# The Diseases of Bustards

.....with particular emphasis on rehabilitated bustards in the Middle East

Tom Bailey

International Wildlife Consultants, Wales, UK.

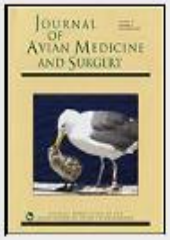
Dubai Falcon Hospital, Dubai, UAE.
National Avian Research Center, Abu Dhabi, UAE.

# Overview

- Health is of paramount importance for projects releasing captive bred birds.
- Disease problems have occurred in other CBRPs
  - Mauritius kestrels
  - Whooping cranes
  - Californian condors
  - Pink pigeons
- Health management and disease investigation are integral part of IUCN guidelines for Re-introduction and Rehabilitation Projects
- Knowledge of bustard diseases essential to minimise the transfer of novel diseases between rehabilitated, captive and free-living populations.



More Rights Options
JSTOR Terms And Con




*Journal of Avian Medicine and Surgery* 11(1):31–33, 1997


*Retrospective Studies*

# Adenovirus Infection in Mauritius Kestrels (*Falco punctatus*)

Neil A. Forbes, BVet Med, FRCVS, Greg N. Simpson, BVSc,
Robert J. Higgins, BVM&S, and Richard E. Gough, FIMLS

*Abstract:* Two similar highly fatal disease outbreaks occurred a year apart in separate populations of captive Mauritius kestrels (*Falco punctatus*) housed at the same raptor center. Sudden death was associated with few premonitory signs, but hemorrhagic diarrhea was sometimes observed. Consistent gross pathologic findings included hepatosplenomegaly and petechial hemorrhages throughout the gastrointestinal tract, which contained hemorrhagic fecal material. Histopathologic examination identified systemic necrotizing vasculitis and hepatitis associated with numerous intranuclear inclusion bodies. Adenovirus particles were subsequently detected in infected hepatocytes by electron microscopy. An adenovirus was isolated from the liver and spleen of a representative dead Mauritius kestrel, as well as from turkey poults and 1-day-old domestic fowl chicks that had been fed to the kestrels at the time of the first and second outbreaks, respectively.

*Key words:* adenovirus, diarrhea, inclusion bodies, kestrel, bird

## Introduction

clinically healthy birds at a raptor center. The kes-

# Summary of major causes of morbidity of imported and captive adult bustards

| Clinical finding | Imported adult | Captive adult |
|---|---|---|
| Soft tissue related traumatic injuries | 26.3% | 50.7% |
| Musculoskeletal findings | 4.9% | 22.9% |
| Parasitic findings | 24.7% | 15.5% |
| Viral findings | 20.1% | 0.4% |
| Fungal findings | 0.7% | 2.2% |
| Opthalmological findings | 15.1% | 5.4% |
| Miscellaneous disorders | 8.2% | 2.9% |

Avian Dis 1996: 40; 121-129
Avian Dis 1996: 40; 296-305

AVIAN DISEASES 40:121–129, 1996

# Causes of Morbidity in Bustards in the United Arab Emirates

T. A. Bailey,[A] J. H. Samour,[A] J. Naldo,[A] J. C. Howlett,[A] and M. Tarik[B]

[A]Veterinary Science Department, National Avian Research Center, Box 45553, Abu Dhabi, United Arab Emirates
[B]Central Veterinary Laboratory, Al Ain, United Arab Emirates

Received 14 March 1995

SUMMARY. The findings of 1746 clinical examinations of 594 bustards of six different species are presented, and the differences of causes of morbidity between imported adult, captive adult, and captive juvenile bustards are discussed. Wing tip trauma, keel trauma, and other soft tissue-related traumatic injuries were the most commonly diagnosed clinical conditions in captive adult bustards, accounting for 50.7% of the total findings in this category. Soft tissue-related related traumatic injuries accounted for 26.3% and 5.3% of the findings of imported adult and captive juvenile bustards, respectively. Musculoskeletal disorders were the most commonly diagnosed clinical conditions in captive juvenile bustards, accounting for 61.3% of the total findings in this category. Nutritional bone disease was the single most important musculoskeletal disorder in captive juvenile bustards, accounting for 24% of total findings. Musculoskeletal disorders accounted for 22.9% and 4.9% of findings in captive adult and imported adult bustards, respectively. Parasitic observations accounted for 24.7% of the total findings in imported adult bustards. Infectious viral diseases were almost exclusively confined to the imported adult bustards, making up 20.1% of findings in this group. Opthalmologic conditions accounted for 15.1% of findings in imported adult bustards.

AVIAN DISEASES 40:296–305, 1996

# Postmortem Findings in Bustards in the United Arab Emirates

T. A. Bailey,[A] P. K. Nicholls,[B] J. H. Samour,[A] J. Naldo,[A]
U. Wernery,[C] and J. C. Howlett[A]

[A]Veterinary Science Department, National Avian Research Centre, Box 45553, Abu Dhabi, United Arab Emirates
[B]Animal Pathology Division, Cambridge Veterinary School, Madingley Road, Cambridge, England
[C]Central Veterinary Research Laboratory, Dubai, United Arab Emirates

Received 14 November 1994

SUMMARY. A review was conducted of 236 postmortem examinations of six species of captive and imported bustards in the United Arab Emirates for the period 1979–94. The most common causes of death in adult imported houbara bustards (*Chlamydotis undulata macqueenii*) were euthanasia due to Newcastle disease, aspergillosis, and post-transportation-related deaths. Helminth parasites were a common finding in imported houbara bustards, and large parasite burdens occasionally caused intestinal obstruction and death. The most common causes of death in adult captive houbara bustards were trauma-related deaths and euthanasia for or death following treatment for capture myopathy. Fatty liver change was an important postmortem finding of captive adult houbara bustards. The main cause of death in juvenile houbara bustards was yolk-sac infection and septicemia. The most common causes of death in adult kori bustards (*Ardeotis kori*) were capture myopathy and handling injuries or euthanasia following these injuries. The most common causes of death in

# Health screening results for smuggled houbara bustards

| Disease | Live or Dead | Analysis | No. | No. positive (%) |
|---|---|---|---|---|
| Chlamydia | Live | ELISA swab | 194 | 49 (25.3) |
| Chlamydia | Live | ELISA sera | 132 | 67 (50.8) |
| Salmonella | Dead | Cloacal swab | 41 | 11 (26.8) |
| Salmonella | Live | Cloacal swab | 93 | 2 (2.2) |
| Aspergillosis | Dead | PME | 163 | 55 (33.7) |
| Paramyxovirus 1 | Live + dead | Virus isolation | 121 | 5 (4.1) |
| Paramyxovirus 1 | Live | HI sera | 270 | 151 (55.9) |
| Paramyxovirus 2 | Live | HI sera | 126 | 5 (3.9) |
| Paramyxovirus 3 | Live | HI sera | 126 | 0 (0) |
| Avian pox | Live | Clinical exam | 585 | 139 (23.7) |
| Avian pox | Dead | PME | 161 | 19 (11.8) |
| Avian pox | Live | AGP sera | 146 | 11 (7.5) |
| Infectious bronchitis | Live | HI sera | 79 | 0 (0) |
| Avian influenza | Live | AGP sera | 79 | 0 (0) |
| Avian pneumovirus | Live | ELISA sera | 24 | 0 (0) |
| Avian leucosis | Dead | Histopathology | | |
| Avian reovirus | Live + dead | Virus isolation* | Nd | Nd |
| Avian adenovirus | Dead | Virus isolation* | Nd | Nd |
| Trichomonas sp | Live | Clinical exam | 491 | 76 (15.5) |
| Haemoproteus | Live | Blood smear | 165 | 90 (54.5) |
| Leucocytozoon | Live | Blood smear | 165 | 3 (0.02) |
| Endoparasites | Dead | PME | 93 | 34 (36.5) |

Oryx Vol 34 No 4 October 2000

# Health considerations of the rehabilitation of illegally traded houbara bustards *Chlamydotis undulata macqueenii* in the Middle East

Tom Bailey, Christu-Das Silvanose, Jesus Naldo, Olivier Combreau, Frederic Launay, Ulrich Wernery, Joerg Kinne, Richard Gough and Ruth Manvell

**Abstract** There is a large illegal trade in trapped houbara bustards *Chlamydotis undulata macqueenii*, which are smuggled into the Middle East for falconry. Mortality is high and is associated with poor transport conditions, malnutrition, overcrowding and exposure to multiple infectious diseases, in particular aspergillosis, avian pox and paramyxovirus type 1 virus. Other pathogens include *Salmonella* sp., *Pseudomonas* sp., *Trichomonas* sp., intestinal endoparasites, avian leucosis, reovirus, adenovirus, paramyxovirus type 2 and *Chlamydia* sp. Various regional initiatives have been initiated to confiscate and rehabilitate illegally trapped birds. This paper reviews the causes of morbidity and mortality seen in illegally traded houbara bustards and provides health recommendations for those involved in rehabilitation.

**Keywords** Health, houbara bustard, illegal trade, Middle East, rehabilitation.

# Known mortality of smuggled houbara bustard flocks

| Year | Country | No. birds | % mortality | Cause |
|---|---|---|---|---|
| 1986 | UAE | 30 | 100 | PMV-1 |
| 1993 | Bahrain | 123 | 100 | Avian pox |
| 1993 | UAE | 36 | 33 | PMV-1 |
| 1994 | UAE | 22 | 77 | Aspergillosis |
| 1995 | UAE | 200 | 50 | PMV-1 |
| 1996 | Pakistan | 1,400 | 25 | Multiple* |
| 1997 | Pakistan | 1,500 | 22 | Multiple* |
| 1998 | UAE | 34 | 59 | Avian pox |
| 1998 | UAE | 24 | 25 | PMV-1 |
| 1998 | UAE | 95 | 49 | Multiple** |

*includes pox, PMV-1, aspergillosis and secondary mixed bacterial infections.

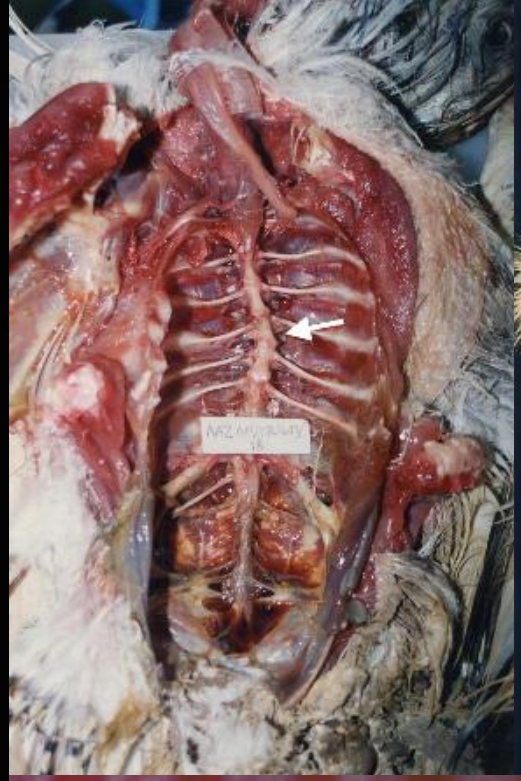
**includes pox, PMV-1, reovirus, adenovirus, *Pseudomonas* sp. and *Salmonella* sp.

# Biomedical reference collection - role in disease investigation

- Monitoring causes of disease and mortality of bustards in wild and captivity can minimise transfer of novel diseases between wild/captive populations during re-introductions.
- Disease monitoring protocols of captive animals should include provision of a **Biomedical Reference Collection.**
- Retrospective studies on banked tissues, sera etc invaluable to determine historical prevalence of diseases and exposure to infectious organisms.
- The NARC collection was used as a source of material for a range of studies to build up a disease and biological profile of bustards.

# Biomedical reference collection - material collected from dead birds.

| | |
|---|---|
| **Bird ID** | Give the bird a unique accession number e.g. AAZ/HB/170493/3. |
| **History** | Record any observations or known previous history in the record sheet. |
| **Radiography** | Take radiographs prior to post-mortem. |
| **Biometrics** | Weigh the bird and record biometric measurements. |
| **Blood samples** | If the bird is still alive awaiting euthanasia collect serum. |
| **PM organ measurements** | Record the weight and/or sizes of the following tissues; Weight - liver, abdominal fat, kidney, gizzard (full, empty), spleen, pancreas. Length and width and activity – gonad Length - oesophagus, SI, LI, caeca, pancreas |
| **Archived tissues** | Tissues must be retained in formalin or frozen. As a minimum; Formalin - brain, liver, kidney, spleen, lung, oesophagus, SI, LI, caeca, heart, pectoral muscle, sciatic nerve, air sac, gonad Frozen - brain, liver , abdominal fat, kidney, lung, SI, LI, feathers. |



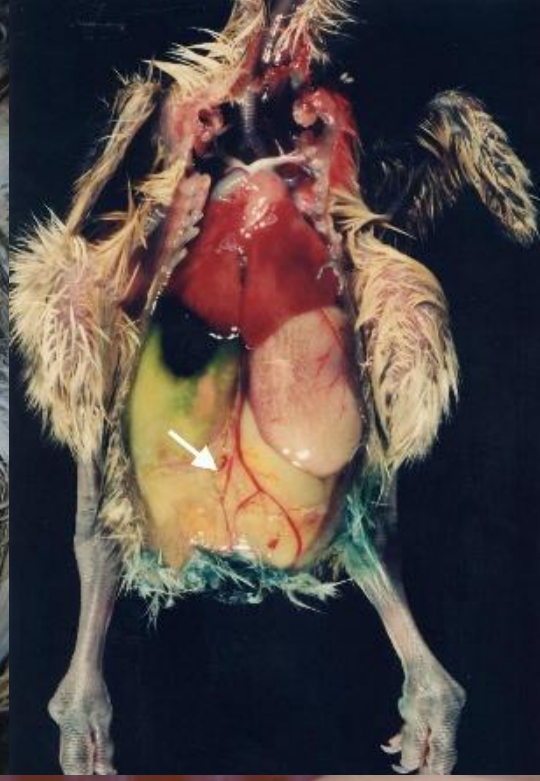


**Chart 11.1.** - An example of the standard form used to record post-mortems at NARC.

## Carcass Post Mortem Examination Form

**Bird ID** [_____]

**Submission information** (Please fill / tick √)

**Species** [_____] **Sex** [_____] **Ring code** [_____] **Ring colour** [_____] **Ring leg** L ( ) R ( )
**NARC site:** AAZ ( ) SRS ( ) **Others** [_____] **PIT No** [_____]
**Date of death** [__/__/__] **Time found** [_____] **Circumstance of death:** Anaesthetic associated ( ) Euthanasia ( )
Restrain associated ( ) Environmental conditions ( ) Infection associated ( ) Injury from cagemate ( )
Predation ( ) Self-inflicted injuries ( ) Death in transit ( ) Other ( )

**Laboratory use only**

**Post mortem data** (Please fill / tick √)

**PMR date** [__/__/__] **Carcass condition:** Fresh ( ) Autolysed ( ) Frozen ( ) Decomposed ( )
**Clinical summary** [_____]

**Post mortem condition data** (Please fill / tick √)

**Weight** [_____g] **Pectoral muscle condition:** Obese ( ) Good ( ) Fair ( ) Poor ( ) Emaciated ( )
**Feather condition:** Good ( ) Poor ( ) **Ectoparasites:** None ( ) Lice ( ) Ticks ( ) Other ( ) specify [_____]
**Condition comments** [_____]

**Post mortem systematic examination**

(Circle number and describe lesions under notes below) Key : NAD (Nothing abnormal detected) NE (Not examined)

| Number | | | | Number | | | |
|---|---|---|---|---|---|---|---|
| | 1. Body cavity | NAD | NE | 7. Musculoskeletal system | NAD | NE | |
| | 2. Cardiovascular system | NAD | NE | 8. Nervous system | NAD | NE | |
| | 3. Digestive system | NAD | NE | 9. Reproductive system | NAD | NE | |
| | 4. Endocrine system | NAD | NE | 10. Respiratory system | NAD | NE | |
| | 5. External examination | NAD | NE | 11. Urinary system | NAD | NE | |
| | 6. Lymphoreticular system | NAD | NE | 12. Other examinations [_____] | | | |

| No | Organ | Location | PM Findings |
|---|---|---|---|
| 9 | Testis ( ) Ovary ( ) | L ( ) R ( ) | **Gonadal activity:** Very active/cycling ( ) Active ( ) Inactive ( ) Immature ( ) **Gonad score:** 0 ( ) 1 ( ) 2 ( ) 3 ( ) 4 ( ) 5 ( ) |
| 1 | Abdominal fat | Entire | |
| | | | |
| | | | |
| | | | |
| | | | |
| | | | |
| | | | |
| | | | |
| | | | |
| | | | |
| | | | |

