLOGY - ONCOSURGERY

MINIMALLY INVASIVE THERMOABLATION AS A NOVEL THERAPY FOR MALIGNANT NASAL TUMORS IN TWO DOGS

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INTRODUCTION

Nasosinal tumors account for approximately 1% of all canine tumors. Thermotherapy has been a method for tumor treatment by direct killing of tumor cells with coagulative necrosis. Minimally invasive thermoablation (MITA) is a new modality applies a high temperature on the specific part of the needle to treat target tumors.

OBJECTIVES

To evaluate the effects and side effects of MITA used in treating malignant nasal tumors in dogs.

METHODS

Two client-owned dogs with nasal melanoma and transitional carcinoma respectively. MITA was performed with computed tomography scan guidance under general anesthesia every 2-3 weeks. The temperature reached to 90-100°C in 60 seconds and then was maintained for 240 seconds to complete a 300-second procedure. The effective area of each needle after heating was 7.5 mm in radius from center of needle.

RESULTS

The dog with nasal melanoma had complete remission after twice MITA and showed no relapse for 445 days to the date of this abstract submitted. The dog with nasal transitional carcinoma had received 8 times of MITA and showed partial remission with progression-free interval of 407 days. Side effects were self-limited and included sneezing, nasal discharge. No major side effects were observed.

CONCLUSIONS

MITA is an effective, promising treatment of canine nasal tumor with minimal side effects.

WSVA8-0174

ONCOLOGY - ONCOSURGERY

EVALUATION OF LIQUID BIOPSIES TO DETECT CIRCULATING TUMOR DNA IN DOGS WITH CUTANEOUS MAST CELL TUMORS

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INTRODUCTION

Mast cell tumor (MCT) is the most common skin tumor affecting dogs, therefore improved diagnosis and prognosis of MCTs is crucial. Approximately 20% of MCTs are positive for a *c-kit* mutation. Such tumors exhibit more aggressive behavior but on the other hand can be specifically targeted by tyrosine kinase inhibitors. Detection of the mutation currently requires testing on tissue samples. Detection of *c-kit* gene and mutation in liquid biopsies would provide a less invasive technique for diagnostic, therapeutic and prognostic purposes.

OBJECTIVES

Evaluate blood and urine from dogs with MCTs to detect *c-kit* and its internal tandem duplication mutation in exon 11 compared to control dogs.

METHODS

Cutaneous MCTs were removed and blood and urine were collected from eleven dogs; liquid biopsies were also collected from five healthy, control dogs. Qiagen QIAamp® kits were used to extract DNA from tissue samples and liquid biopsies, and gel-based PCR was completed for detection of *c-kit* mutation.

RESULTS

c-kit gene was detected in all biopsy samples. It was also detected in the plasma of all dogs, including the control group, and in urine of 80% of the dogs with MCTs. Two of the eleven dogs with MCTs were positive for the mutation in biopsy samples, but *c-kit* mutation in the corresponding liquid biopsies was not detected.

CONCLUSIONS

The detection of circulating *c-kit* gene in plasma was successful; however, with the methods used in this study, it was not beneficial for diagnosis. Detection of the mutation in liquid biopsies was not accomplished.

WSVA8-0107

ONCOLOGY - ONCOSURGERY

DOG MASTOCYTOMA AND MAMMARY TUMORS FATTY ACID PROFILE: POSSIBLE IMPLICATIONS FOR TUMOR GRADING AND PROGNOSIS

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INTRODUCTION

Lipid metabolism has been accepted as a major metabolic pathway that is involved in many aspects of cancer cell pathogenesis. Attempts to understand the role of different lipids in cancer physiology depend on the ability to accurately monitor alterations in lipid composition at cell level due to the diversity of lipid classes.

OBJECTIVES

Mastocytoma and mammary gland tumor are common tumors in pet veterinary medicine. We investigated the differences in fatty acid profile of tumor and normal tissue sample in attempt to get better insight into the lipid metabolism of tumor cells as well as to improve grading of studied tumors.

METHODS

Paired samples of tumor and adjacent non-tumor tissue were isolated from 21 mastocytoma and 37 mammary tumors. After the pathohistological examination of tumor and non-tumor samples, fatty acid profile was determined by GC-MS.

RESULTS

All tumors showed significant difference compared to the normal tissue. The most significant differences were the increased content in C20:4n6 in both tumors, the increased content in 18:0, 18:1n9, 22:4n6 and 22:5n3 for mastocytoma and the increased content of 18:1n7, 20:3n6, C22:4n6 and C22:5n6 for the mammary tumors.

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Arachidonic acid
(C20:4n6)
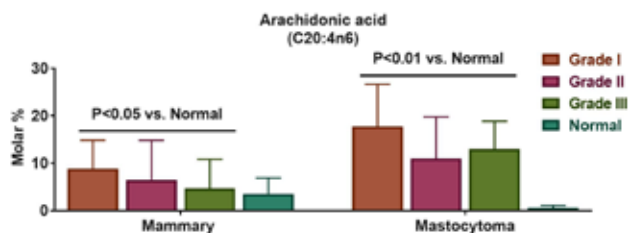
CONCLUSIONS

These preliminary results showed significant variations in fatty acid profile between tumor and non-tumor tissue as well as within tumor tissues depending on tumor type. Further researches should address the correlation between alterations in the fatty acid profile of different lipid classes and tumor prognosis.

WSVA8-0178

ONCOLOGY - ONCOSURGERY

EXPRESSION OF LEPTIN AND LEPTIN RECEPTOR IN CANINE MAMMARY TUMORS AND CORRELATION WITH OBESITY

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INTRODUCTION

Obesity is already considered as risk factor for mammary neoplasms in woman and female dogs. Leptin, a major protein that increases in concentration in obesity, may promote carcinogenesis of the mammary tissue through its interaction with the leptin receptor.

OBJECTIVES

In the present study, we analyzed leptin and leptin receptor (OBr) expression in canine mammary carcinomas (CMC) by immunohistochemistry and correlated with body condition score (BCS) and age of the bitch, tumor size and histological grade. Tumor size, age and histological grade were also related with BCS.

METHODS

The subjects were divided on the basis of their BCS: lean, optimal bodyweight, overweight or obese. Eighty-four bitches and ninety-five CMC specimens were randomly selected for the study.

RESULTS

Following microscopic examination, the samples were categorized as complex carcinomas (41% - 39/95) and simple carcinomas (59% - 56/95). Concerning to the body condition score (BCS), 9.52% bitches were lean (8/84), 29.76% bitches had optimal bodyweight (25/84), 33.33% had overweight (28/84) and 27, 39% bitches were obese (23/84) at the time of mastectomy. Obese animals presented mammary carcinomas at younger age and had larger tumors when compared to other body condition score (BCS). Leptin expression was not related to BCS; however, expression of ObR tended to be higher in tumors of bitches with higher BCS. Leptin expression was higher in older animals.

CONCLUSIONS

In conclusion, carcinomas of older animals expressed leptin and obese dogs presented mammary tumor at young age. Besides this larger carcinomas presented higher Obr expression

WSVA8-0180

ONCOLOGY - ONCOSURGERY

P53 OVEREXPRESSION IS ASSOCIATED WITH TO SHORTER SURVIVAL TIME OF BITCHES WITH MAMMARY TUMOR

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INTRODUCTION

Mammary tumor is the most important neoplasm in intact female dogs. Mutations in p53 tumor suppressor gene lead to overexpression of p53 protein in mammary neoplasms and have been associated a worse prognosis in women, but its prognostic value in canine mammary tumors (CMT) is controversial.

OBJECTIVES

In the current study we evaluate the p53 expression in CMT and verify its correlation with tumor features and overall survival (OS), also evaluating the disease free interval (DFI) and tumor development interval in the mammary gland remnant (TDIMG) of bitches submitted to partial mastectomy.

METHODS

One hundred bitches were followed up for a minimum of two years. Determination of p53 expression was performed by immunohistochemistry using the streptavidin-biotin-peroxidase technique. Samples with more than 10% of labeled neoplastic cells were considered positive, and percentage and marking intensity were evaluated.

RESULTS

P53 overexpression was observed in 65% (65/100) of the tumors. There was a correlation between p53 overexpression and histological subtype, which was higher in tubular and complex carcinomas (p = 0.017), and a higher marker score and higher labeling intensity was observed in high histological grade. However, there was no correlation between p53 expression and DFI (p = 0.441) and TDIMG (p = 0.240). The age of the dog at diagnosis was a factor that also influenced OS, being worse in bitches with more than eight years (p = 0.023).

CONCLUSIONS

This study demonstrated that the p53 overexpression in CMT should be considered as an important prognostic factor, related to a worse survival.

WSVA8-0108

ONCOLOGY - ONCOSURGERY

RESECTION OF INSULINOMA USING BIPOLAR VESSEL-SEALING DEVICE IN A DOG

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INTRODUCTION

Tumors of pancreatic corpus are relatively rare and more difficult to remove than those of other pancreatic site with conventional ligating methods(CLM). In this case, pancreatic tumor located in pancreatic corpus was successfully resected using bipolar vessel-sealing device(BVSD).

OBJECTIVES

This case is to evaluate the prognosis after surgical resection using BVSD for pancreatectomy in insulinomas.

METHODS

An 11years old, 7.1kg, spayed female, Italian greyhound was referred to Chonbuk Animal Medical Center with ataxia. The result of serum chemistry test consistently revealed hypoglycemia. In Computed Tomography (CT), mass of pancreatic body about 10 x10mm was detected. Increased insulin concentration was revealed in immunoreactive insulin test.

After detecting dark and firm mass at pancreatic corpus, partial pancreatectomy was performed by using BVSD. Intraoperative complications were not observed. In postoperative histopathologic examination, pancreatic mass was definitely diagnosed with insulinomas.

RESULTS

Postoperative blood analysis showed normal glucose level. Although the patient had vomit on the day of surgery, clinical sign was improved after medical therapy. Since that time, the patient has been doing well without any complications.

CONCLUSIONS

Surgical resection using BVSD is safe and reduce intra/postoperative complications in insulinomas. This case suggested that BVSD could be good option in pancreatectomy.

WSVA8-0146

ONCOLOGY - ONCOSURGERY

FIBROUS DYSPLASIA IN THE MANDIBLE OF A DOG

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INTRODUCTION

Fibro osseous lesions are poorly defined lesions of the jaws and cranio-facial bones. The diagnosis is best arrived at through a combination of clinical symptoms, excisional biopsy and radiology.

OBJECTIVES

Present the diagnosis and treatment of the monostotic intraoral fibro osseous growth of the left mandible through surgical excision

METHODS

A young native Indian breed(Rajapalayam) aged 13 months was presented with a fast growing, painless, cauliflower like in appearance, non-ulcerating smooth surfaced and hard swelling in the left mandible and extending from the level of the canine to the fourth premolar tooth.

Radiographs of the left jaw showed diffuse radiolucent area involving the entire area of the mandible extending from the level of the canine to the fourth premolar tooth.

Excisional biopsy revealed fibro osseous dysplasia.

A left hemi-mandibulectomy was performed allowing a margin of one centimeter on either borders under xylazine-atropine-propofol-sevoflurane anaesthesia.

RESULTS

Histopathological studies revealed acanthotic nonkeratinised squamous epithelium with an underlying mass composed of bland spindle shaped cells surrounded by moderate amount of fibrovascular stroma and trabeculae of woven bone. The bony trabeculae show focal areas lined by single layer of osteoblasts.

There was no evidence of any dysplastic tissue.
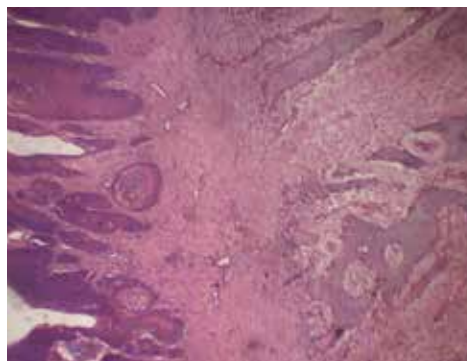
CONCLUSIONS

Identification and differentiation of benign fibro osseous proliferations like fibrous dysplasia from malignant osteosarcoma in a young canine patient is important from a prognostic perspective. Surgical resection en-mass provides quick and reliable relief to the patient.

WSVA8-0025

ONCOLOGY - ONCOSURGERY

INGNEOSA™: GAMIFICATION IN SMALL ANIMAL ONCOLOGY FOR VETERINARY UNDERGRADUATES

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INTRODUCTION

Educational games in digital learning environment has increasingly important role in the veterinary curriculum. InGNeoSA™ is an interactive e-gaming tool developed at the Faculty of Veterinary Medicine of Universiti Putra Malaysia to facilitate teaching and learning in small animal oncology.

OBJECTIVES

InGNeoSA™ is intended for final year DVM students. To play, students should have completed basic radiology, anatomy, physiology, canine and feline medicine and clinical pathology; histopathology and diagnostic imaging subjects which are relevant to clinical oncology.

METHODS

InGNeoSA™ was developed using the Unity© 2017.1, online 2D application based on oncology cases presented to the University Veterinary Hospital of UPM. It is equipped with background music, score database and time management elements; and players can compete and rank by scores. This game require specific login information which enable students to play and engage interactively at their convenience.

RESULTS

There are 6 Gaming Rooms which include: CANCHIST (history of cancer, metastasis, terminologies), TUMBIOL (differentials, fundamental cancer biology), GUESS THE TUMOUR (images of animals with tumour and likely diagnosis), IMAGINE (puzzles on diagnostic imaging), HISCYTO (histopathology, cytology), and CHEMOX (clinical pathology, chemotherapy, toxicities). Each room has a total score of 100 which can be used as assessment.

CONCLUSIONS

InGNeoSA™ is a useful tool to facilitate learning in small animal oncology in the DVM curriculum. This game make student centered learning fun, time saving, visually appealing, reduce utilization of printed materials in addition to tapping student's affinity towards the use of media technology in learning oncology. Students found the game very colourful, informative, exhilarating and engaging!

WSVA8-0152

OPHTHALMOLOGY

VISUAL PERFORMANCE OF HYDROPHILIC AND HYDROPHOBIC MUTLIFOCAL INTRA OCULAR LENS (IOL) IMPLANTATION AFTER ENDOPHACOEMULSIFICATION IN DIABETIC AND NON DIABETIC CATARACTOUS DOGS

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INTRODUCTION

Phacoemulsification lens extraction and artificial intraocular lens (IOL) implantation offer a favorable success rate and is considered as the most appropriate technique for the treatment of cataract. Endophacoemulsification is a more refined method of phacoemulsification.

OBJECTIVES

» To study the pre operative, intra operative and post operative variables.
» To compare the incidence of posterior capsular opacification (PCO).
» To select suitable IOL specific to diabetic or nondiabetic dogs.

METHODS

A period of 24 months from March 2013 to February 2015 to the Small Animal Ophthalmology unit of the Madras Veterinary College Teaching Hospital, Chennai, India were screened for the incidence and stage of cataract. A total of 24 cases were included in the study. The cases were divided into 2 groups, group I and group II of 12 each and each group was again sub divided into subgroup A (nondiabetic) and subgroup B (diabetic) consisting of 6 dogs each.

RESULTS

Highest incidence was recorded in Spitz (34%). Gender wise males had more incidence (58.49%) compared to the female dogs. The age group of 6 to 10 years had the highest incidence of 40.25 per cent. An improved grade scores in dogs implanted with multifocal hydrophobic IOLs were seen.

CONCLUSIONS

From the study it could be concluded that the lower PCO rates were observed in hydrophobic acrylic IOLs when compared with the hydrophilic acrylic IOLs. Nondiabetic and diabetic dogs with multifocal hydrophobic IOLs had better improvement when compared with multifocal hydrophilic lenses, due to fewer complications in the acute postoperative period (80 days).

WSVA8-0154

PALLIATIVE RADIOTHERAPY IN COMBINATION WITH EXENTERATION AND PARTIAL ORBITECTOMY FOR ORBITAL CHONDROGENIC OSTEOSARCOMA IN A COCKER SPANIEL

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INTRODUCTION

Osteosarcoma commonly was radioresistant tumor. Exenteration with partial orbitectomy were recommended for orbital osteosarcoma. No report of radiotherapy in combination with exenteration and partial orbitectomy for canine orbital osteosarcoma has been published.

OBJECTIVES

To present the efficacy of surgery with palliative radiation therapy for treatment of orbital chondrogenic osteosarcoma in a Cocker Spaniel.

METHODS

A 11-year-old female Cocker Spaniel dog presented with a history of globe displacement at the right eye for 3 months. Ophthalmic examination revealed exophthalmos, third eyelid protrusion and increased intraocular pressure OD. Computed tomography finding revealed mass effect at right retrobulbar, zygomatic, and temporal area. Right zygomatic and temporal bone lysis were seen. Cytological examination revealed spindle cell carcinoma. Physical examination, hematology and blood chemistry results were in normal limits. Exenteration with zygomatic arch removal was performed.