**Post mortem systematic examination (continued)**

| | | | |
|---|---|---|---|
| | | | |
| | | | |
| | | | |

**Gross PME findings/diagnosis**

**Post mortem summary**

**Post mortem number** [_____]

**Not For Disease Inputting**

**Reference samples taken : Code:** Fo (Formalin / fixed) Fr (Frozen) AL (Alcohol)

| Tissue/organ | FR | FO | AL | Tissue/organ | FR | FO | AL | Tissue/organ | FR | FO | AL |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Eye | | | | Pancreas | | | | Brain | | | |
| Eyelid | | | | Liver | | | | Sciatic nerve | | | |
| Cere | | | | Gall bladder | | | | Brachial nerve | | | |
| Beak | | | | Spleen | | | | Heart | | | |
| Tongue | | | | Bursa | | | | Aorta | | | |
| Oro-pharynx | | | | Thyroid | | | | Pulmonary artery | | | |
| Crop | | | | Parathyroid | | | | Pectoral muscle | | | |
| Oesophagus | | | | Adrenal | | | | Bone femur | | | |
| Proventriculus | | | | Kidney | | | | Joint hock | | | |
| Gizzard | | | | Ureter | | | | Wing | | | |
| Duodenum | | | | Trachea | | | | Faeces | | | |
| Jejunum | | | | Lung | | | | Abdominal fat | | | |
| Ileum | | | | Air sac | | | | Endoparasites | | | |
| Caecum | | | | Testis | | | | Ectoparasites | | | |
| Rectum | | | | Ovary | | | | | | | |
| Cloaca | | | | Oviduct | | | | | | | |

**Reference comments** [_____]

Veterinary surgeon

*Int. Zoo Yb.* (1997) **35:** 337–342 

# The establishment of a biomedical reference collection for a captive-breeding and restoration programme

TOM A. BAILEY[1,2], JAIME H. SAMOUR[1], JESUS L. NALDO[1], JOHN E. COOPER[2] & PHIL K. NICHOLLS[3]

[1]*National Avian Research Center, Veterinary Science Department, Box 45553, Abu Dhabi, United Arab Emirates,* [2]*Durrell Institute of Conservation and Ecology, University of Kent, Canterbury CT2 7PD and* [3]*Animal Pathology Division, Department of Clinical Veterinary Medicine, University of Cambridge, Cambridge CB3 0ES, Great Britain*

Biomedical data and material are invaluable in research and allow those managing captive-breeding and restoration programmes to build up accurate biological profiles of free-living and captive populations. This paper describes the establishment of a collection of biomedical reference material for the Houbara bustard *Chlamydotis undulata macqueenii* at the National Avian Research Center, Abu Dhabi. The numerous practical uses of the material are also described.

*Key-words:* biomedical reference collection, captive breeding, houbara bustard

tards and (3) investigating the diseases which affect wild Houbara bustards.

Literature on the biomedical characteristics of bustards is scarce (Bailey *et al.*, 1996; Ostrowski *et al.*, 1996). Monitoring the causes of disease and mortality of bustards in the wild and in captivity can minimize the transfer of novel diseases between captive and free-ranging populations when animals are reintroduced to

# Diseases of Bustards

- Viral
- Fungal
- Bacterial
- Parasites
- Multifactorial

# Viral Diseases

- Poxvirus
- Paramyxovirus type 1
- Influenza A virus
  - Reovirus
  - Adenovirus
  - Lymphoid leukosis
  - Other

# Viruses

- Background:
- Unique class of infectious agents consisting of genetic material (RNA or DNA) surrounded by a protein coat
- Unable to replicate outside a living host cell and use the biochemical mechanisms of the host cell to synthesise and assemble their separate components
- Some viruses e.g. poxvirus, produce intracytoplasmic inclusions in infected host tissues, which can be detected during histopathological examination
- Some viruses e.g. PMV-1, can be grown in chick embryos and identified by testing against standard sera

# Avian Pox

- Large DNA virus
- Induces intracytoplasmic inclusion bodies (Bollinger bodies)
- Reported in > 232 bird species
- Species specific strains
  - Raptorpox
  - Pigeonpox
  - Turkeypox
  - Canarypox
- Transmission by biting mosquitoes, direct contact with open skin lesions, blood borne trauma (pecking).
- Disease more common in warm latitudes during mosquito season.

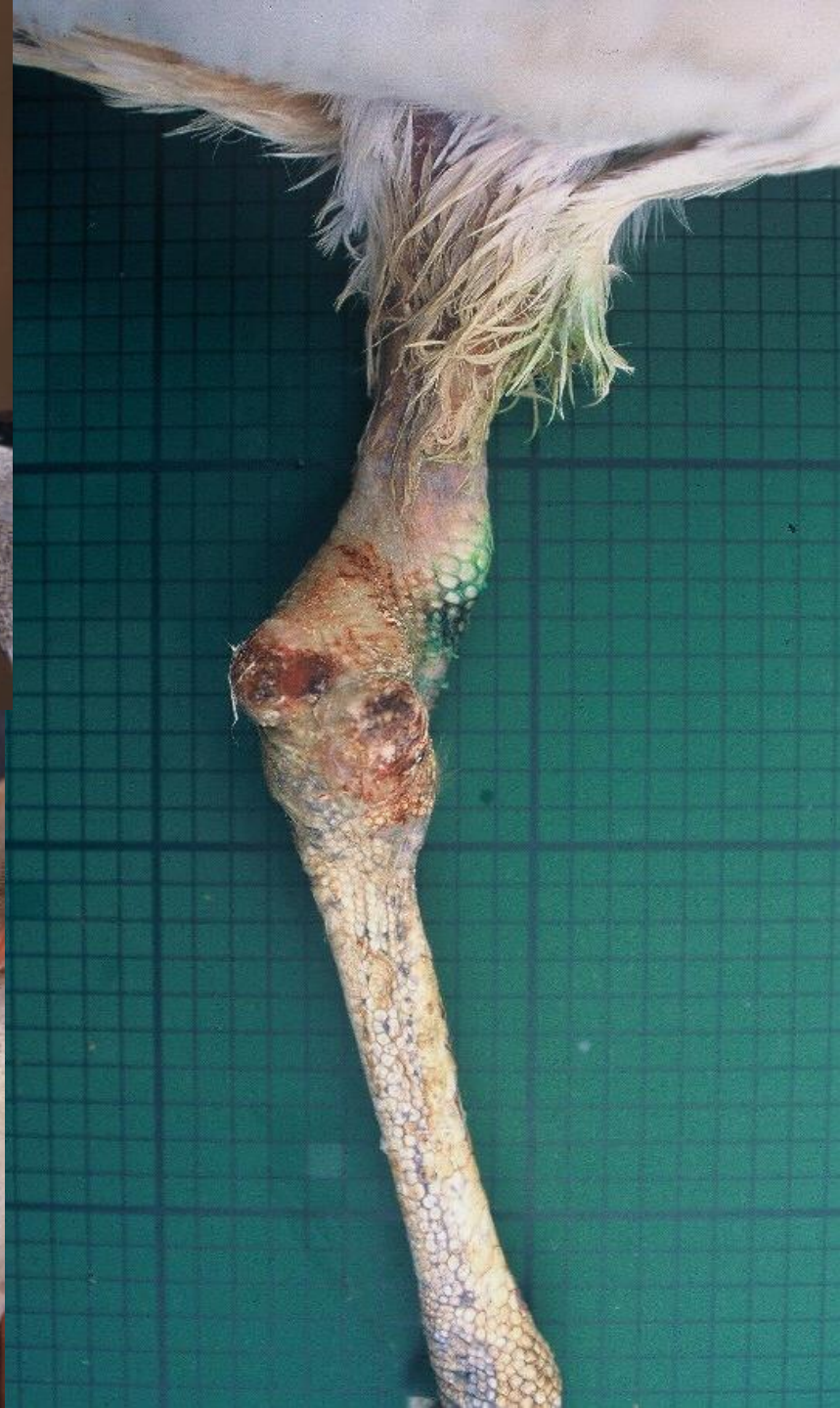
# Avian Pox

- **Clinical disease:**
  - Course of disease influenced by strain virulence, mode of infection & resistance of the bird
  - 4 forms of disease noted

1. Cutaneous
2. Diphtheroid
3. Tumerous
4. Neurological

# Avian Pox

- **Cutaneous form:**
  - Most common
  - Lesions on unfeathered areas: cere, eyelids, feet
  - Incubation period 4-9 days
  - Papules develop into crusty lesions
  - Strong birds start to recover
  - Immunocompromised birds develop secondary infections
  - Deep lesions develop and birds can die
  - See Avian Pathol 1995: 24; 573-577



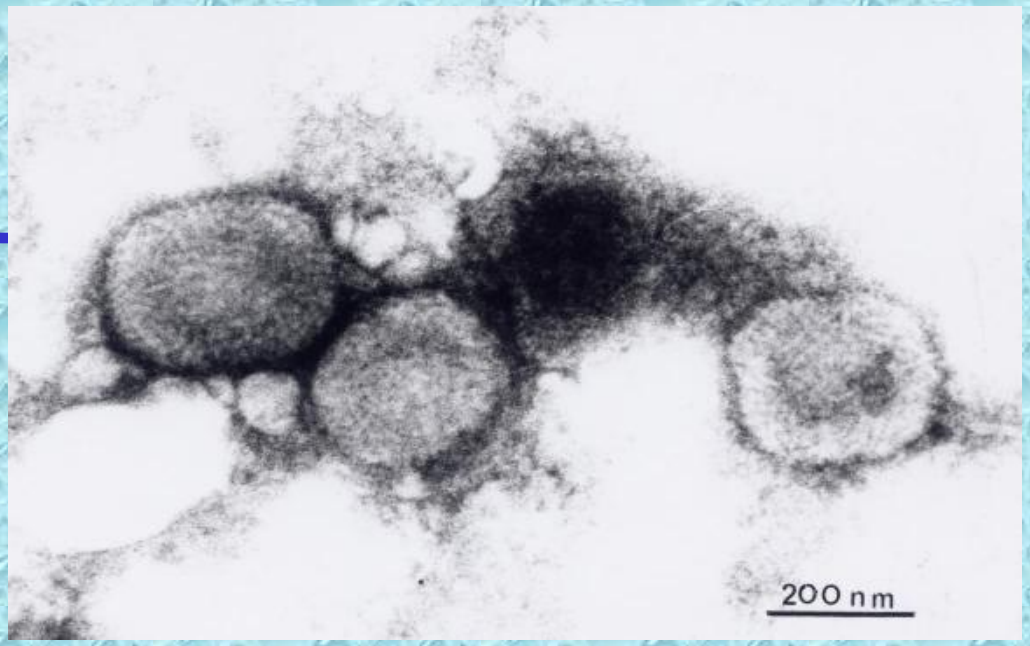
# Avian Pox

Birds that survive often left with cutaneous scars

# Avian Pox

- **Diphtheroid form:**
  - Less common, upper alimentary and respiratory tract affected, pseudomembranous diphtheritic lesions, bleeding erosions.
- **Tumerous form:**
  - Not common
- **Neurological form:**
  - Avipox infected birds reported with CNS signs, ?? could have had concurrent PMV-1 infections

# Avian pox




**Diagnosis**

- Clinical signs
- Virus detection (EM)
- Virus isolation (Chorio-allantoic membrane of embryonated chicken eggs and intracytoplasmic inclusion bodies)
- Histopathology (swollen and vacuolated epithelium, intracytoplasmic - Bollinger - inclusion bodies)

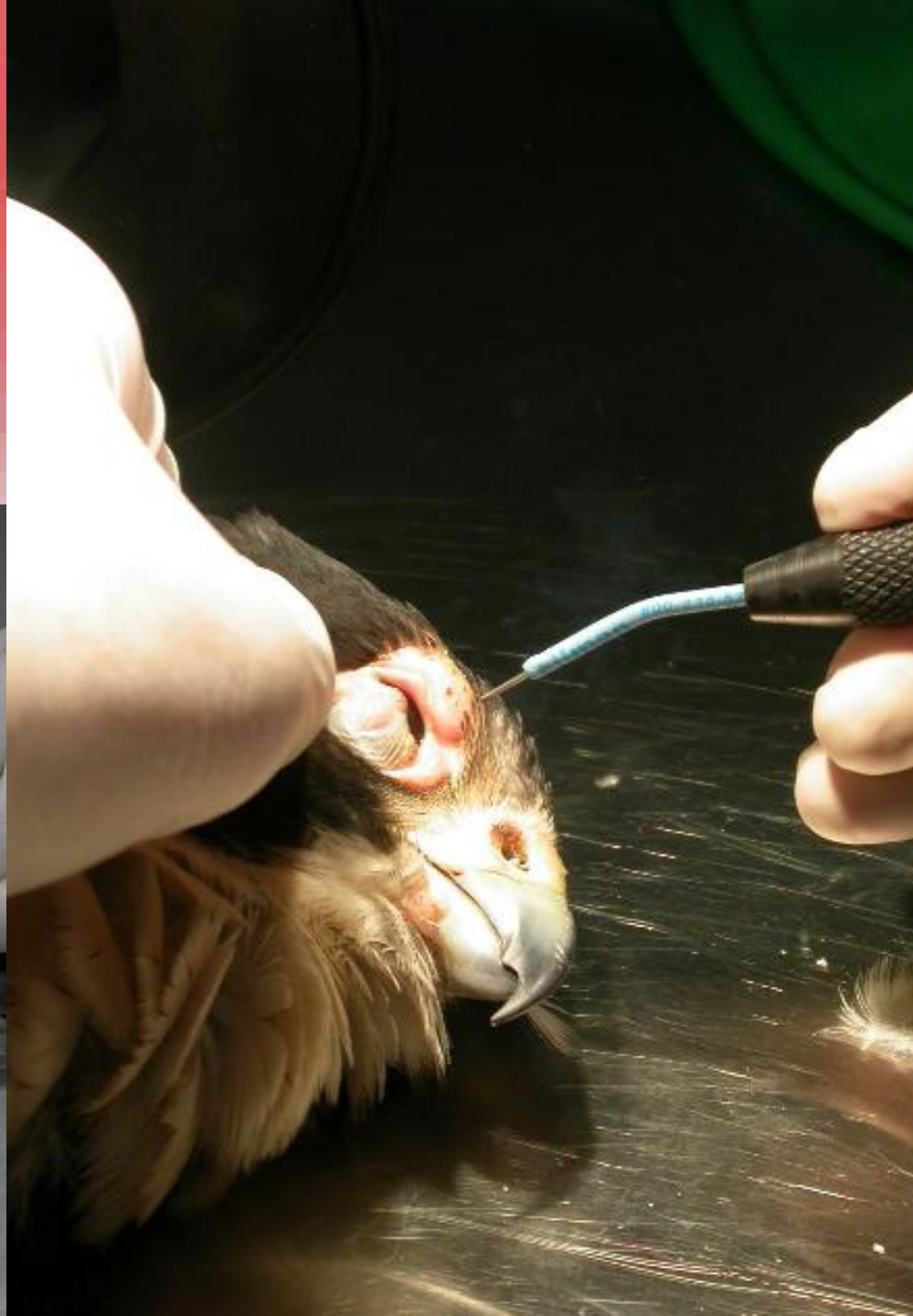
# Avian pox

**Medical treatment**

- Supportive treatment with antibiotics for concurrent bacterial infections, vitamin A, immunostimulation
- Surgical treatment - electrocautery, chemical cautery, surgical excision.