RESULTS

During surgery, a firm 10 cm 2 lobe mass adhesive with mandible was found to locate at retrobulbar area OD. Histopathological finding revealed chondrogenic osteosarcoma. Palliative radiotherapy with the 6 fractions of 6 Gy on days 0, 7, 14, 21, 28 and 35 was performed. At one, four, eight and eleven months follow-up after radiotherapy, the mass was gradually reduced in size and finally stable. The dog has been alert with no enlargement of the orbital mass for more than one and a half-year follow-up period.

CONCLUSIONS

Exenteration and partial orbitectomy combined with palliative radiotherapy was effective for orbital chrondrogenic osteosarcoma management in this dog for at least one and a half year.

WSVA8-0188

OPHTHALMOLOGY

B-MODE ULTRASONOGRAPHIC ASPECTS AND BIOMETRY OF LEOPARD CAT (PRIONAILURUS BENGALENSIS)

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OBJECTIVES

To describe the normal ultrasonographic appearance and biometry of the leopard cat's eye (Prionailurus bengalensis) using B-mode ultrasonography.

METHODS

8 leopard cats (Prionailurus bengalensis) from Chiangmai night safari with no history of previous ophthalmic disease were examined using the portable ultrasound machine with high frequency curvilinear transducer (10 MHz) in order to describe normal sonographic appearance and biometry measurement. These captive wild cats were premedicated and inducted with xylazine and zoletil, respectively. The anesthesia was maintained with isofurane.

RESULTS

The biometric findings were as follows; mean and standard deviation (Mean+SD) of the corneal thickness was 0.07+0.0006 cm Depths of the anterior and posterior chamber were 0.34 + 0.009 cm. Lens thicknesses were 0.81 + 0.014 cm. Depths of the vitreous chamber were 0.60 + 0.009 cm. No significant difference was found between the ocular biometry of the left and right eyes.

CONCLUSION

This study provides ultrasonographic appearance and baseline information of ocular biometry in leopard cat for further clinical investigations of ocular abnormalities using B-mode ultrasonography.

WSVA8-0077

ORTHOPEDICS

IN VITRO STUDY OF DEMINERALIZED FREEZE-DRIED BONE ALLOGRAFT STERILIZED BY GAMMA RADIATION AS BONE FILLER FOR COMMINUTED FRACTURE IN FELINE PATIENT

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INTRODUCTION

The incidence of fracture in cat, especially stray cats, is quite high in Indonesia. Among the type of fractures, the comminuted fracture is still considered challenging to handle. In this case, the demineralized freeze-dried bone allograft (DFDBA) can be an option to replace the missing part of the bone.

OBJECTIVES

This research is aimed to evaluate cell viability of DFDBA by in vitro study using MTT assay.

METHODS

The demineralized freeze-dried bone allograft (DFDBA) was produced from feline (cadaver) bones. The long bones of extremities were used as raw materials. Both cancellous and cortical bones were collected and processed into 3 different sizes of 20, 40, and 60 mesh for each type of the bone. Sterilization of DFDBA was conducted using gamma radiation with doses of 15kGy and 25kGy to each type and size material produced resulting in 12 DFDBA variants. The bone graft produced then evaluated with the MTT assay using CPAE cells in D-MEM.

RESULTS

The results show that the highest cell viability (80,2%) lead by DFDBA derived from cortical bones with 20 mesh in size and 15kGy radiation dose. Followed by 20 mesh DFDBA derived from cortical bone sterilized by 25kGy with 72,8% cell viability.

CONCLUSIONS

From these results, we concluded that the CPAE cells growth was affected by the type of the bone, particle size, and radiation dose. The highest cell viability (lowest cell growth inhibition) was found in 20 mesh DFDBA derived from the cortical bone sterilized by 15kGy gamma radiation.

WSVA8-0081

ORTHOPEDICS

LONG-TERM EFFECTS OF WHOLE BODY VIBRATION EXERCISE ON RENAL, LIVER AND MUSCLES PARAMETERS IN HEALTHY DOGS

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INTRODUCTION

Whole-body vibration exercise (WBVE) is used for rehabilitation and treatment of some conditions in human patients. However, safe WBVE protocols must be investigated to prevent deleterious conditions particularly in long-term WBVE. Studies regarding to effects of long term WBVE in dogs are poor in the literature.

OBJECTIVES

The aim of the study was to investigate the long term effects of WBVE on serum biochemistry (renal, liver and muscles parameters) in adult healthy dogs.

METHODS

Ten clinically healthy beagle dogs, three females and seven males, aged from 1 to 5 years, weighing from 8 to 14 kg were evaluated. Exclusion criteria included use of medications or supplement, or previous surgical treatment. The dogs standing up on all four feet on a vibrating platform, and sessions of 30 Hz for 5 min, followed by 50 Hz for 5 min and finishing with 30 Hz for 5 min was performed *once a day for 5 days*. The velocity and amplitude of the vibration platform were 12-40 m/s2 and 1.7-2.5 mm, respectively. Jugular blood samples were collected, before and immediately after the WBVE session, 1 and 6 hours after the end of each session for five days, and 24 hours and 48 hours after the last WBVE session. Alanine aminotransferase, aspartate aminotransferase, creatine kinase, blood urea nitrogen, creatinine, serum total protein were the data analyzed.

RESULTS

The data analyzed did not show significant variation during the study.

CONCLUSIONS

In conclusion, the protocol adopted was considered adequate in relation to renal, liver and muscles serum biochemistry parameters.

WSVA8-0187

ORTHOPEDICS

TREATMENT OF PELVIC CANAL NARROWING BY REMOVAL OF THE BONE FRAGMENTS DISPLACED VIA PELVIC CANAL

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INTRODUCTION

Depending upon the severity of the pelvic fractures, the conservative management can lead to complications such as compromise of the pelvic inlet or outlet, degenerative joint disease, and a non-functional limb.

OBJECTIVES

The purpose of this report was to describe the surgical procedures used to treat pelvic canal narrowing.

METHODS

Two male crossbred dogs were admitted due to severe constipation secondary pelvic fracture managed conservatively. The dogs had been treated with enemas, and laxatives without success. Digital rectal examination and survey radiographs confirmed a pelvic stenosis of more of 60% due to chronic malunion fractures. In both cases, contrast radiography revealed a narrowing of the descending colon at the level of fracture malunion. The surgical procedures in both cases included a celiotomy and removal of the bone fragments displaced medially into the pelvic canal using bone chisel and hammer, and goiva. Excision of the femoral head that was located into the pelvic canal was necessary in a dog. All procedures were done via pelvic canal.

RESULTS

The dogs did not show episodes of obstipation after the surgery, and limb function was maintained.

CONCLUSIONS

In conclusion, removal of the bone fragments displaced via pelvic canal is an option to treat pelvic stenosis

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WSVA8-0037

ORTHOPEDICS

TREATMENT OF COMMINUTED FRACTURE USING TITANIUM MESH AND BIPHASIC CALCIUM PHOSPHATE CERAMIC SCAFFOLD IN CANINE

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INTRODUCTION

Reconstruction and fixation of bilateral mandibular fracture required a good dental occlusion and normal mastication function after the healing. Several techniques have been reported in canine. However, there is no consensus method for fixation of comminuted fracture of the mandible when dealing with bone loss.

OBJECTIVES

To study the feasibility of using titanium mesh and biphasic calcium phosphate ceramic scaffold (BCP) in fixation of the comminuted fracture of canine mandibles.

METHODS

A canine patient with bilaterally comminuted fracture of the mandibles was presented to the Kasetsart University Veterinary Teaching Hospital for treatment. The CT images were obtained and preprocessed to construct the 3D image of the skull. The images were transferred to rapid prototyping machine to create a resin skull model for surgical planning. Titanium meshes and 2-mm titanium miniplates were reshaped and contoured to match the morphology of the resin mandibles. Both implants were used to neutralize tensile and compression forces of the mandible during mastication. For each mandibular ramus, the 2-mm miniplate and screws were placed along the alveolar border onto the tension surface. The ventral border of each mandible was stabilized using titanium mesh and screw fixation to counteract compression force. The BCP scaffolds were filled in areas of bone loss underneath the titanium meshes.

RESULTS

There were no post-operative complications detected. Clinical bone healing was obtained with excellent dental occlusion and normal mastication function within 6-months period.

CONCLUSIONS

Titanium mesh and biphasic calcium phosphate ceramic scaffold can be utilized in fixation of the comminuted fracture of canine mandible.

WSVA8-0095

REPRODUCTION, PEDIATRICS

COMPARISON OF VAGINAL BACTERIA OF CATS IN ESTRUS AND INTERESTRUS

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INTRODUCTION

A variety of organisms inhabit the reproductive tract of cats and is believed to be affected by stages of the estrous cycle.

OBJECTIVES

This study aimed to determine the relationship of estrus and interestrus phases with aerobic bacterial culture of the vagina of cats.

METHODS

Samples were obtained from twenty-six intact, female, Domestic Shorthair cats aged 1-year-old or older from two areas in the state of Selangor. Sterile cotton swabs were used for sample collection for vaginal cytology and bacterial culture. Three animals were in estrus while 23 were in the interestrus stage. Blood and MacConkey agar were used for bacterial culture.

RESULTS

Gram negative bacteria were isolated only from interestrus cats while gram positive bacteria were isolated from both estrus and interestrus cats. Amongst all the animals, the most aerobic bacteria isolated were *Staphylococcus intermedius* (26%), *Streptococcus canis* (26%), *Escherichia coli* (14%) and *Staphylococcus pseudintermedius* (12%). There was no association observed between aerobic bacteria cultured from cats in estrus and interestrus.

CONCLUSIONS

Types of bacteria cultured from the vagina of cats were not related to the phases of estrus and interestrus.

WSVA8-0098

REPRODUCTION, PEDIATRICS

CLINICAL PRESENTATION, DIAGNOSIS, TREATMENT AND OUTCOME OF 77 DOGS AND 137 CATS WITH PYOMETRA

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INTRODUCTION

Pyometra is the accumulation of purulent material in the uterus and is life-threatening to intact female dogs and cats.

OBJECTIVES

This study aimed to determine the clinical signs at presentation, diagnostic methods, treatments and outcomes in bitches and queens with pyometra.

METHODS

Medical records of 77 bitches and 137 queens presented to the Universiti Veterinary Hospital (UVH), Universiti Putra Malaysia (UPM), Malaysia with pyometra between 2008 – 2017 were evaluated.

RESULTS

The median age at presentation for bitches was 96 months and 18 months for queens. Most patients experienced estrus more than 4 weeks prior to presentation and were presented more than 7 days after the first clinical sign. The most prominent clinical sign was vaginal discharge (bitches=74.0%; queens=85.4%). Blood parameters showed leukocytosis (bitches=53.2%; queens=35.0%) with neutrophilia and monocytosis. Alkaline phosphatase was seen to be increased in bitches (55.2%). Ultrasonography had higher confirmatory percentages (bitches=93.1%; queens=78.3%) than radiography (bitches=58.7%; queens=72.3%). Ovariohysterectomy with concurrent antibiotics was the treatment of choice in 61% of cases. Post-operative complications occurred in 8.48% of patients. The average length of hospitalization was 4.5 days for bitches and 3.89 days for queens and was associated with decreases in haematocrit (P=0.022) and increases in blood urea nitrogen (P=0.014). Correlation was seen between days before surgery was performed, length of hospitalization and survival of patients (P=0.028).

CONCLUSIONS

Clinical signs at presentation, diagnostic methods to confirm pyometra and treatment modalities were similar for bitches and queens. Reduction in time to surgery and hospitalization length could potentially improve patient survival.

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WSVA8-0020

REPRODUCTION, PEDIATRICS

SEX DETERMINATION OF CANINE SPERMATOZOA BY SYBR® GREEN QUANTITATIVE PCR

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INTRODUCTION

Effective preselection of sex has been done in various species of livestock and domestic animals using the flow cytometry. A guaranteed high sorting accuracy is a key performance for the widespread use of sperm sexing. However, the sexing technique needed to be validated to ensure the accuracy of the technology.

OBJECTIVES

To be able to specifically produce either male or female offspring in the dog, we developed a technique to determine the sex of canine spermatozoa using SYBR® Green real-time quantitative PCR (qPCR).

METHODS

Two sets of primers, ZFX and SRY were designed specifically for X- and Y- chromosome canine genes respectively. Plasmid was inserted with ZFX and SRY gene fragment separately to create standard curves that ranged from 300 to 3,000,000 copies. In canine genome, both ZFX and SRY genes were exist as single copy number and commonly used as a DNA marker in sex determination.

RESULTS

Real-time qPCR of bovine spermatozoa DNA samples and cloned plasmid ZFX and SRY genes for creating standard samples were performed simultaneously. There was no significant difference in percentages of between the theoretical ration (1:1) and unsexed X- and Y- chromosome-bearing canine spermatozoa in any of the nine dogs. In addition, the mean purities of sorted sex chromosomes in spermatozoa of the nine dogs were 91% for the X chromosome fraction and 90% for the Y chromosome fraction.

CONCLUSIONS

This technique was a rapid and reliable technique in quantifying the sex ratio of X- and Y- chromosome bearing spermatozoa in semen sample.

WSVA8-0104

REPRODUCTION, PEDIATRICS

A COMPARATIVE STUDY OF FETAL HEAD DIAMETER MEASURED BY RADIOGRAPHY AND ULTRASONOGRAPHY IN DOGS AND CATS

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INTRODUCTION

Radiography and ultrasonography are both widespread methods used in veterinary obstetrics. In a ventro-dorsal radiographic view, measurements of fetal biparietal or head diameter (HD) and maternal pelvic diameter are commonly used to predict the risk of dystocia in dogs and cats due to relatively oversized fetus. However, HD measurement is easier to perform with ultrasound scan.

OBJECTIVES

To compare and find the correlations of fetal HD measured by radiography and ultrasonography in dogs and cats.

METHODS

Twenty seven dogs and seventeen cats were diagnosed in last trimester of pregnancy at Kasetsart University Veterinary Teaching Hospital during 2017-2018. Fetal HD was measured by radiography and transabdominal ultrasonography on the same day. The correlations between two techniques and linear regressions of HD were statistically analyzed.

RESULTS

Radiographic measurements of fetal HD were larger than those measured by ultrasonography in both dogs (P<0.0001) and cats (P<0.0001). The correlations were significant (r = 0.85 in dogs and r = 0.94 in cats, p<0.0001). Linear regression formulas were y = 1.0716x + 0.1661 (R^2 = 0.72) in dogs, and y = 0.9627x + 0.3148 (R^2 = 0.88) in cats. (y= HD by radiography, x= HD by ultrasonography)

CONCLUSIONS

Fetal HD measurements were different between two modalities. The formulas above can be implied to estimate relatively oversized fetus.

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WSVA8-0050

REPRODUCTION, PEDIATRICS

APPLICATION OF DOPPLER ULTRASONOGRAPHIC ASSESSMENT OF MATERNAL AND FOETAL BLOOD FLOW IN CANINE PREGNANCY

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INTRODUCTION

Doppler ultrasonographic assessment of maternal and foetal blood flow has become an indispensable diagnostic tool in veterinary obstetrics and gynaecology.

OBJECTIVES

To evaluate blood flow in uteroplacental artery(UPA), umbilical artery(Uma) and foetal abdominal aorta by measuring pulsatility (PI) and resistive indices (RI).

METHODS

Doppler examinations were conducted from fourth week of pregnancy at 10 d interval in twenty apparently healthy bitches of different breeds presented at University Veterinary Hospital, Thrissur, Kerala.

RESULTS

Throughout the study the UPA showed biphasic wave. Up to 40 days of gestation the UPA showed diastolic notch and from 41-50 days it completely disappeared. The PI and RI decreased significantly at 5 per cent level between each 10 d interval. Similarly, the PI and RI of Uma decreased significantly at 5 per cent level between each 10 d interval. The PI of foetal abdominal aorta increased significantly at 5 per cent level from ≤ 30 d to 31 – 40 d of gestation and thereafter it decreased significantly up to whelping. During early pregnancy the diastolic wave form was absent for Uma and abdominal aorta although at later stages both these vessels showed biphasic wave forms. The study also included the diagnosis of an incomplete abortion in a Rottweiler bitch by Doppler ultrasound and its critical management. Fourteen days from the date of the incomplete abortion of three puppies, the bitch whelped five healthy puppies.

CONCLUSIONS

The study demonstrated the potential applicability of Doppler Ultrasound in the management of abnormal pregnancy and predicting survival chances of neonates after whelping.