**Management treatment**

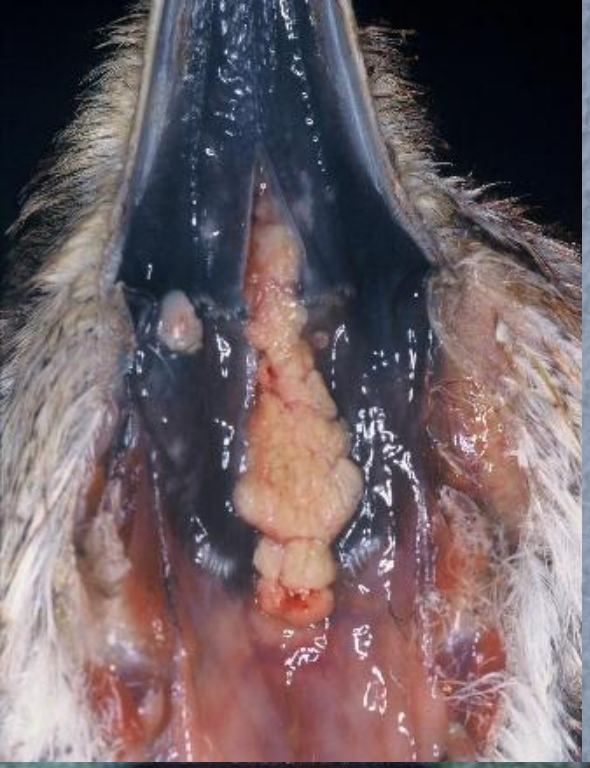
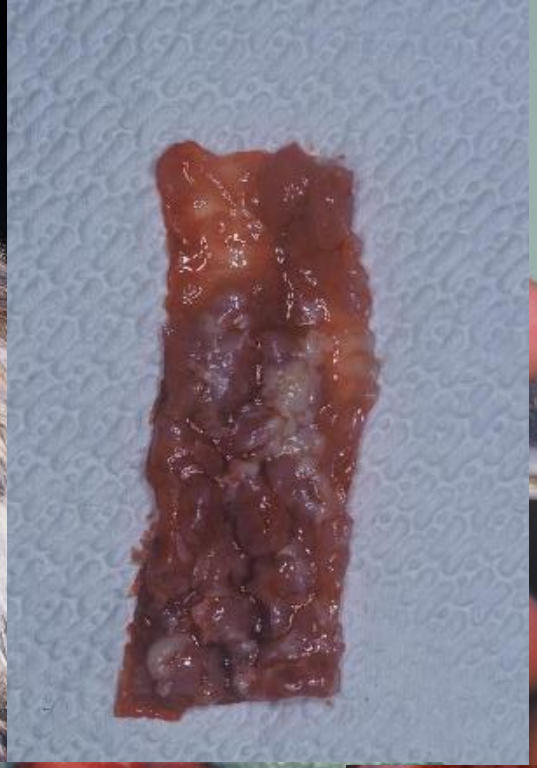
- Remove unaffected birds from clinical cases.
- Reduce exposure to vectors - insect killers and repellent, secure insect netting
- Ensure availability of good nutrition and water
- Prevention by vaccination

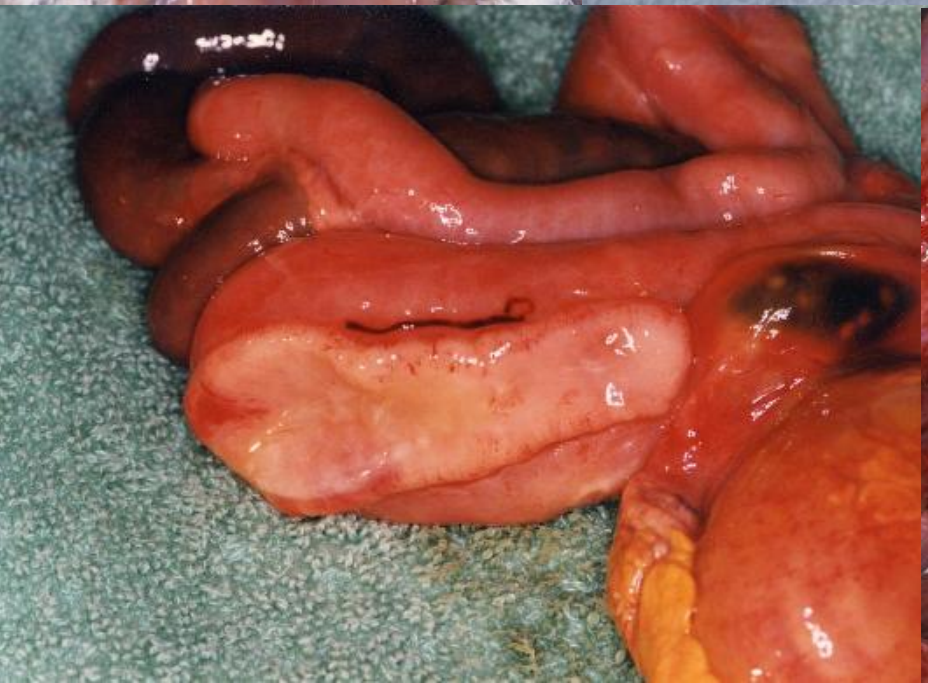
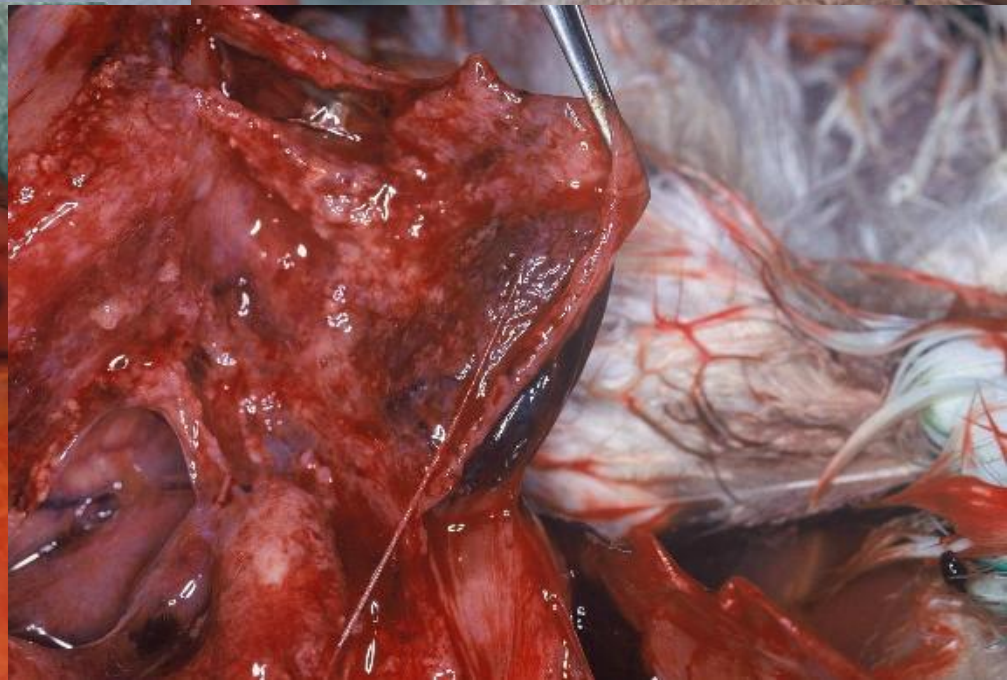


# Avian pox

- Diphtheritic and septicemic form can cause devastating mortality of 100%.
- Clinical signs = conjunctivitis, excessive lacrimation, papillomatous lesions in upper alimentary and digestive tract.
- Houbara pox not related to turkey or fowl pox virus strains based on lack of neutralisation by turkey pox immune serum.

- See Avian Dis 1995: 39; 907-911
- See J Vet Med 1996: 43; 287-292

# Newcastle disease

**Cause**

- Paramyxovirus type 1 virus. Group C1 (velogenic) in UAE.

**Epidemiology**

- Spread by contact with domestic poultry via respiratory aerosols and fecal contamination of food/water.
- Transportation methods for smuggling HB associated with PMV-1 outbreaks
- Incubation period several days - several weeks.
- New cases can appear 5-8 weeks after onset of outbreak.
- Mortality variable - 25-100%.

# Newcastle disease

**Clinical signs**

- PMV-1 is a variable disease. Diseases depends on virus pathotype, species, age, immune status, general health and environmental conditions.
- CNS signs in bustards in the UAE - inco-ordination, walking backwards, torticollis, opisthotonus, ataxia, paralysis, circling, tucking head under keel, head tilt
- PMV-1 and avian pox in bustards in KSA caused - poor appetite, depression, ruffled feathers, dehydration, ocular and nasal discharges.
- See J. Zoo and Wildl Med 1997: 28; 325-330

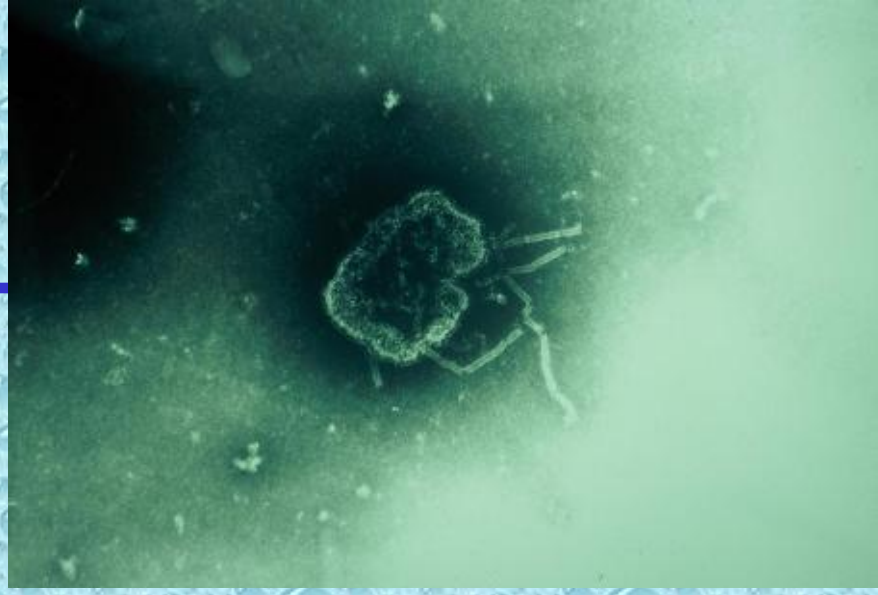
*Journal of Zoo and Wildlife Medicine* 28(3): 325–330, 1997


# AVIAN PARAMYXOVIRUS TYPE 1 INFECTION IN HOUBARA BUSTARDS (*CHLAMYDOTIS UNDULATA MACQUEENII*): CLINICAL AND PATHOLOGIC FINDINGS

**Thomas A. Bailey, B.V.Sc., M.R.C.V.S., Philip K. Nicholls, B.V.Sc., M.R.C.V.S., Ulrich Wernery, Ph.D., Jaime Samour, M.V.Z., Ph.D., John E. Cooper, F.R.C.V.S., M.R.C.Path., and Marion T. O'Leary, B.V.Sc., M.R.C.V.S.**

*Abstract:* Clinical and pathologic findings of avian paramyxovirus type 1 (PMV-1) in 19 houbara bustards (*Chlamydotis undulata macqueenii*) imported from Pakistan into the United Arab Emirates and one captive-bred bird are reported. Clinical signs included circling, walking backward, ataxia, opisthotonos, torticollis, recumbency, head tilt, head shaking, head tremor, tucking of head under keel, nasal discharge, conjunctivitis, and diarrhea. The length of time imported birds exhibited clinical signs varied from 4 days to 18 mo after importation. Hemagglutinating antibodies

# Newcastle disease



## Diagnosis

- Virus isolation (lung, GIT contents and brain tissue).
- PMV-1 HI test - rising antibody titre.
- Histological changes consistent with a viral encephalitis and interstitial nephritis.

## Treatment

- None, so prevent by vaccination.
- Quarantine protocols essential.
- Some birds have survived with chronic CNS signs for 18 months, but CNS signs did not regress. Survivors may become latent carriers.

# Table 1: PMV-1 strains isolated from different avian species in the UAE over a decade (Wernery, 2000)

| Avian Sp | Groups | | | | | | |
|---|---|---|---|---|---|---|---|
| | A | B | C | F | Pigeon | None | Total |
| Falcon | 1 | 16 | 17 | 8 | 4 | 15 | 61 |
| Pigeon | 2 | 5 | 10 | 6 | 15 | 4 | 42 |
| Quail | 0 | 0 | 9 | 1 | 1 | 5 | 16 |
| Houbara | 0 | 5 | 13 | 0 | 0 | 0 | 18 |
| Chicken | 0 | 5 | 5 | 1 | 0 | 0 | 11 |
| Partridge | 0 | 0 | 2 | 1 | 0 | 0 | 3 |
| Peacock | 0 | 0 | 3 | 0 | 0 | 0 | 3 |
| Pheasant | 0 | 0 | 2 | 0 | 0 | 0 | 2 |
| St curlew | 0 | 0 | 1 | 1 | 0 | 0 | 2 |
| Ostrich | 0 | 3 | 0 | 0 | 0 | 0 | 3 |
| Secretary B | 0 | 5 | 1 | 0 | 0 | 0 | 6 |
| Total | 3 | 39 | 63 | 18 | 20 | 24 | 167 |

Wernery, *Avian PMV-1 in the UAE*. 2000. Falco 15.

# Key features of outbreaks of pox and PMV-1 in imported HB in 1998-1999

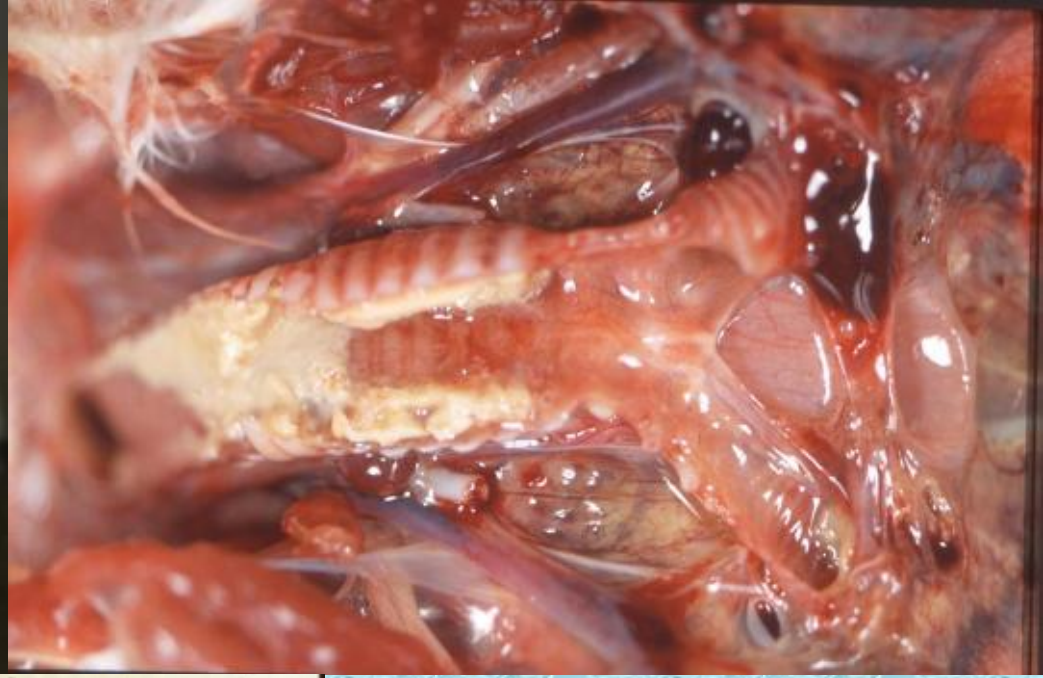
| | Group A (Pox) | Group B (PMV-1) |
|---|---|---|
| Total birds | 34 | 24 |
| Morbidity (% with clinical signs) | 26 (76.5%) | 6 (25%) |
| New cases period | Arrival to 18 days | 4-17 days |
| Number euthanased | 6 | 3 |
| Mortality (%) | 16 (47%) | 6 (25%) |
| Total mortality including other causes | 21 (61.7%) | 6 (25%) |
| Mortality period | 9-25 days | 5-19 days |

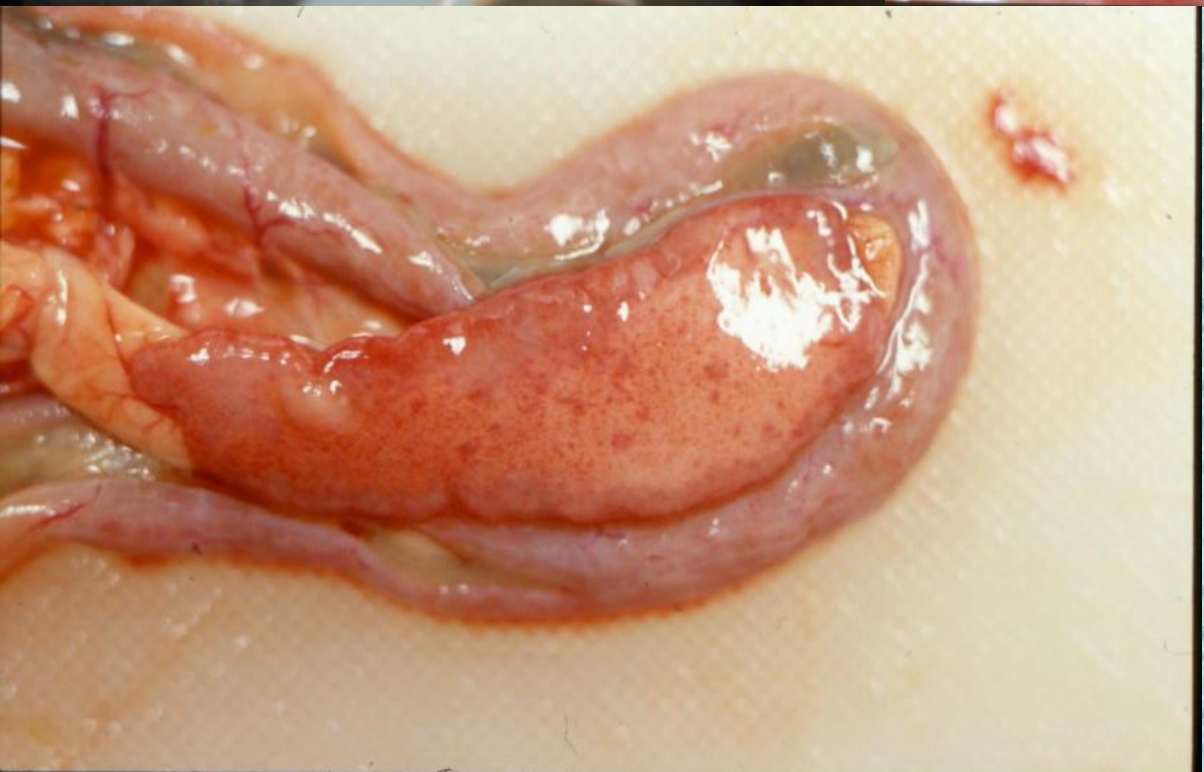
# Influenza A virus

- Avian influenza virus member of Orthomyxovirus family
- Three types of influenza viruses
  - A, B, C
- Only influenza A reported in birds
- Pathogenicity of AIV varies - depends on host species & viral strain
- Virulent chicken strains H5 & H7 subtype
  - HPAI - highly pathogenic AI virulent viruses can result in flock mortality of 100%
  - LPAI - low pathogenic AI cause a milder disease -mild respiratory signs, depression and egg production problems

# Avian Influenza

- **Transmission**
- Infected birds shed the virus via respiratory secretions, conjunctiva and faeces
- influenza strain isolated from UAE bustards bought in with smuggled birds that had been in contact with diseased poultry
- **Clinical disease**
- avian influenza A (type H9N2) outbreak in houbara
- anorexia, lethargy, opisthotonos, head ticking, ocular and nasal discharges and a severe dyspnoea





## Pathology

- fibrinous peritonitis and pericarditis
- enlarged liver and spleen
- red pea-sized spots in the lungs
- trachea filled with thick yellowish fluid & necrotic material
- severe pancreatitis

ELSEVIER
SAUNDERS

VETERINARY
CLINICS
Exotic Animal Practice

Vet Clin Exot Anim ■ (2006) ■ ■

# An Outbreak of Low Pathogenic Avian Influenza in a Mixed-Species Aviculture Unit in Dubai in 2005

Jo Kent[a,b,*], Tom Bailey[b], Christudas Silvanose[b], Sean McKeown[c], Ulrich Wernery[d], Joerg Kinne[d], Ruth Manvell[e]

[a]53 Henver Road, Newquay, Cornwall, TR7 3DQ, United Kingdom
[b]The Dubai Falcon Hospital, PO Box 23919, Dubai, United Arab Emirates
[c]H.E. Sheikh Butti Maktoum's Wildlife Center, PO Box 7237, Dubai, United Arab Emirates
[d]Central Veterinary Research Laboratory, PO Box 597, Dubai, United Arab Emirates
[e]Central Veterinary Laboratory, New Haw, Weybridge, Surrey, United Kingdom

The family Orthomyxoviridae is divided into types A, B, and C. Type A is the only type of veterinary importance, with avian influenza virus (AIV) of recent concern to human and bird health [1]. Type A AIV subtypes are categorized according to the surface antigens hemagglutinin (H) and neuraminidase (N) [2]. To date, 16 H (H1–H16) and 9 N (N1–N9) subtypes have been identified [3]. Further classification is determined by pathogenicity, with the Organization on Infectious Epizootics (OIE) classifying the virus into high or low pathogenic AIV (HPAIV and LPAIV, respectively) [4]. All HPAIV isolated to date has been H5 or H7 subtype; thus, these are of the greatest concern for the health of birds and human beings [5,6].

In the United Arab Emirates (UAE), 34 AIV cases have been isolated (Table 1). Infections with LPAIV H9N2 were diagnosed in farmed chickens and Houbara Bustards (*Chlamydotis undulata*), and an HPAIV H7N3 strain was isolated from a Peregrine Falcon (*Falco peregrinus*) [7,8]. To the authors' knowledge, no clinical cases of AIV in the Stone Curlew (*Burhinus oedicnemius*), White-Bellied Bustard (*Eupodotis senegalensis*), or Blacksmith Plover (*Anitibyx armatus*) have been previously reported in the literature.

* Corresponding author. 53 Henver Road, Newquay, Cornwall, TR7 3DQ, United Kingdom.
*E-mail address:* jokent_@hotmail.com (J. Kent).

1094-9194/06/$ - see front matter © 2006 Elsevier Inc. All rights reserved.
doi:10.1016/j.cvex.2006.05.012
vetexotic.theclinics.com
vap0188 ■ 27 June 2006 ■ 11:32 pm



Table 1
Influenza virus strains isolated from avian species in the United Arab Emirates

| Avian species | Number | Strain |
|---|---|---|
| Peregrine Falcon | 1 | H7N3 |
| | 1 | H7N1 |
| Chicken | 7 | H9N2 |
| Houbara Bustard | 3 | H9N2 |
| | 7 | H7N1 |
| Quail | 7 | H9N2 |
| Sudanese Bustard | 1 | H9N2 |
| White-Bellied Bustard | 4 | H9N2 |
| Stone Curlew | 2 | H9N2 |
| Blacksmith Plover | 1 | H9N2 |
| Total | 34 | |

Avian Pathol. 2009 Feb;38(1):35-9.

# Isolation and identification of highly pathogenic avian influenza H5N1 virus from Houbara bustards (Chlamydotis undulata macqueenii) and contact falcons.

Khan QA, Shuaib MA, Rhman SS, Ismail MM, Hammad YA, Baky MH, Fusaro A, Salviato A, Cattoli G.

Central Veterinary Diagnostic Laboratory, Riyadh, Kingdom of Saudi Arabia. oakvet@rediffmail.com

## Abstract

Highly pathogenic influenza virus (HPAIV) H5N1 has caused mortality and morbidity in many species of domestic and wild bird. The Houbara bustard (Chlamydotis undulata macqueenii) is a solitary bird that inhabits semi-desert regions. It is known to be susceptible to avianpox, avian paramyxovirus type 1, and low-pathogenicity avian influenza H9N2. We report an outbreak of H5N1 HPAIV in Houbara bustards, which were introduced into the Kingdom of Saudi Arabia for falconry purposes. Ninety-three per cent mortality (38 out of 41 birds) in the infected Houbara bustard flock and about 62.5% mortality (10 out of 16 birds) in falcons that came in contact with these birds were observed. Pooled cloacal and tracheal swabs from Houbara bustards as well as visceral organ homogenates collected in Houbara bustards and falcons were tested by real-time reverse transcriptase-polymerase chain reaction, and virus isolation was attempted in specific pathogen free hens' eggs. The viruses isolated were characterized as HPAIV H5N1. Phylogenetic analysis of the haemagglutinating and Neuraminidase (NA) genes revealed that the viruses isolated from Houbara bustards and falcons were closely related to each other and to Kuwaiti H5N1 strains isolated in 2007. Interestingly, they were genetically distinguishable from the co-circulating A/H5N1 viruses in Kingdom of Saudi Arabia causing outbreaks in domestic birds. This case emphasizes the need for surveillance of this endangered species in its natural habitat.

# Avian Influenza

- Differential diagnosis list for avian influenza
- PMV-1
- Infectious laryngotracheitis
- Chlamydophilosis
- Mycoplasmosis
- Other respiratory and gastro-intestinal infectious diseases
- Note: concurrent infection with other viruses and bacteria often seen

# Avian Influenza

- **Diagnosis**
- Definite diagnosis depends on isolation and identification of the virus
- H7N1 and H9N2 confirmed from Dubai outbreaks
- Swabs from cloaca and upper respiratory tract suitable for virus isolation from live birds.
  - Liver, trachea, liver, lungs, spleen and brain are best organs to sample from dead birds.
- Serology using HI and ELISA is possible, but the HI test does not recognise all antibody classes and the ELISA is no more specific than the group specific antigen
- Paired samples (acute/convalescent) confirm an infection

# Avian Influenza

- **Treatment and Control**
- no specific treatment exists for infected birds
- methods for controlling the spread of this disease in poultry are based on preventing contamination and controlling the movement of people
- 2005 outbreak in Dubai, the in-contact affected and unaffected white-bellied bustard chicks were treated orally with 10 mg/kg oseltamivir phosphate (Tamiflu®, Roche) q12h for 7 days
- Vaccination

# Reovirus

- Reovirus isolated from confiscated houbara bustards, but has not been associated with morbidity or mortality in this species.

- Wernery and McKinney (unpublished) report infection in Heuglin's bustard chicks from which reovirus was isolated, but was not proven to have been the cause of mortality
  - anorexia, periorbital sinusitis, head shaking and sneezing, mild diarrhoea, and severe dyspnoea,

# Adenovirus

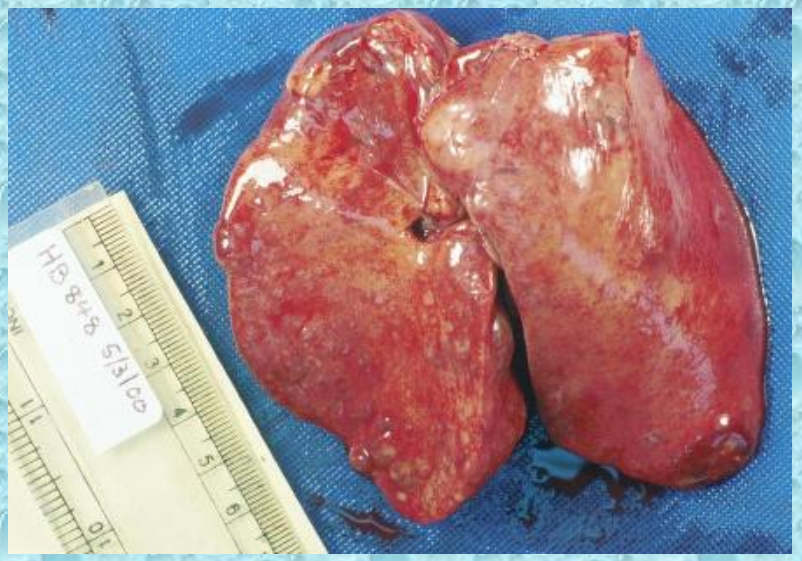
- Confiscated houbara bustards at NARC quarantine unit intermittently shed adenovirus during a 10 month-long health-monitoring programme

- Adenovirus isolated at NARC from a 21-day-old houbara bustard chick that died acutely.
  - post-mortem findings -massively enlarge spleen, pericarditis and hepatic and renal congestion

*Disease is very old and nothing about it has changed. It is we who change, as we learn to recognise what was formerly imperceptible."*
Jean Marie Charcot (1825-1893)

# Lymphoid Leukosis

- Caused by viruses placed in a subgroup of avian type C oncoviruses of the family retroviridae
- Rarely reported in bustards
- Clinical examination revealed massively distended livers
- PM showed ascites and enlarged liver with whitish nodules in the spleen and kidney
- Histopathology of affected organs revealed severe infiltration of the liver and spleen with mononuclear cells
- Virus isolation on frozen organs collected from one bird was unsuccessful



# Diseases of Bustards

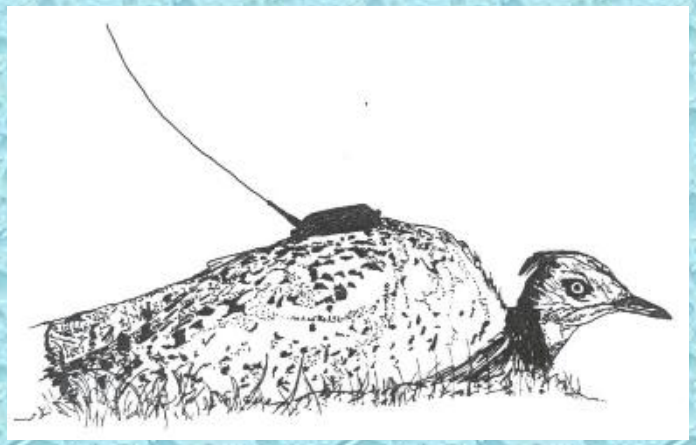
- Viral
- Bacterial
- Fungal
- Parasites
- Multifactorial

# Bacterial Diseases

- **Gram-negatives**
- E. coli
- Salmonella
- Yersinia
- Pseudomonas

- **Gram-positives**
- Staphylococcus
- Streptococcus
- Erysipelothrix
- Mycobacteriosis
- Clostridiosis

- **Other**
- Chlamydophilosis
- Mycoplasma



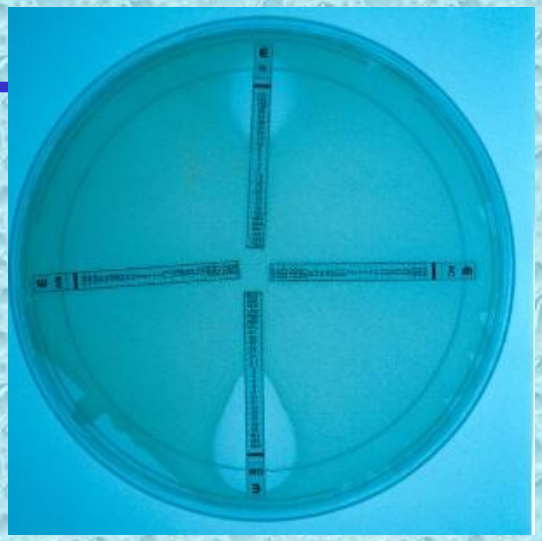
# Bacterial Diseases

- *Most bacterial diseases are the result of a complex relationship between the host, environment & bacteria*

- Normal bacterial flora can cause disease when the normal body defenses are impaired, e.g.:
  - adverse temperatures
  - periods of starvation
  - shipping
  - dietary changes
  - viral infections
  - immunosuppression & general stress

- In a compromised host  infections can result in a septicemia terminating in sudden death.