WSVA8-0019

REPRODUCTION, PEDIATRICS

ABNORMAL FREQUENT ESTRUS AND PROLONGED VULVA SWELLING IN YOUNG DOG

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INTRODUCTION

Granulosa cell tumor is a primary ovarian neoplasia in the dogs which normally occur in elderly bitches with a median age of 12 years old.

OBJECTIVES

This study aims to present the discovery of granulosa cell tumor and ovarian cyst in young dog that they have not been reported yet.

METHODS

A 3-year-old, female Yorkshire terrier, was referred to Theriogenology Center at the Kasetsart University Veterinary Teaching Hospital, Bangkok, Thailand with clinical signs of vulva swelling for 2 years, since she had her first estrus until present time, and sign of abnormal frequent estrus every 2-3 months. Complete blood count examination, vaginal cytology and ultrasonography were performed. However, client denied to measure levels of serum sex hormones.

RESULTS

The results showed normal blood parameters from complete blood count. Vaginal cytology showed about 20% cornification vaginal epithelial cells which it indicated the influence of estrogen. Endometrial hyperplasia and cystic lesion on right ovary were found from ultrasonographic images. The dog was diagnosed as ovarian cyst on right ovary. Surgical correction was performed by ovariohysterectomy.

For further diagnosis, ovarian tissues from both ovaries were collected and submitted for biopsy. The histopathological results revealed that the dog had non-functional ovarian cyst, cystic subsurface epithelial structure, on the right ovary, and granulosa cell tumor with the present of monolayered clusters of cells and acinar to tubular pattern on the left ovary.

CONCLUSIONS

This is the case demonstrating the discovery of granulosa cell tumor and ovarian cyst in young dog which it is quite a rare case.

WSVA8-0089

SOFT TISSUE SURGERY

CLINICAL FEATURES OF PHRENIC VESSEL-RELATED CONGENITAL EXTRAHEPATIC PORTOSYSTEMIC SHUNTS IN 5 DOGS

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INTRODUCTION

Phrenic vessel-related portosystemic shunt (Pr-PSS) is one of the major types in congenital extrahepatic portosystemic shunts (cEHPSS) in dogs. Its morphological characteristics have been well described in several studies. However, there are only few articles on clinical features of Pr-PSS in dogs.

OBJECTIVES

To describe clinical features of Pr-PSS in 5 dogs and compare the characteristics with gastrocaval and splenocaval types of cEHPSS patients in the same institution from August 2015 to February 2018.

METHODS

Medical and surgical records were retrospectively analyzed for clinical presentation, history, physical examination findings, clinicopathologic data, diagnostic imaging findings, medical and surgical treatment, surgical complications, and outcome.

RESULTS

All five patients were non-Maltese breed dogs, while 9/10 (90%) of dogs of portocaval and gastrocaval cEHPSS were Malteses. Two patients who were diagnosed less than one-year-old had congenital abnormalities including hydrocephalus, patent ductus arteriosus, cryptorchism, ureterocele, and ectopic ureter. Two patients showed no evidences related to cEHPSS while three patients presented with neurological signs including abnormal blood examination results. Older patients had age-related diseases such as cardiac disorders or liver mass. There was a major iatrogenic hemorrhage of a shunt vessel in a surgical procedure. In this case, the shunt had to ligate completely to control the bleeding. The patient had no signs of portal hypertension and all patients recovered well.

CONCLUSIONS

It is beneficial to perform computed tomography for non-Maltese small breed dogs with other congenital abnormalities to rule out or diagnose Pr-PSS due to the subclinical characteristics, diagnostic challenge with abdominal ultrasonography, and high prevalence

WSVA8-0004

SOFT TISSUE SURGERY

CASE REPORT: SURGICAL TREATMENT OF DUODENOGASTRIC INTUSSUSCEPTION WITH PANCREATIC INVOLVEMENT IN A DOG

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INTRODUCTION

An Intussusception of gastrointestinal tract is uncommon in adult dogs and duodenogastric intussusception with pancreatic involvement is very rare form in veterinary medicine. There is considerable variation in etiology and most of the cases are idiopathic. The prognosis and treatment outcome are variable depending on the severity of vascular compromise, abdominal organ association and underlying causes.

OBJECTIVES

A 6-year-old intact female crossbred dog was referred because of 4-days history of vomiting and anorexia. The dog was diagnosed of pancreatitis with severe azotemia. Repeated ultrasonography revealed typical views of an duodenogastric intussusception.

METHODS

Within midline celiotomy, the intussusception was identified. The pylorus, proximal duodenum and the segment of right pancreatic limb were prolapsed into the stomach. The intussusception was reduced manually and a duodenopexy was performed. There was no polyp in the gastropyloric outflow and the thickening pyloric region was performed a full-thickness biopsy.

RESULTS

The dog recovered unevenfully from surgery and the biopsy result revealed submucosa edema without an evidence of malignancy. Upon follow-up, the dog has had no evidence of recurrence of duodenogastric intussusception for over 1 year.

CONCLUSIONS

As a rarity of duodenogastric intussusception, this case is unique in the present of pancreatitis due to gastroduodenal outflow obstruction and pancreatic involvement. Severe vomiting is the identifiable condition being the predisposing factor in both terms of primary and expectable recurrent evidences of this intussusception. Manual reduction and duodenopexy were accomplished for treatment and recurrent prevention.

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WSVA8-0026

SOFT TISSUE SURGERY

CHOLECYSTODUODENOSTOMY IN COMMON BILE DUCT LEAKAGE AND BILE PERITONITIS IN 3-MONTH-OLD SIBERIAN

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INTRODUCTION

Bile peritonitis is a severe, nonseptic inflammatory response caused by leakage of bile, may occur with traumatic rupture of the extrahepatic biliary tree or secondary to necrotizing cholecystitis or chronic obstruction. Cholecystoduodenostomy is the one of procedures used to repair common bile duct injury.

OBJECTIVES

To identify and repair the leakage site of biliary tree and divert bile flow.

METHODS

A 3-month-old, Siberian was diagnosed with bile peritonitis that may have been caused by extrahepatic biliary tree injury which presenting ascites and anorexia. Blood profile and ultrasonography were assessed. Abdominal paracentesis and fluid analysis were performed. The peritoneal effusion was green colour and total bilirubin level was higher than in serum. An abdominal exploratory was performed on the patient which found adhesion, fibrosis and a leakage site at common bile duct. Leakage site was sutured, cholecystoduodenostomy and abdominal drainage was performed.

RESULTS

The patient was admitted for fluid therapy and ABO therapy. Peritonitis, bile leakage and dehiscene were monitored by colour and turbidity of the peritoneal fluid. The patient had hypoalbuminemia after sugery, was fed a low fat diet, did not show leakage or dehiscence and was discharged 10 days after surgery.

CONCLUSIONS

Bile peritonitis in a young dog is likely caused by trauma. Bile flow diversion is recommended when the common bile duct injury is severe and distal to the entrance of the hepatic duct. In this case report, It was small and the leakage site was adhesion and fibrosis and consequently cholecystoduodenostomy was performed. Ascending cholangiohepatitis may occur in the long term.

WSVA8-0106

SOFT TISSUE SURGERY

URETERONEPHRECTOMY IN DOG WITH NEPHROLITH AND URETEROLITH

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INTRODUCTION

Ureteronephrectomy has been performed in irreparable trauma, persistent infection, obstructive calculi with persistent hydronephrosis and etc.. Our case has severe renal failure due to bilateral nephrolithiasis, unilateral ureterolith. So we decide to perform ureteronephrectomy.

OBJECTIVES

To evaluate the good prognosis after ureteronephrectomy in dog nephrolith and ureterolith.

METHODS

A 8-years-old intact female shih tzu was referred to the Chonbuk Animal Medical Center with histories of vomiting, anorexia and lethargy.

In laboratory examination, ALP, GGT, TBIL, Cholesterol, BUN and CRP were increased, and ALB was decreased. In ultrasonography, left & right nephrolith, right kidney pelvic dilation, hydronephrosis, right ureterolith, right ureter dilation and gallbladder sludge was diagonsed.

Ureteronephrectomy was decided based on the results and performed general procedure at the right kidney and ureter.

After surgery, patient was treated with antibiotics, antiemetic drugs, analgesic drug and Peripheral Parenteral Nutrition.

RESULTS

After surgery, patient condition was improved remarkably. Following the decreased inflammatory level and returned to normal range hematologic figures, vomiting and lethargy was reduced. Voluntary ingestion had become possible. The patient was discharge one week after surgery.

CONCLUSIONS

From this case, Ureteronephrectomy in patient with severe renal failure due to bilateral nephrolithiasis and ureterolith was good option.