# Bacterial Diseases

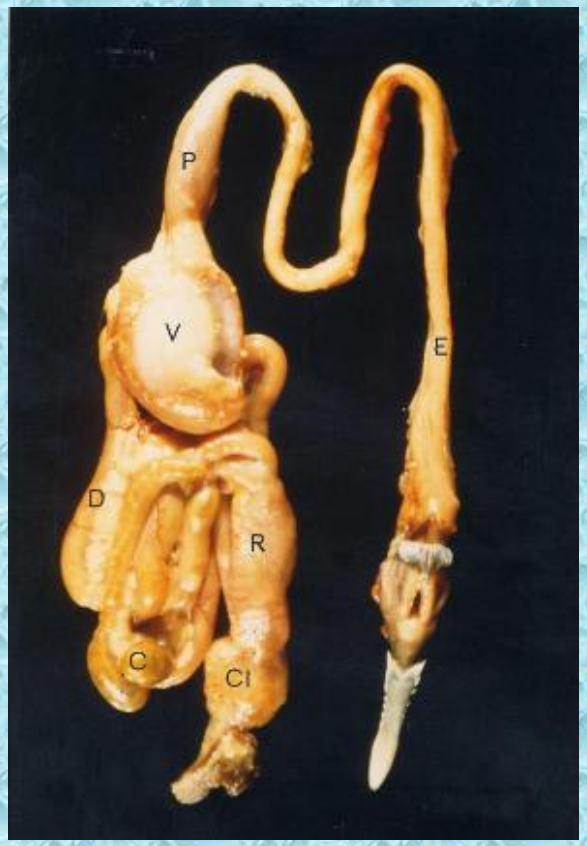


- **Background :**
- Bacterial susceptibility to antibiotics is important
  - bacteria rapidly become resistant to commonly used antibiotics

- Many surveys of resistance patterns of bacteria isolated from domestic fowl
  - little data reported for bustards
- When using oral antibiotics consider effect on normal intestinal flora
  - Establishment of antibiotic resistant (e.g. *P. aeruginosa*) infections observed following oral antibiotic use
  - Increase in incidence of fungal gastritis after oral antibiotics

# Bacterial Diseases

- **Lack of lymph nodes:**
- Important aspect of avian anatomy is the lack of lymph nodes
  - In particular mesenteric lymph nodes which could inhibit the movement of intestinal bacteria from the GIT into the blood circulation
- In healthy birds wbcs in intestinal mucosa prevent this
  - In stressed birds these wbcs become non-reactive and allow bacteria to enter the general circulation, causing septicaemia
  - Can occur at any age when birds are placed under conditions of stress.



# Bacterial Diseases

- Understanding aetiology of bacterial infections in bustards assisted by knowledge of normal flora and its role in disease
- Gut flora influenced by diet
  - In one study *E. coli* most common organism isolated from bustard feces – these birds were on a diet with mince and mice
  - *Proteus* spp., *Enterobacter* spp., *E. coli, Klebsiella* spp., *Aerococcus* spp. and *Enterococcus* spp. Considered normal aerobic intestinal flora.....- but all of these bacteria were also isolated from food items

# Interpreting bacterial culture results

- Interpreting bacterial results can be confusing
  - Following considerations by Gerlach indicate if a bacterial isolate is a component of a disease process:
  - Isolation of bacteria in almost pure culture (>80%).
  - Isolation of large numbers of bacteria in almost pure culture from heart tissue suggestive of bacteraemia.
  - Isolation of a bacterium with pathogenicity markers suggests it may be involved in disease process.
  - Isolation of bacteria from parenchyma with gross pathological or histopathological lesions.
  - Isolation of a bacterium without identification of other microorganisms such as viruses, protozoa, fungi, *Chlamydophila* etc.

# Chlamydophila

**Cause**

- *Chlamydophila psittaci*
- **Clinical signs**
- Highly variable clinical signs
- Sporadic, acute, fatal enteritis, tracheitis, airsacculitis, peritonitis syndrome seen in KSA
- Upper and lower respiratory tract signs seen at NARC (sinusitis, airsacculitis)
- In KSA outbreak morbidity rate was 45% and mortality was 25%

# Chlamydiosis

**Diagnosis**

- Culture of organism
- Impression smears of spleen and liver (inclusions)
- Detection of antigen by ELISA
- Detection of antibodies by ELISA

**Treatment**

- Appropriate antimicrobial agents (doxycycline)

- See Avian Dis 1993: 37; 1117-1120

# Bacterial sinusitis

**Causes**

- This is a common syndrome in adult and juvenile bustards;
- Commonly *Pseudomonas* spp.
- Others - *E. coli*, *Mycoplasma* spp., *Chlamydia* spp.

**Clinical signs**

- Swollen periorbital region
- Serous or purulent naso-ocular discharge
- Anorexia

# Bacterial sinusitis

**Diagnosis**

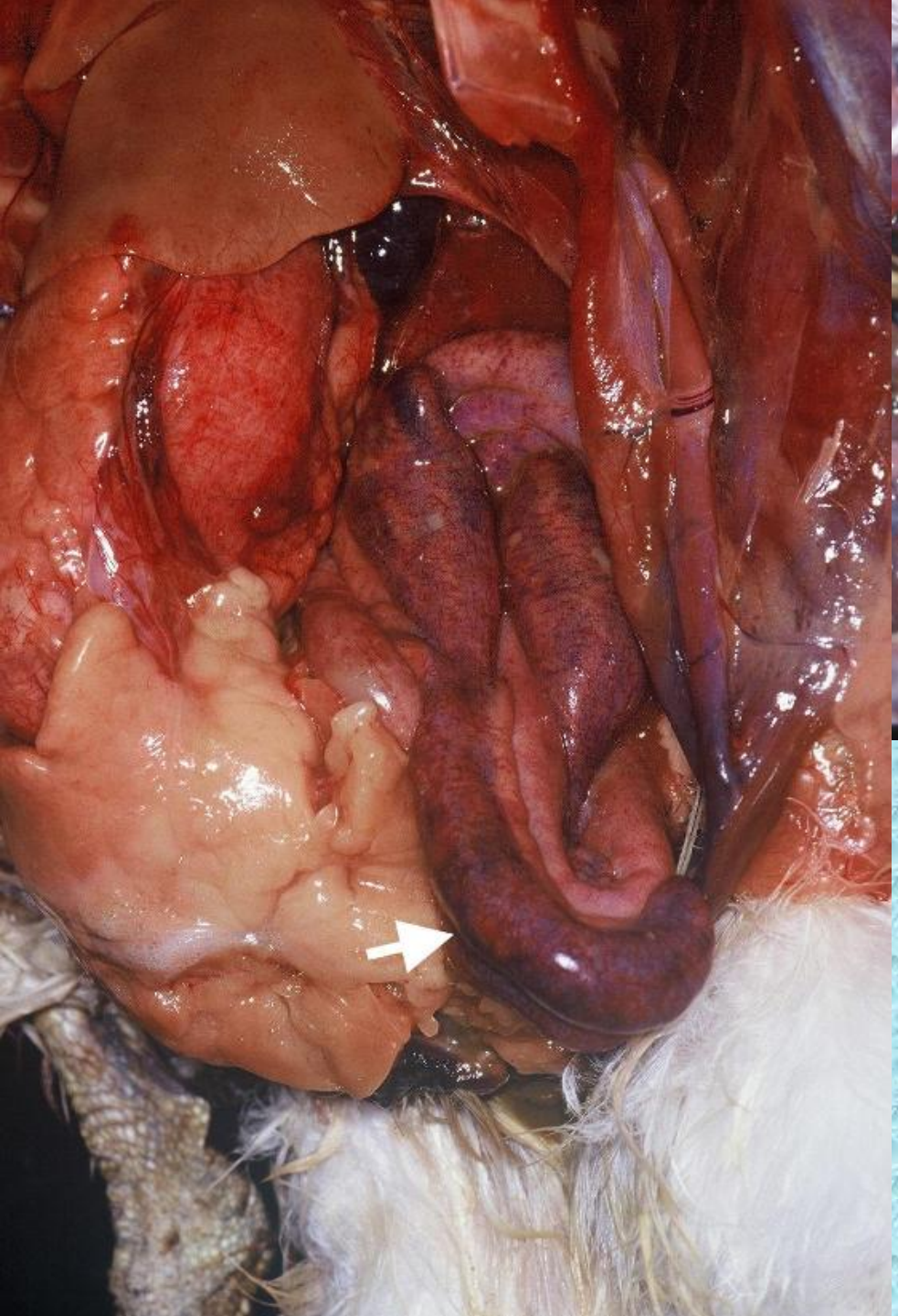
- Cytology and microbiological culture of aspirate, washings.
- Chlamydia ELISA.
- Rule out protozoal (*Trichomonas* spp.) and viral (PMV-1) causes.

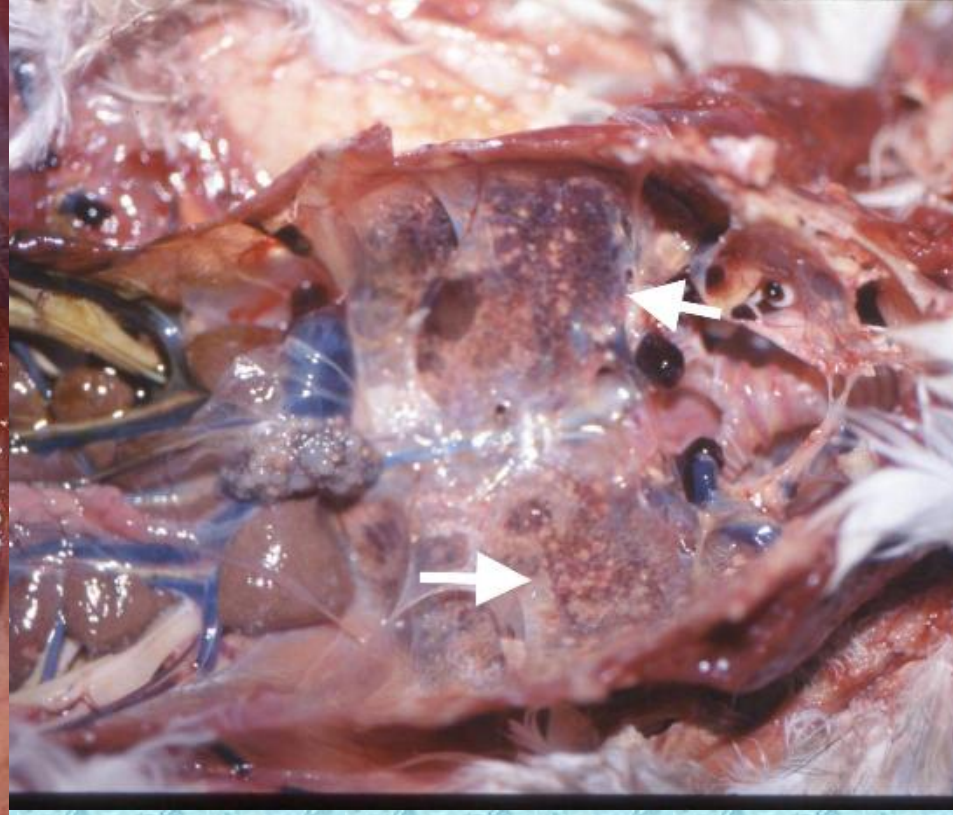
**Treatment**

- Antibacterial
- Sinuses may need to be flushed
- Antibiotic sensitivity essential (resistant *Pseudomonas* spp.)

# Pseudomonas sinusitis

- **Clinical signs**
  - mucopurulent nasal/choanal discharge, mucoid ocular discharge, coughing, choanal inflammation, sinus swelling and anorexia. Morbidity of 30% in one outbreak at NARC.
- **Predisposing factors**
  - nutritional deficiencies, inappropriate antibiotic use, transportation, age, contaminated water, temperature extremes.
  - concurrent infections with viruses are also affect susceptibility to *Pseudomonas* spp. infection .
- **Treatment tips**
  - medicate for one week after clinical signs disappear, relapses common





Pseudomonas serositis
and lung consolidation

# Diseases of Bustards

- Viral
- Bacterial
- Fungal
- Parasites
- Multifactorial

# Aspergillosis

**Cause**

- *Aspergillus* spp
- Immunosuppression is an important factor in chronic aspergillosis.
- Other predisposing factors include malnutrition, pre-existing disease and unhygienic environmental conditions.
- Common in imported HB (22% mortality).

**Clinical signs**

- Weight loss, anorexia, dyspnea, depression.