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SOFT TISSUE SURGERY

LOW DENSITY TUBULAR BOVINE COLLAGEN GRAFT FOR URETHRAL REGENERATION: PRELIMINARY CANINE MODEL

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INTRODUCTION

Patients with urethral stricture and hypospadias require urethral reconstructive surgery. The US Food and Drug Administration (FDA) requires the demonstration of safety and efficacy of medical device in at least 2 animal models before clinical translation.

OBJECTIVES

This study is aimed to evaluate the efficacy of a tubular bovine collagen graft in urethral regeneration in a canine model.

METHODS

A 4 cm long urethral graft was surgically implanted in an artificially created urethral defect proximal to the baculum in a pilot study involving 3 dogs. A transurethral urinary catheter was kept post-op.

RESULTS

Dog 1 had successful implant procedure but compromised by iatrogenic urinary catheter removal. The complications were urine leakage, scrotal excoriation and cystitis. Dog 1 able to micturate normally following urinary catheter removal on post op day 14. Post op day 75, Dog 1 was euthanized. Histological data showed narrowing of the urethra at the distal anastomotic site but patent urethra proximally. Both Dog 2 and 3 had catheters removed on post op day 10. They able to micturate normally. Ultrasound on post op week 6 revealed no stenosis. Contrast study on Dog C post op 5 months revealed patent urethra at the grafted site and narrowing of the urethra at the distal anastomotic site. No clinical micturition difficulty observed. To date (February 2018), Dog 2 and 3 are still under observation for long term graft complication.

CONCLUSIONS

This pilot study reveal canine is a promising model for urethral clinical translation studies and potential implant application in cases of canine urethral stenosis.

WSVA8-0102

SOFT TISSUE SURGERY

SURGICAL CORRECTION OF A THORACIC ESOPHAGEAL DIVERTICULUM IN A DOG

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INTRODUCTION

Esophageal diverticula are uncommon disorders in dogs, which may be classified in congenital or acquired.

OBJECTIVES

To describe esophageal diverticulum in 4.6 months of age, 17 kg, female German Shepherd dog.

METHODS

The dog was admitted due to episodes of emesis, apathy, and discharge nasal and ocular for 3 days. The owner suspected foreign body ingestion. Physical examination revealed hyperthermia and harsh lung sounds were heard. Survey radiography of the thorax radiographs showed a gas-filled large circular dilation (10.2 x 8.2 cm) located in the cranial mediastinum, esophagus filled with gas, and alveolar pattern compatible to aspiration pneumonia. A contrast radiographic study and computed tomography revealed esophageal diverticulum in cranial region of the thorax and mild megaesophagus along the thoracic esophagus. Esophageal endoscopy also confirmed the diverticulum, but mucosa surface had no abnormality. Left intercostal thoracotomy at the second space was conducted under general anesthesia. The diverticulum was identified, the diverticular pouch was resected, and restoration of the esophageal wall was done by manual two-layer closure.

RESULTS

A contrast radiographic examination performed 10 days after surgery showed no signs of suture-line leak, but a cranial residual portion of the diverticulum was present. A gastrostomy feeding tube was maintained during this post-operative period. The animal recovered without complications, and the clinical signs of dysphagia and pneumonia had ceased. After 2 months after surgery, contrast radiographic examination showed improvement of the megaesophagus. However, the owner was instructed to feed the dog in an elevated position.

CONCLUSIONS

Probably this esophageal diverticulum was associated with esophagus weakness.

WSVA8-0032

SOFT TISSUE SURGERY

SURGICAL CORRECTION OF BILATERAL AND VENTRAL OF PERINEAL HERNIA IN MALE DOGS WITH MODIFIED SACROISCHIAL SLINGS SUTURE

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INTRODUCTION

Perineal hernia (PH) is a complex disease in dogs. The recurrence of the PH after standard intervention often occurs and with more complicated problem involving bilateral with ventral PH. A suitable method to solve the bilateral with ventral PH is needed.

OBJECTIVES

To evaluate a new surgical technique for correction of bilateral with ventral PH using polyethylene (Leader line) in male dogs.

METHODS

Twelve dogs (Body weight mean 10.2+/- 5.6 kg) had swollen mass at both side of anus and alimentary tenemus. These dogs were diagnosed with bilateral with ventral PH using rectal palpation. Six of the twelve dogs had recurrent after correction with standard surgical technique and other six dogs were diagnosed with bilateral and ventral PH for the first time. The PH were treated with modified sacroischial suture (MSS), which covered the hernia defect. The lateral line of sacroischial suture were sutured to external anal sphincter muscle and connected from both sacroischial suture by lower line to cover the ventral defect of the pelvic floor. All dogs were regularly monitored for 2 years.

RESULTS

All dogs had no complication in the first 14 days after the operation. All dogs were free from bilateral PH without related complication throughout a 2-year period.

CONCLUSIONS

Results of the present study suggest that MSS is an effective surgical method for correction of bilateral with ventral PH in dogs, especially with a presence of thin or friable perineal diaphragmatic muscles.

WSVA8-0118

SOFT TISSUE SURGERY

SURGICAL OCCLUSION WITH CELLOPHANE BANDS METHODS IN CONGENITAL EXTRAHEPATIC PORTOCAVAL SHUNT

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INTRODUCTION

Surgical intervention has been recommended for congenital portosystemic shunt in dogs, but acute complete ligation of shunt is associated with life-threatening portal hypertension. To reduce the risk of portal hypertension, gradual vascular occlusion is needed. The use of cellophane bands incites inflammatory reactions associated with a chronic foreign body and leads to gradual occlusion of shunted vessel.

OBJECTIVES

To report outcome after cellophane banding of single congenital portosystemic shunt in 4-month-old dog.

METHODS

On radiography, microhepatica with smooth margination was observed. On computed tomography, there was shunt vessel. from portal vein to CdVC. On laboratory test, serum ammonia level was 76µmol/L. ALP, AST, and GGT level was elevated and ALB, BUN, CREA level was decreased.

Based on these results, it was diagnosed as congenital portosystemic single shunt. As a treatment, surgical occlusion of shunt was performed followed 2 weeks medical management with hepatoprotective therapy, antibiotics and oral lactulose.

The patient had a ventral median celiotomy and surgical time was 50 minutes. Portosystemic shunt which entered CdVC from portal vein was identified by abdominal exploration. Cellophane bands was placed as reported previously. The patient was monitored intensively for 48 hours after surgery to observe for postligation neurologic dysfunction or portal hypertension.

RESULTS

Two weeks after surgery, neurologic dysfunction of patient was recovered and postoperative serum ammonia concentration was decreased to 18µmol/L. There wasn't complications associated with surgical shunt attenuation without recurrence of clinical signs.

CONCLUSIONS

Cellophane banding is a safe and effective alternative to ameroid constrictors methods of attenuation for large portosystemic shunt.

WSVA8-0167

(CLINICAL) PATHOLOGY

ANGIOMYXOMA OF UNKNOWN ORIGIN IN THORACIC CAVITY OF A DOG - A CASE REPORT

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INTRODUCTION

An 8-year-old intact male French bulldog was presented to necropsy at the Pathology Department of University of Veterinary Medicine, Budapest. It was a case of sudden collapse and death the day before presentation. There was no illness in the dog's anamnesis.

OBJECTIVES

The necropsy was done to examine the cause of sudden death.

METHODS

During the necropsy, the following gross pathological lesions were seen. In the thoracic cavity we found 320 ml of mucinous red fluid and several gelatinous soft masses in connection with the pleura, pericardium and sternum. The lungs were collapsed due to the surrounding fluid. No other lesions were found in the body. Pieces of the masses were fixed in 8% neutral buffered formalin, and routinely processed and embedded in paraffin blocks. Sections of 3-4 µm thickness were obtained from the blocks and stained with haematoxylin and eosin (H.E.), periodic acid schiff (PAS), and later immunohistochemistry (IHC) was also performed.

RESULTS

With H.E. and PAS stainings we have seen several spindle-shaped cells in a myxoid stroma, surrounded by many small vessels. The cells were loosely arranged in the stroma. IHC staining with anti-Claudin-5, anti-α-SMA, anti-S-100, anti-CD31, anti-Ki-67 and anti-vimentin primary antibodies proved the diagnosis of angiomyxoma.

CONCLUSIONS

Angiomyxoma is a rare type of myxoid tumor in humans and animals. Only a few reported cases exist in veterinary medicine, none of them presented in the thoracic cavity. We report the first case of thoracic angiomyxoma as far as our best knowledge.

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WSVA8-0031

NUTRITION

ALTERATIONS IN GUT MICROBIOME BY THE PROTEIN TO CARBOHYDRATE RATIOS IN OBESE VS. LEAN DOGS

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INTRODUCTION

An estimated 54% of dogs in the US are overweight or obese. Dysbiosis in gut microbes has been associated with obesity in humans and animal models. High protein low carbohydrate (HPLC) diets have been recommended for body weight management for decades, but their effects on canine gut microbiota is not well understood.

OBJECTIVES

To evaluate the effect of protein and carbohydrate ratios on gut microbiome in obese vs. lean dogs.

METHODS

63 Labrador Retrievers and Beagles, half obese or overweight (OW) and half lean or normal (LN), were fed the common baseline diet for 4 weeks, followed by 4 weeks of experimental diet: HPLC (49.4% protein, 10.9% carbohydrate) or low protein high carbohydrate diet (LPHC) (25.5% protein, 38.8% carbohydrate). Fecal samples collected at the end of baseline and experimental diets were subject to 16S rRNA gene sequencing analysis.

RESULTS

Diets predominantly exerted their effects on the two phyla, *Bacteroidetes* and *Firmicutes*. The effects were greater in OW dogs than in LN dogs, but independent of breed. The HPLC-fed dogs had decreased *Bacteroidetes* to *Firmicutes* ratios, and enriched microbial gene networks associated with weight maintenance, when compared with those on LPHC. The abundances of *C. hiranonis*, *C. perfringens*, and *R. gnavus* were higher in the HPLC group, while *B. uniformis* and *C. butyricum* were enriched in the LPHC group.

CONCLUSIONS

This study provided an initial framework that will allow modulation of gut microbiota by nutrition interventions and may provide an alternative therapeutic option for canine obesity.

WSVA8-0169

NUTRITION

DOCOSAHEXAENOIC ACID IN MICROALGAE SCHIZOCHYTRIUM SP. IN A WET PET FOOD IS STABLE DURING RETORT AND 24 MONTHS OF STORAGE AT AMBIENT TEMPERATURE

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INTRODUCTION

Significant amounts of evidences have shown the health benefits of docosahexaenoic acid (DHA) in humans and animals. Dried microalgae *Schizochytrium sp.* is a sustainable and commercially available ingredient with a high concentration of bioavailable DHA.

OBJECTIVES

The objective of this study was to measure the stability of DHA in a wet pet food during retort and storage.

METHODS

The dried microalgae *Schizochytrium sp.* (DHAgoldTM S17-B, DSM Nutritional Products, Columbia, MD, USA) was added at 2.3% in a typical loaf type of wet pet food. It was mixed with chicken, chicken liver, chicken broth, brown rice, flaxseed, sweet potatoes, powdered cellulose, choline chloride, salt, potassium chloride, taurine, cassia gum, carrageenan, guar gum, vitamins and minerals in a mixer for 4 minutes and heated to 43 °C before filling the mixture in standard steel cans (156 g). The cans were sealed, retorted at 122 °C for 60 minutes, and cooled to room temperature before they were stored at 32 °C for 12 months and ambient temperature (21 °C) for 24 months. Samples were taken at various time points during the storage for DHA measurements with a GC method.

RESULTS

No noticeable loss of DHA was observed during the pet food process and retort. DHA retention were 66% at 12 months and 87% at 24 months under the storage conditions at 32 °C and ambient temperature, respectively.

CONCLUSIONS

In conclusion, DHA in DHAgold S17-B is stable in wet pet food during retort and long-term storage.

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WSVA8-0137

ONE HEALTH

SERODETECTION AND RISK ANALYSIS OF LEPTOSPIRAL INFECTION IN DOGS AND DOG HANDLERS (SHELTERED AND WORKING)

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INTRODUCTION

A global upward trend of leptospirosis warrants unified initiative in managing this zoonotic disease. Dogs were speculated to contribute in disease transmission posing risk to humans.

OBJECTIVES

Study aimed to detect leptospiral infection serologically and examined the risk factors towards leptospirosis among dogs and dog handlers.

METHODS

Serum were collected from 266 apparently healthy vaccinated (quadrivalent vaccine) dogs and 194 dog handlers. Microscopic agglutination test (MAT) was performed using 20 leptospiral serovars with a cut-off titre ≥1:100 (dog) and ≥1:50 (dog handlers). Risk factors were analysed using odd ratios.

RESULTS

Seventy dogs (26.3%) were seropositive mainly against serovars *icterohaemorrhagiae, ballum, bataviae and javanica* (titres:1:100-1:800). Sixty-seven dog handlers (34.5%) were seropositive mainly against serovars *grippotyphosa, icterohaemorrhagiae and malaysia* (titres:1:50-1:200). Risk factors for dogs and dog handlers were as shown in Table 1.

Risk Factor	Odd Ratio	p	Confidence Interval
Dog			
Rat Contact	4.29	0.05	0.98-18.78
Sharing Area	5.87	0.01	2.25-15.32
Environment Setting	1.8	0.03	1.06-3.21
Dog Handlers			
Rat Contact	6.60	0.01	2.80-15.60
Small Mammal Contact	4.46	0.01	1.91-10.44
Contact Time with Dog	4.20	0.01	1.70-10.50

Table1: Risk factors of leptospiral infection among dogs and dog handlers

CONCLUSIONS

Seropositive dogs were likely due to post-vaccination, post-exposure or subclinical infection, hence further investigation were required. Vaccine immunity may not be adequate as other serovars were detected. Low titres among dog handlers could indicate post-exposure. Rat contact poses risk for both groups. Prolonged contact time with dogs increased risk for handlers. Therefore, leptospirosis awareness among dog handlers could assist disease prevention

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WSVA8-0062

SEROPREVALENCE OF BORRELIA INFECTIONS IN HOUSE DOGS AND STRAY DOGS IN BANGKOK AND VINICITY, THAILAND

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INTRODUCTION

Lyme disease is a zoonotic infectious disease caused by spirochete *Borrelia burgdorferi* and transmitted to humans by the bite of infected *Ixodes* ticks. *Borrelia burgdorferi s*ensu stricto is the most pathogenic organism among those which cause Lyme disease in both human and dogs. Direct detection of *Borrelia burgdorferi* using PCR techniques or cultivation is reliable in tissue samples but not in blood samples Therefore, serological detection of antibodies is the method of choice for laboratory diagnosis of Lyme borreliosis in dogs. With its wide antigen spectrum, the Anti-*Borrelia* ELISA Dog is ideally suited for use as a screening test.

OBJECTIVES

The objective of this study was to determine the seroprevalence of *Borrelia* infection in dogs in Bangkok and vicinity, Thailand using the serological method.

METHODS

A total of 138 dogs (93 stray dogs and 45 house dogs) were collected from Bangkok and vicinity, Thailand. Sera were screened using IgG and IgM ELISA coated with mix of whole antigen extracts of *Borrelia burgdorferi* sensu stricto, *Borreliaafzelii* and *Borrelia garinii*.

RESULTS

These findings suggest that the percentage of the dog population who generated antibodies against *Borrelia* were 26.7% (12/45) and 59.1% (55/93) for house dogs and stray dogs, respectively.

CONCLUSIONS

This is the first report of *Borrelia* infection in house and stray dogs in Bangkok and vicinity, Thailand. Further exploration on source of infection and possible vectors is needed to understand the potential risks to animals and humans besides with tick diversity in domestic and wild animals in Thailand.

WSVA8-0096

ONE HEALTH

STAPHYLOCOCCAL SKIN MICROBIOTA AND METHICILLIN-RESISTANT STAPHYLOCOCCUS AUREUS (MRSA) IN DOGS AND CATS IN REMOTE NSW, AUSTRALIA

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INTRODUCTION

Staphylococcus is a diverse genus including several species of clinical importance to human and veterinary medicine. Little is known about the diversity of staphylococci, especially coagulase negative species within the microbiota of dogs and cats. The pets of remote NSW represent a unique population in which to investigate skin microbiota with low levels of exposure to antimicrobials and contact with a human population with a high incidence of antimicrobial-resistant staphylococcal infections.

OBJECTIVES

This study aimed to characterise the staphylococcal microbiota of a population of dogs and cats from remote NSW, Australia.

METHODS

Three swabs (nostrils, oropharynx, perineum) were collected from dogs and cats participating in a Companion-Animal Health Program in north-west NSW. Swabs were cultured on selective media for *Staphylococcus* spp. and for methicillin-resistant *Staphylococcus* spp. Species identification was confirmed by matrix-assisted laser desorption/ionisation-time of flight (MALDI-TOF) mass spectrometry.