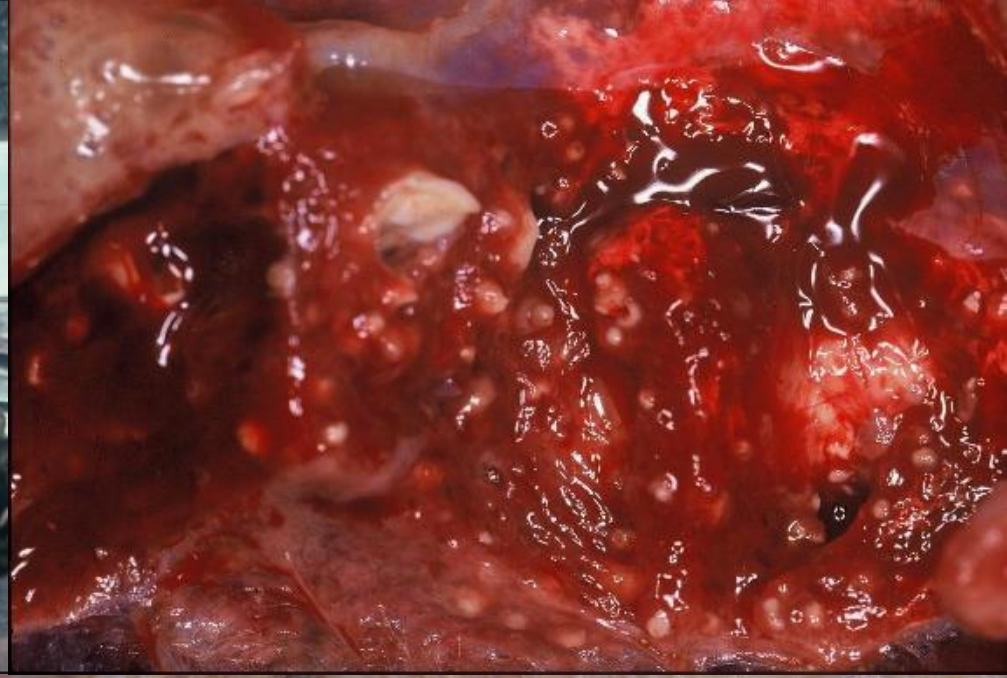
# Aspergillosis

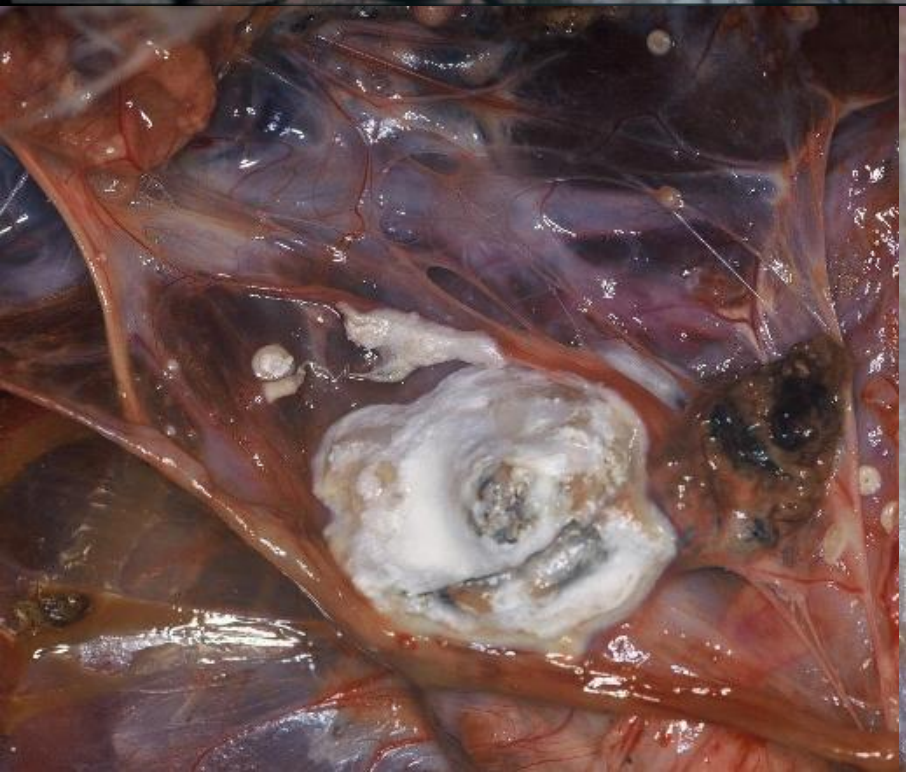
**Diagnosis**

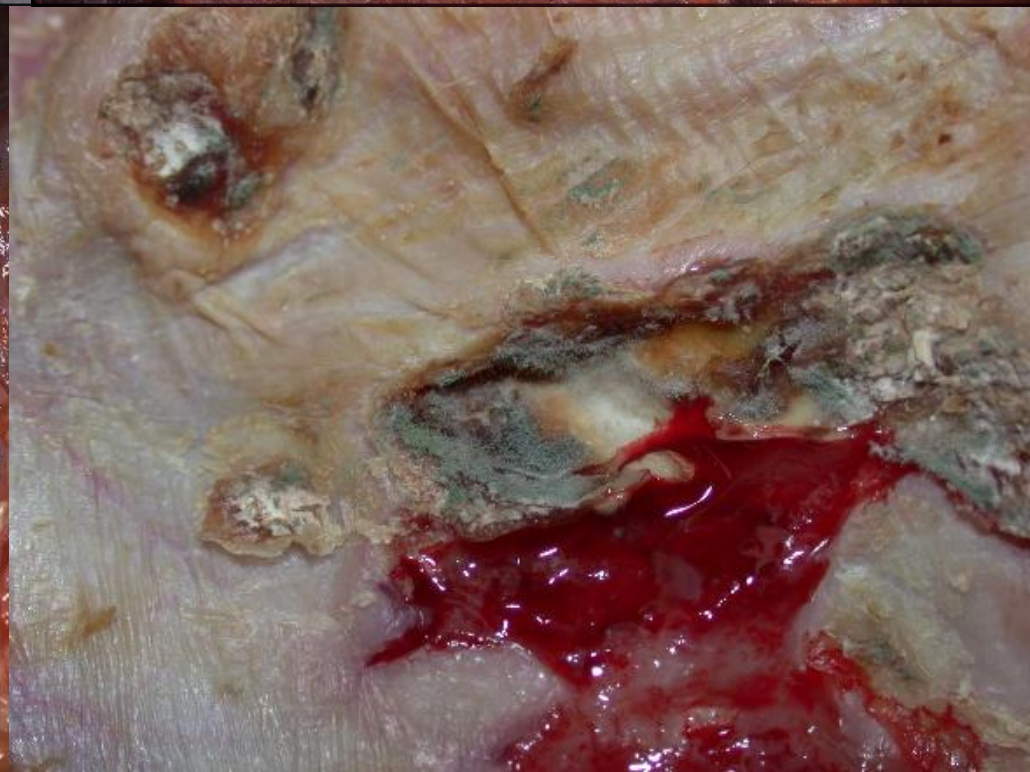
- Tracheal culture.
- Radiography.
- Haematology.
- Endoscopy and biopsy (cytology, culture).

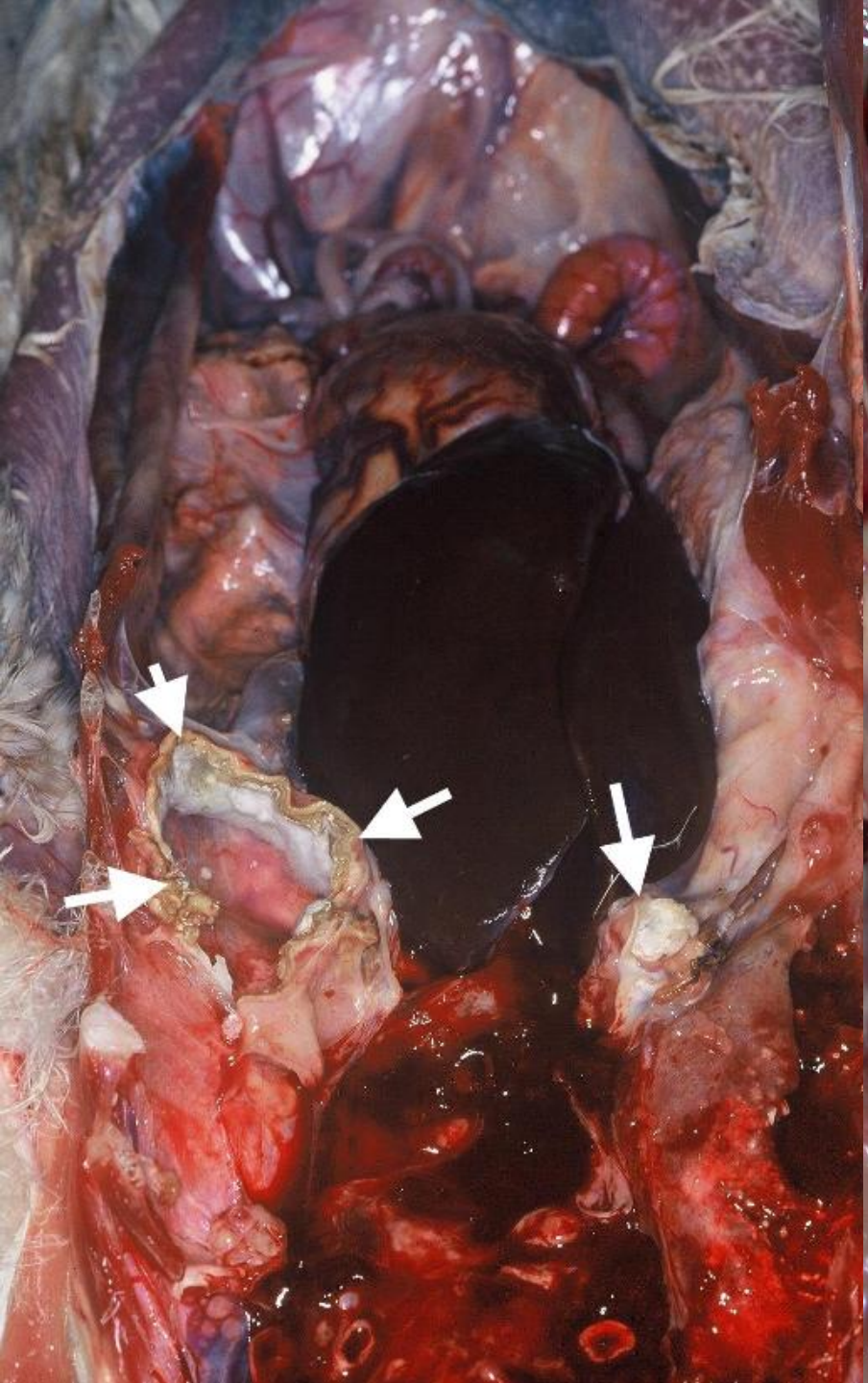
**Treatment**

- Individual medical treatment using antifungal agents and supportive therapy.
- Good management important to prevent this condition









# Candidiasis

**Cause**

- *Candida albicans*
- Predisposing factors include immunosuppression, oral trauma, antibiotic therapy, bad hygiene, systemic illness.

**Clinical signs**

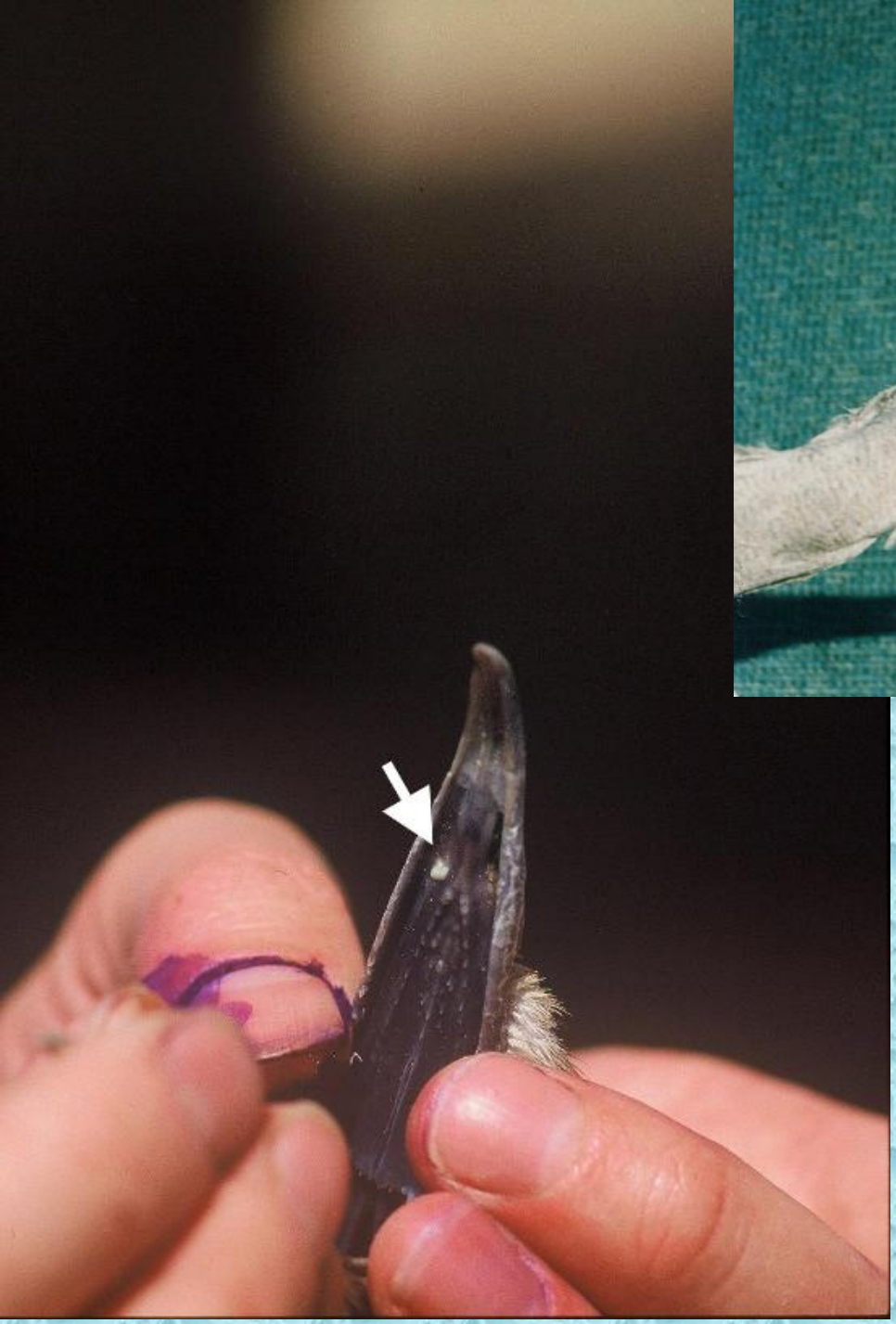
- Anorexia, loss of condition, OP mucosa covered with soft, white, cheesy material.

**Diagnosis**

- Culture and smears showing oval yeast cells and hyphae.

**Treatment**

- Antifungal agents



# Diseases of Bustards

- Viral
- Bacterial
- Fungal
- **Parasites**
- Multifactorial

# Lice infestation

**Cause**

- *Mallophaga* spp

**Clinical signs**

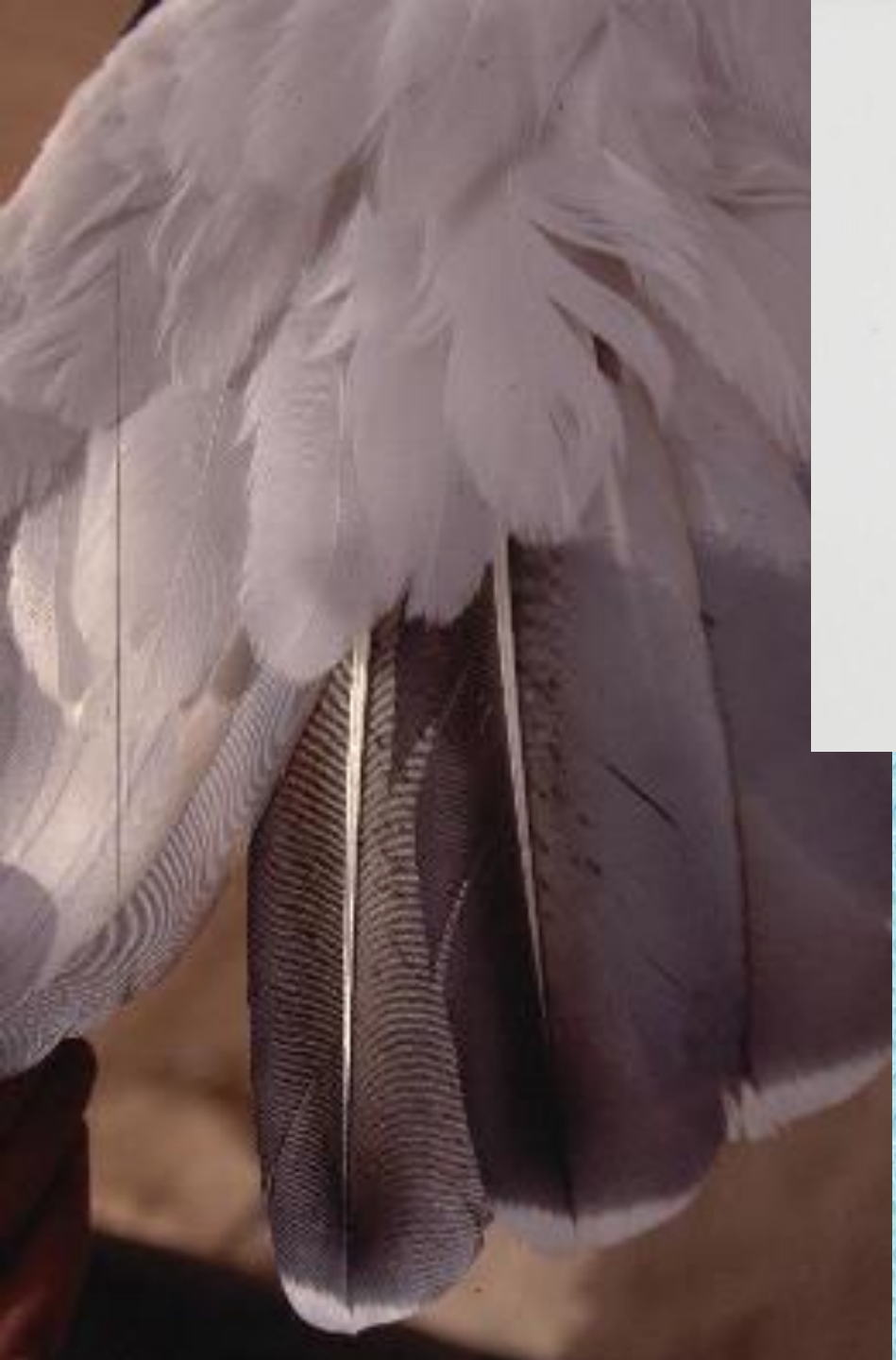
- Asympomatic to ragged plumage
- Heavy burdens seen when birds in poor condition and kept in crowded environment

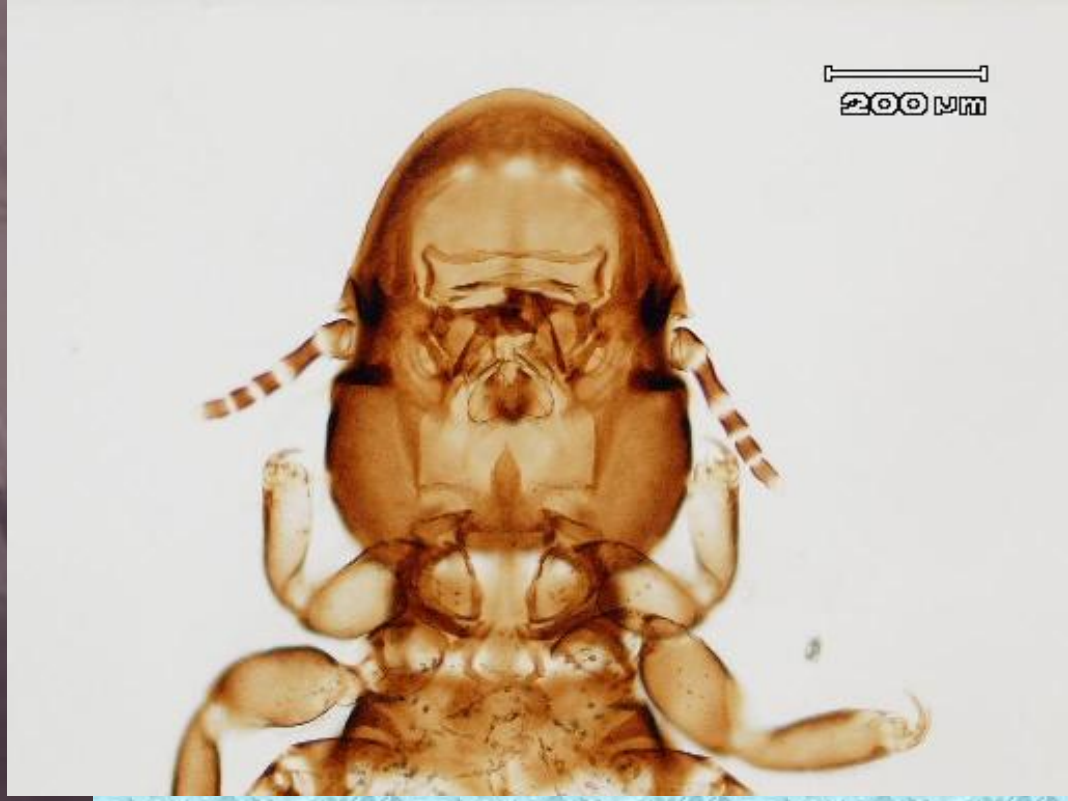
**Diagnosis**

- Detection esp. on ventral aspect of wing

**Treatment**

- Insecticidal agents




200 µm

# Tapeworm infestation

**Cause**

- *Otiditaenia* spp., *Raillietina* spp., *Idiogenes* spp., *Ascometra* spp., *Hispaniolepis* spp. have been recovered in the UAE.
- Common - intestinal parasites found in 34% imported HB and 16% captive HB in one survey.