RESULTS

Isolates from 218 dogs and 39 cats were identified to species level. MRSA was isolated from 2.3% of dogs and no cats. No methicillin-resistant *S. pseudintermedius* was isolated from dogs or cats. The diversity of *Staphylococcus* spp. was high with 16 species represented, including 13 coagulase negative species (Tables 1&2). *Staphylococcus pseudintermedius* was the most frequent isolate from dogs and *S. felis* from cats. *Staphylococcus aureus* was only isolated from 3.7% of dogs.

Table 1: *Staphylococcus* spp. microbiota of 218 dogs from remote NSW, Australia

	Staphylococcus species	Dogs (%)
Coagulase positive	S. pseudintermedius	89 (40.8)
	S. intermedius	26 (11.9)
	S. aureus	8 (3.7)
Coagulase negative	S. sciuri	23 (10.6)
	S. vitulinus	10 (4.6)
	S. felis	7 (3.2)
	S. xylosus	8 (3.7)
	S. nepalensis	3 (1.4)
	S. fleuretti	2 (1.0)
	S. lentus	2 (1.0)
	S. succinus	2 (1.0)
	S. equorum	6 (2.8)
	S. saprophyticus	1 (0.5)
	S. muscae	1 (0.5)
	S. cohnii	1 (0.5)
	S. simulans	5 (2.3)
Unknown		2 (1.0)

Table 2: *Staphylococcus* spp. microbiota of 39 cats from remote NSW, Australia

	Staphylococcus species	Cats (%)
Coagulase positive	S. pseudintermedius	4 (10.3)
	S. intermedius	0
	S. aureus	0
Coagulase negative	S. felis	20 (51.3)
	S. sciuri	1 (2.6)
	S. simulans	1 (2.6)
	S. xylosus	1 (2.6)
Unknown		1 (2.6)

CONCLUSIONS

MRSA was isolated from a high proportion of dogs relative to comparable populations, despite a low prevalence of *S. aureus*. This study confirms staphylococcal microbiota of dogs and cats is diverse and includes a wide range of coagulase negative species.

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OTHER

HYDRALLANTOIS IN A 4 YO FRENCH BULLDOG

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INTRODUCTION

Dystocia is a common condition in dogs. It can be categorized as resulting from *maternal*, or *fetal factors* or most commonly a *combination of both*.

OBJECTIVES

To describe the presenting symptoms with the condition known as hydroallantois.

METHODS

Pronounced abdominal distention, pale mucous membrane, tachypnea, and tachycardia were noted upon physical examination. Complete blood analysis, electrolyte and serum chemistry tests were done. Abdominal radiographs and ultrasound were performed to evaluate fetal status and size. Accumulation of fluid in the allantoic sac was revealed during ultrasound. Emergency Caesarian section with panhysterectomy was performed and grossly, an insurmountable amount of fluid was noted in the allantois. Six neonates were extracted, four of which presented with anasarca.

RESULTS

Hydrallantois is the result of the dysfunction of the placenta. It is more commonly observed in cows, ewes and mares, rarely in dogs and cats. Prognosis for future fertility of the bitch is poor and prognosis is guarded to poor.

CONCLUSIONS

Careful assessment of whelping patients must be done. Rectal palpation, ultrasound, and radiographs are useful tools in diagnosing this rare condition in dogs.

WSVA8-0195

OTHER

DIAGNOSTIC QUALITY OF COMPUTED TOMOGRAPHIC RETROGRADE URETHROGRAPHY IN MALE DOGS

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INTRODUCTION

The urethra is a common area of urologic dysfunction in male dogs for conditions such as strictures, obstructive urolithiasis, rupture, neoplasia and incontinence. Examination of the male urethra is difficult and often requires a diagnostic imaging work-up. CT Urethrography (CTU) is a promising technique that we have developed.

OBJECTIVE

This study was undertaken to establish the diagnostic quality of CTU studies in the evaluation of the canine urethra.

METHODS AND MATERIALS

This is a retrospective case series. Medical records from the University of Edinburgh were reviewed to identify male dogs presenting with lower urinary tract symptoms receiving a CTU study under our protocol. Images were blindly reviewed scoring the quality of the study, number of CTUs performed and the urethral width at different levels (0— Collapsed; 1- Partially Filled; 2- Distended; 3- Stenotic; and 4- Obstructed) for transverse, sagittal and curvilinear studies. Results were correlated with the final clinical diagnosis.

RESULTS

21 male dogs with a diagnostic quality CTU study were included. Urethral width was scored similarly across different locations and image orientations. There was complete correlation between urethral width grades 3 & 4 and clinically diagnosed stenosis and obstruction. 11/22 dogs (50%) required 1 CTU study, 9/22(41%) dogs required 2 CTU studies; and 2/22 (9%) required 3 CTU.

CONCLUSION AND DISCUSSION

CTU gives an accurate assessment of the urethral lumen in the male dog allowing reliable and time-efficient diagnosis of stenotic and obstructive conditions. Additional reconstructions in sagittal and curvilinear planes were helpful for the confidence of diagnosis, but did not change scoring results.

WSVA8-0145

OTHER

COMMON REASON FOR DEATHS OF NEW ZEALAND WHITE RABBITS MAINTAINED UNDER INDIVIDUAL HOUSING SYSTEM AT THE MEDICAL RESEARCH INSTITUTE OF SRI LANKA

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INTRODUCTION

NZW rabbits were brought to Sri Lanka from Japan in 1990. They were given a feed prepared at MRI according WHO formula using locally available ingredients and this formula was given to them for more than 25 years. They were maintained under proper bio security measures. Regular postmortem procedures are performed to identify any potential pathogenic organisms.

OBJECTIVES

To find out common reason for death of NZW rabbits for the period of 04/01/2017- 26/02/2018.

METHODS

Above 3 months to two years animals were recruited to the study (07). Sudden deaths or death after diarrhea were observed in the most of rabbits. Clinical signs were recorded and carcass was subjected to detailed postmortem (PM) investigations. Abnormalities in the external and internal organs were recorded. Samples were sent for histopathological and bacteriological investigations where necessary.

RESULTS

Two animals (28.5%) died due to torsion in the intestinal tract whilst two animals (28.5%) died due to stomach rupture. Sudden death, fasting and diarrhea were the commonest clinical signs. Congested blood vessels were observed in the area of torsion. Congested lungs also were observed. Tracheal swabs were negative for pathogenic streptococcus and staphylococcus. In two animals ((28.5%) diarrhea and dehydration were observed. But gut samples were negative for Salmonella, Shigella and Camphylobactor species. In one animal (14.28%) no clinical sign was observed. Stomach filled with food and congested lungs and blood filled trachea was observed. After PM and laboratory investigations no abnormality detected.

CONCLUSIONS

NZW rabbits at MRI are susceptible to diseases related to intestinal tract.

WSVA8-0068

OTHER

GENERAL OVER VIEW OF SMALL ANIMAL PRACTICE IN INDIA AND MUMBAI IN PARTICULAR.

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INTRODUCTION

Animals have been an integral part of Indian Society since hundreds of years Animals were not only looked after but were also worshiped in ancient India. Review of literature indicates that animal hospital existed in India in 300BC. There are about 95172 stray dogs as per 2014 census and pet dog population is 50000 in Mumbai as per municipal census. Pet dog population of India as per 2014 census is 10 million.

OBJECTIVES

In Mumbai with a geographical area of 603 sqkm there are more than 700 veterinarians some full time some part time with around 120 private clinics, 60 veterinarians doing house visit and 55 veterinarians in periphery of Mumbai called as MMRDA area. At present there is one Animal Hospital (SPCA) with facility for inpatients, Intensive care units and a crematorium.

METHODS

Facilities with each private clinical unit varies ranging from a mere OPD facility to having an operation theater, X ray machine, Ultrasonography facility, in house lab facility, own pharmacy. For transportation of pets to Veterinary facility people have to rely on private vehicles, limited public vehicles or animal ambulances. Since commutation is time consuming and expensive people prefer going to convenient veterinary close vicinity.

RESULTS

The availability of veterinary drugs is limited to select pharmacy stores. Specialty clinics are few and wide with specialist in surgery, medicine, radiology, exotic species, and birds.

CONCLUSIONS

Since the geographical area of Mumbai is small, small animal veterinarians will need to have more specialization to sustain in this competitive world.

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