**Clinical signs and pathology**

- Rarely pathogenic - partial obstruction of GIT lumen
- General debility, sometimes diarrhea and anorexia.
- Heavy burdens can cause inflammation, mild atrophy, collapse and fibrosis of the intestinal mucosa.

# Tapeworm infestation

**Diagnosis**

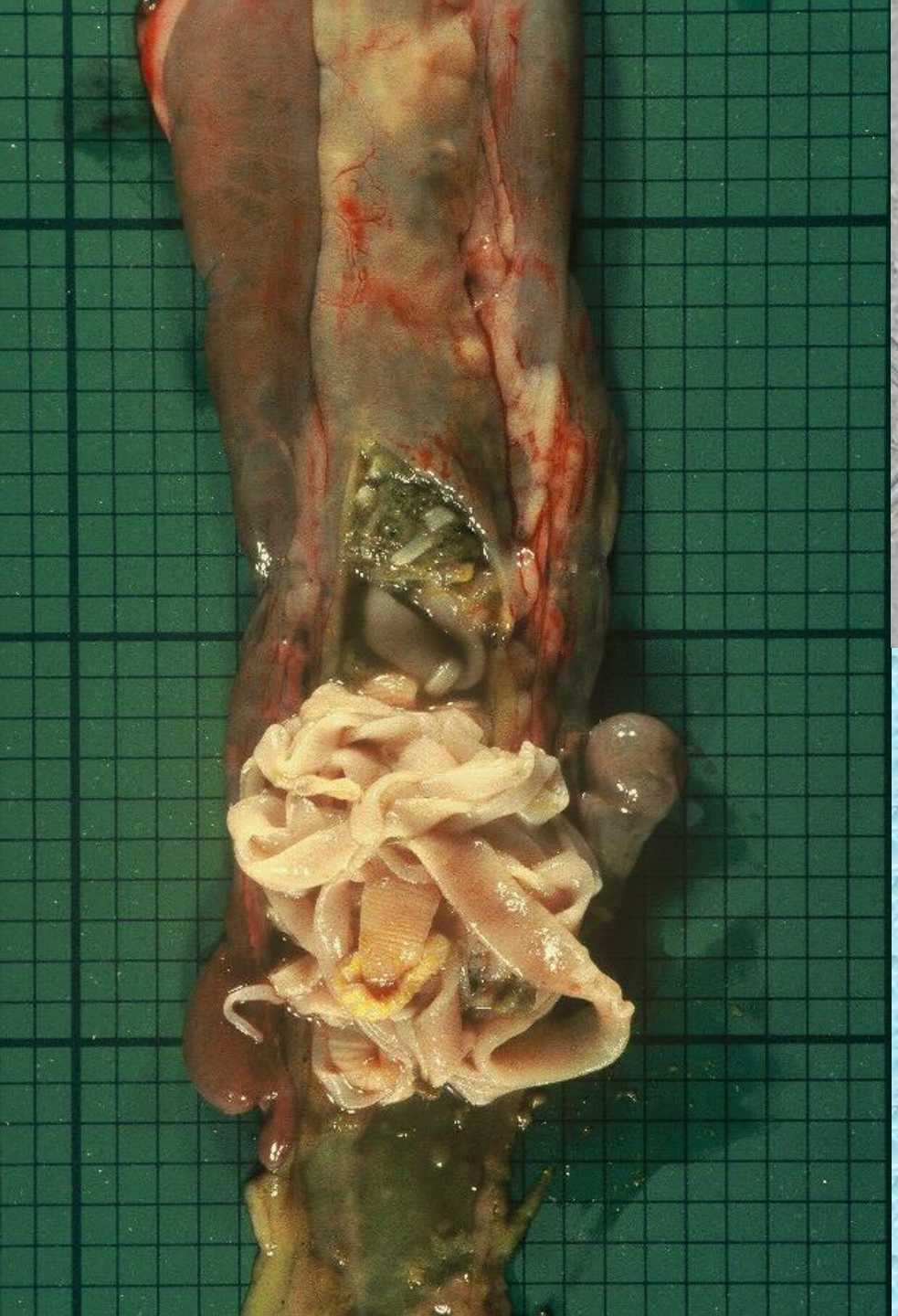
- Finding proglottids or expelled parasites in faeces

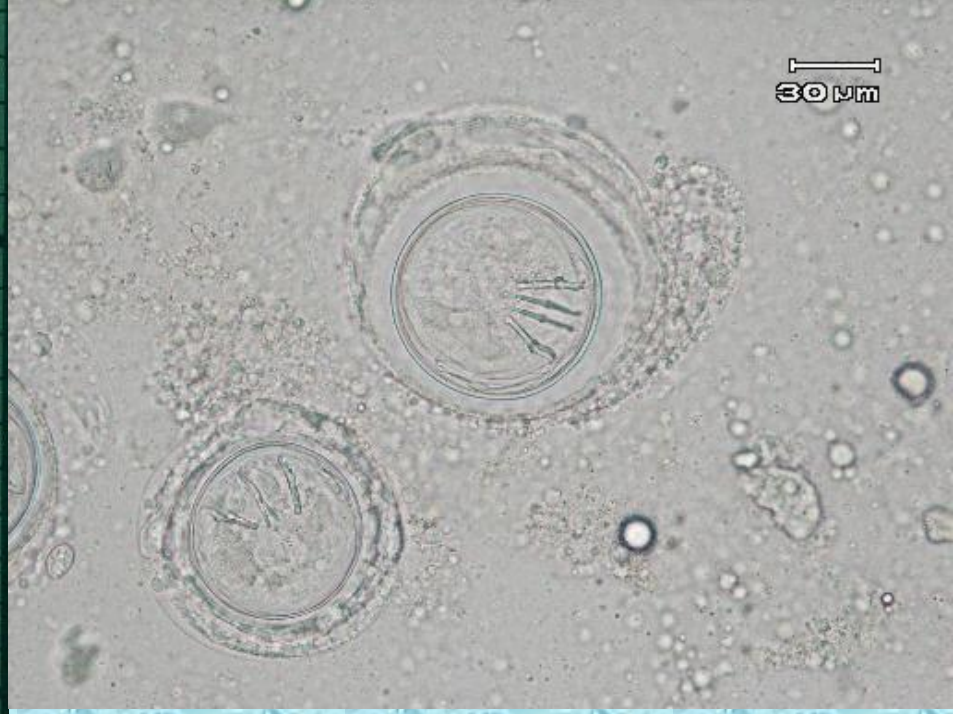
**Treatment**

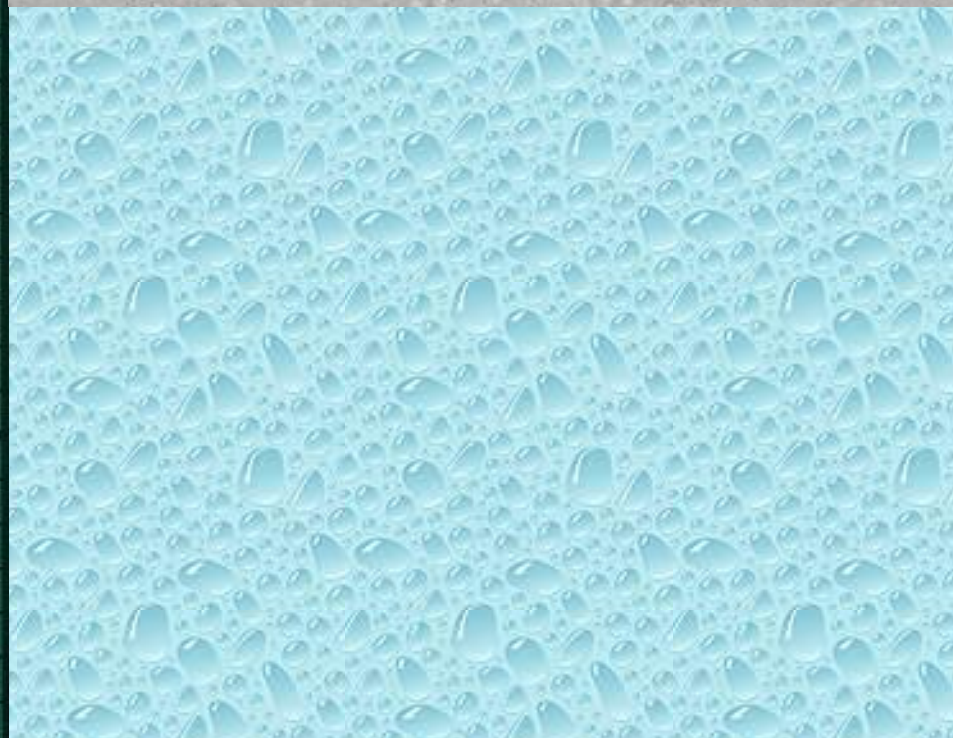
- Anthelmintics

Note: heavy burdens seen when birds in poor condition and kept in crowded environment or recently imported. Also seen commonly in birds housed in naturalistic aviaries. Helminths shown to affect reproductive productivity of gamebirds. Control important in CBRPs

- See J Zoo and Wildl Med 1996: 27; 201-208




30 µm



# Trichomoniasis

**Cause**

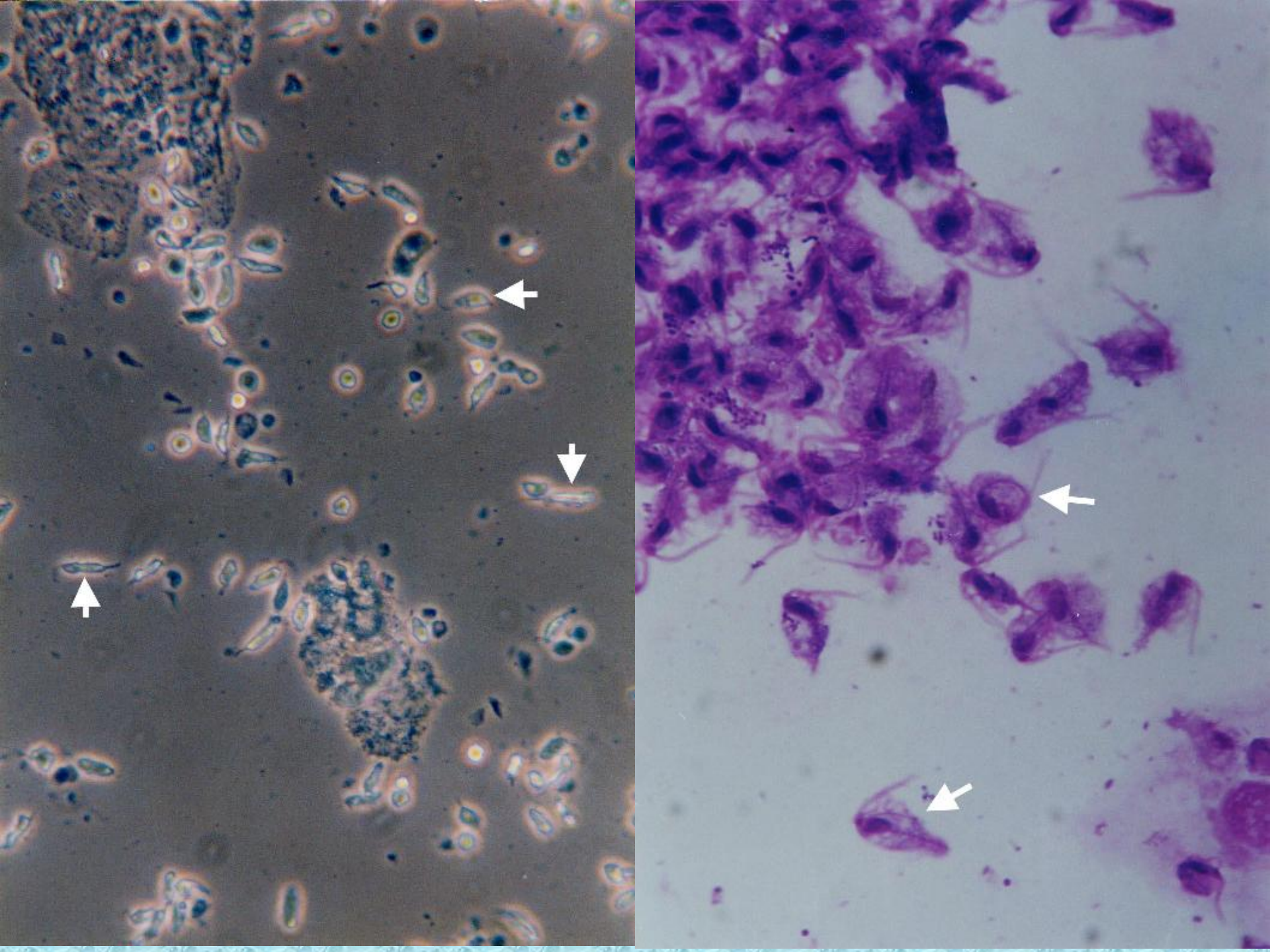
- *Trichomonas gallinae*
- Common finding in captive (4%) and imported (8%) HB.

**Clinical signs**

- Inappetance, dribbling, OP swelling, dyspnoea, unthriftiness, weight loss, white cheesey material in OP, oesophagus.
- Less commonly - sinusitis, otitis, respiratory tract.

**Diagnosis**

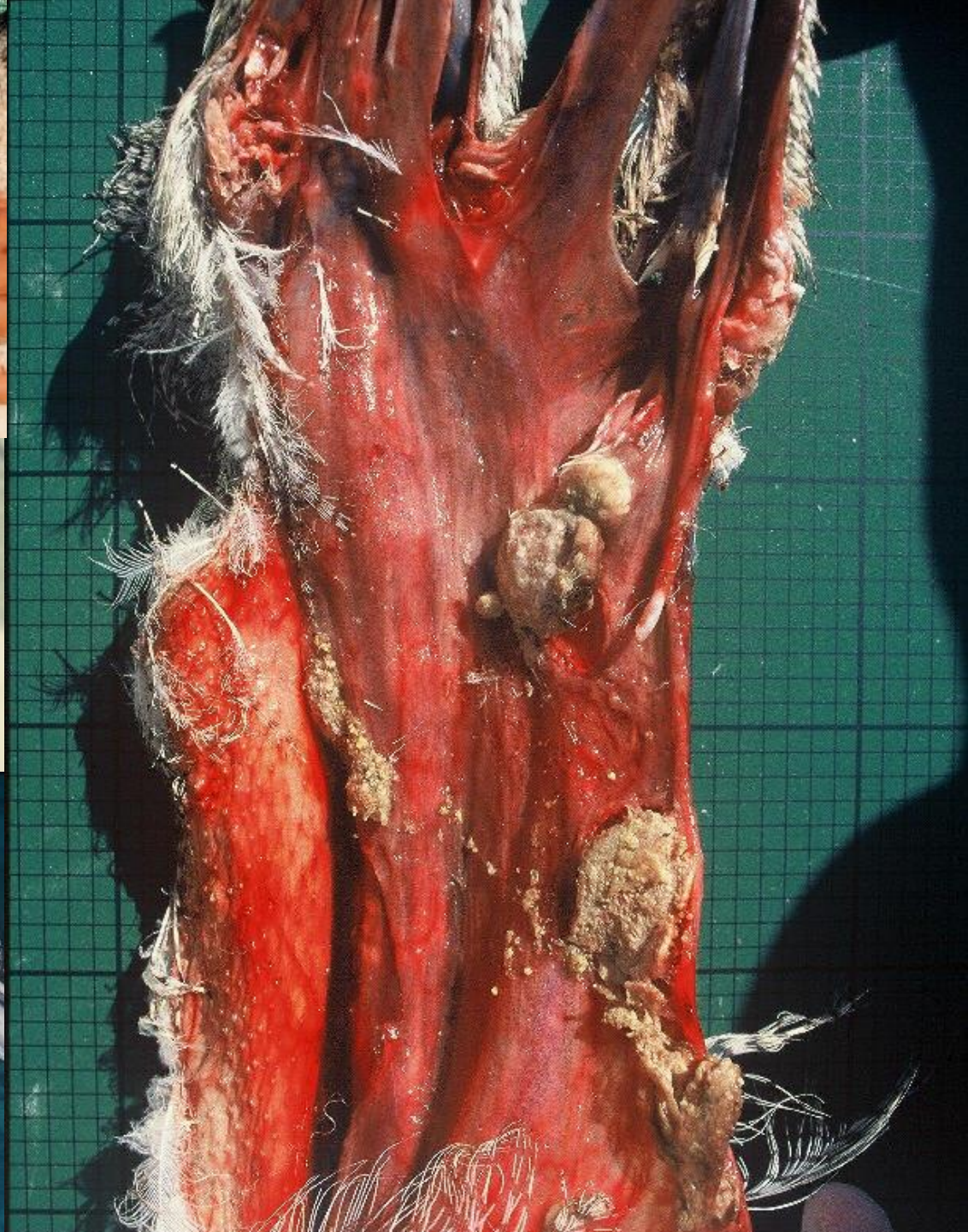
- Microscopic examination of lesions for motile parasite.



# Trichomoniasis

**Epidemiology**

- Infection follows contact with food or water contaminated by feral birds. Pigeons are asymptomatic carriers.
- Seasonal distribution (UAE). Most cases seen in February, April, May and October-December. Lowest number of cases seen in July and August, when the summer temperature in the Middle East reaches its peak.
- Trichomonas organisms are not able to survive unfavourable conditions, they are killed instantly on drying.



# Microbiological findings from oro-pharyngeal trichomonas lesions.

| **Pathogenic bacteria** | **Non-pathogenic bacteria** |
|---|---|
| *Staphylococcus aureus* | *E. coli* |
| *Streptococcus viridians* | *Enterobacter* spp. |
| *Pseudomonas aeruginosa* | *Klebsiella* spp. |
| *Haemophilus* spp. | *Citrobacter* spp. |
| | *Staphylococcus sciuri* |
| | *Proteus* spp. |
| | *Aeromonas hydrophila* |
| | *Micrococci* spp. |
| | *Sarcinia lutea* |
| | *Spingomonas paucimobilis* |

# Trichomoniasis

**Medical treatment**

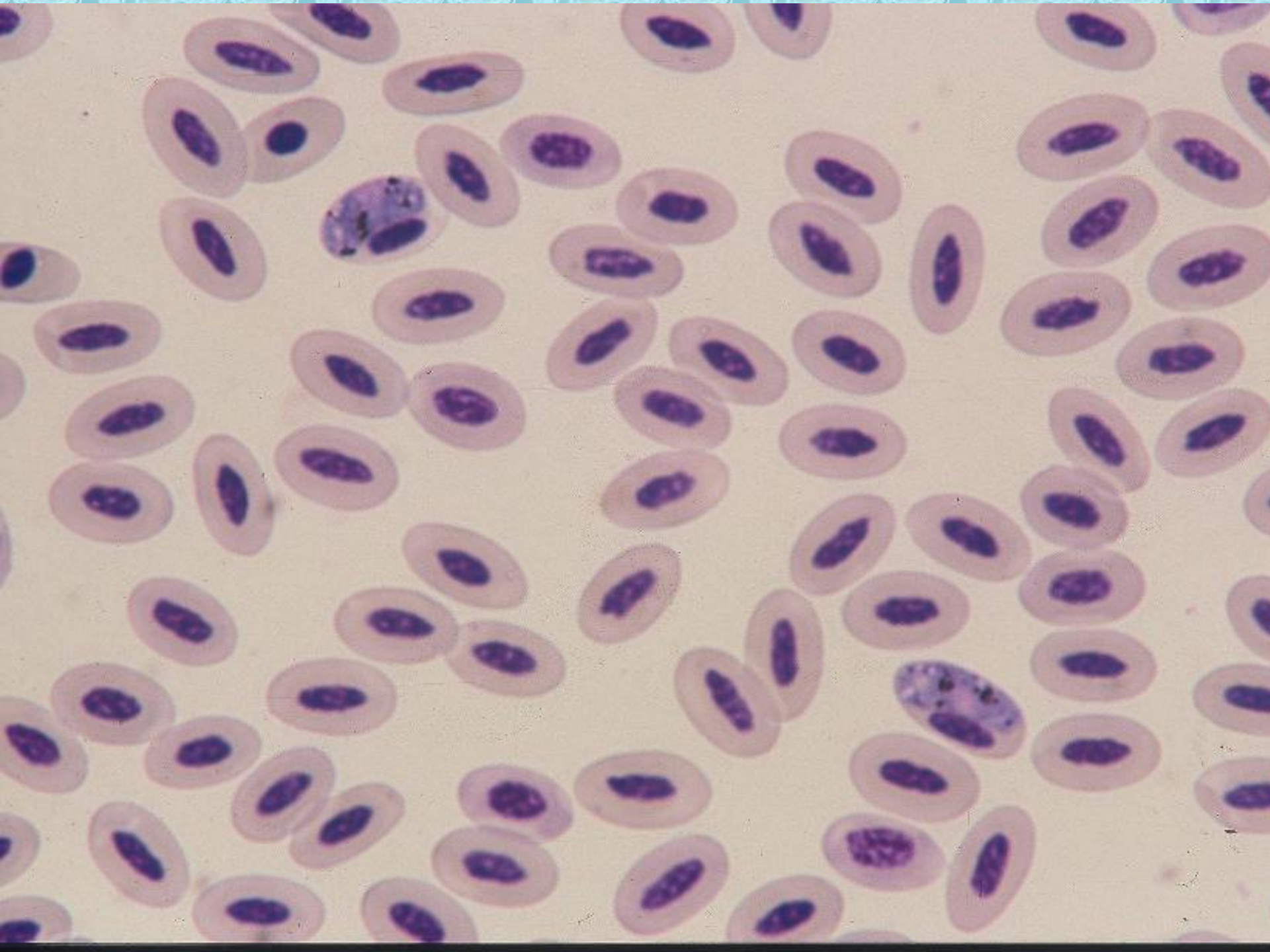
- Antiprotozoal agents (individual and flock).
- Often secondary bacterial (fungal) infections so provide supportive treatment (antibiotics, fluid therapy).

**Management treatment**

- Exclusion or control of wild birds from aviaries.
- Treat all birds on premises as asymptomatic carriers exist.
- Regular prevention, in badly affected aviaries water medication every other month may be necessary.
- Note: common disease of bustards, but kori bustards appear to be particularly susceptible.

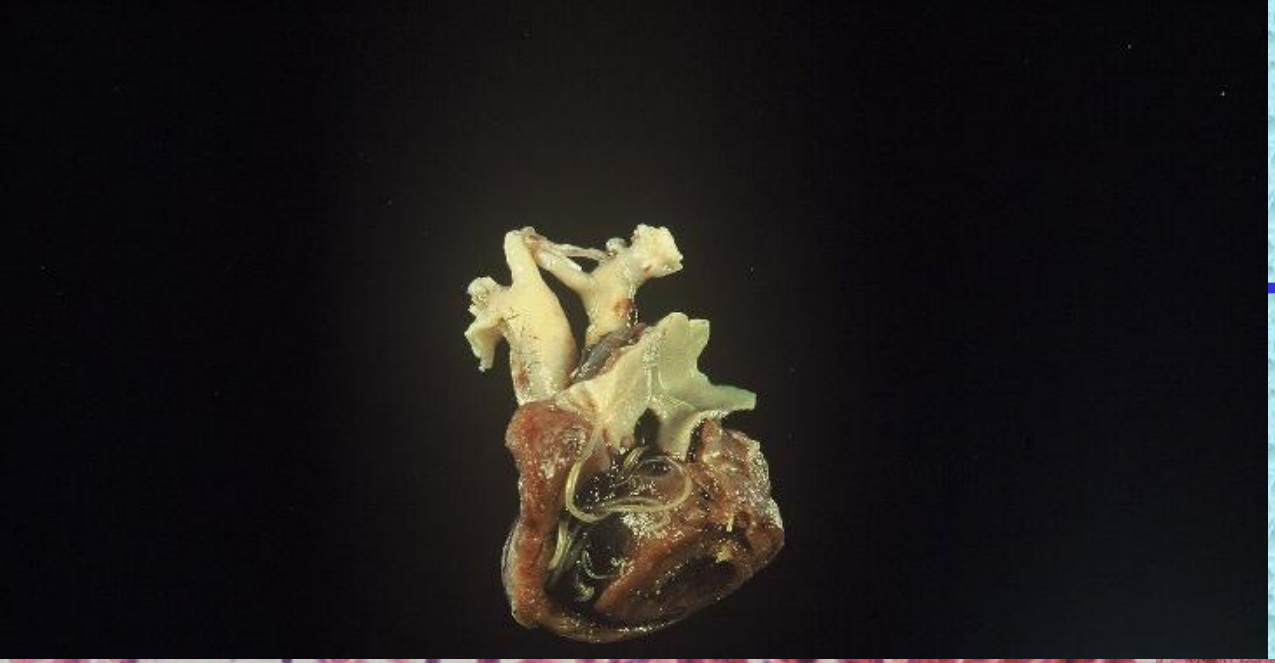
# Blood parasites

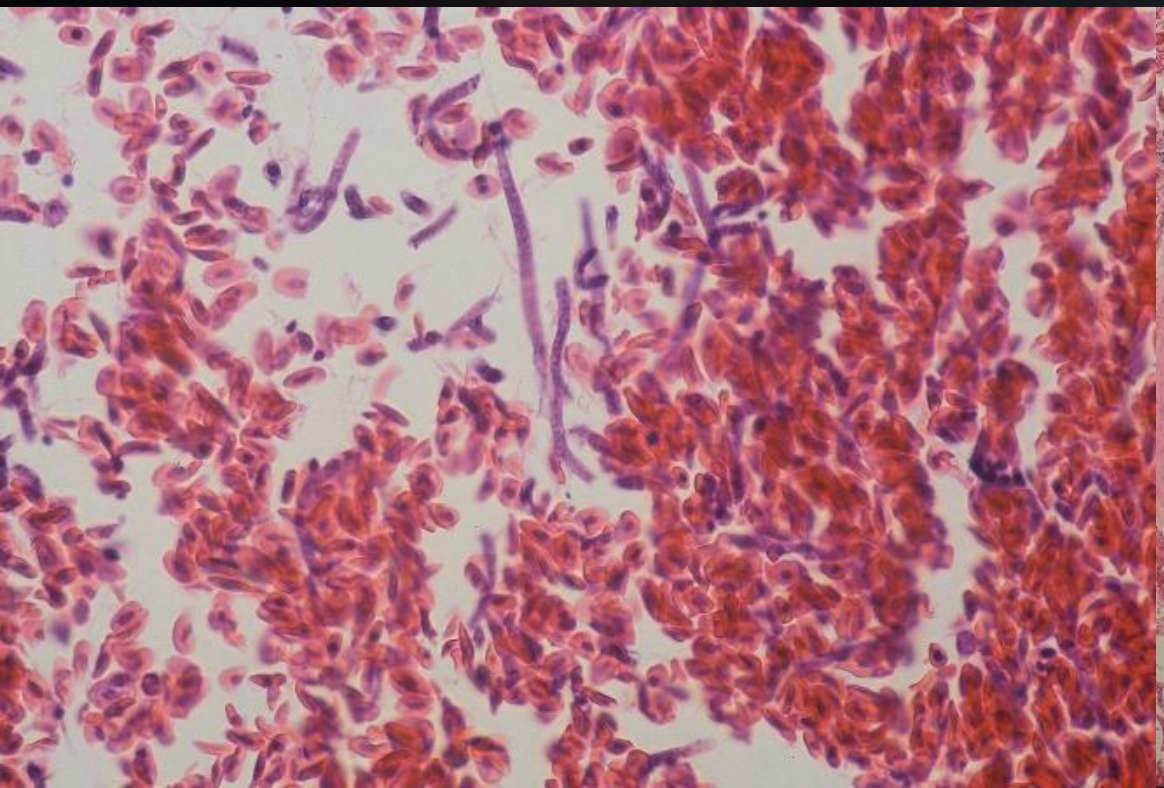
- Hameoproteids = intracellular sporozoan parasites which complete their life cycle in tissues and blood
- *Haemoproteus tendeiroi* and *H. telfordi*
- Prevalence of 61.5% in freshly imported HB, ex wild HB have low level parasitism (0.05% rbcs) after 7-10 years in captivity in UAE. Vectors are usually Diptera
- Captive-bred HB at NARC not infected (Vector absent).
- Not usually pathogenic, but latent infections can be triggered by stresses (e.g. Aspergilllosis)
- See: Avian Pathol. 1996: 25;49-55

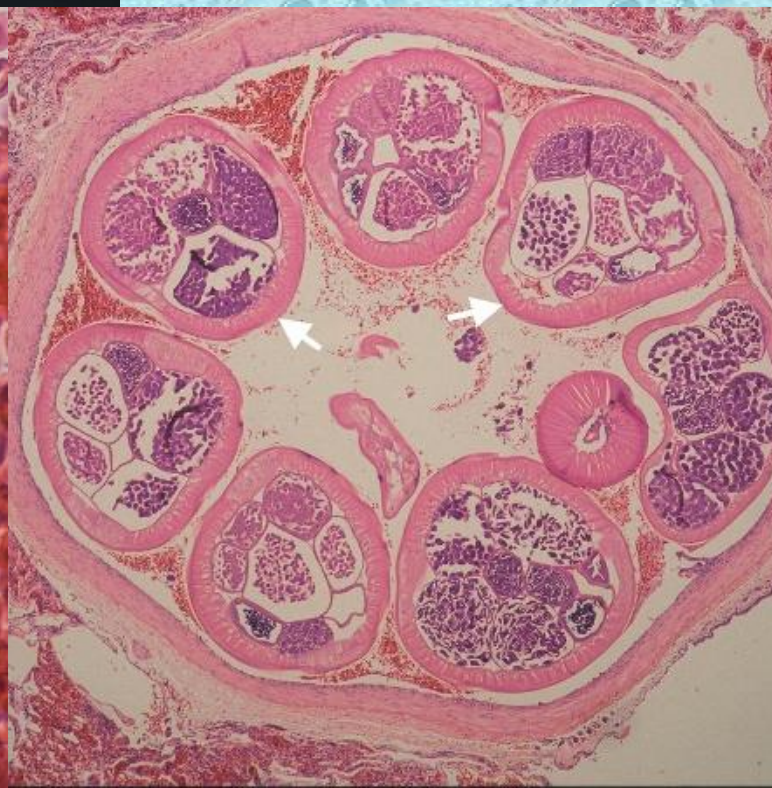


# Heartworm infestation

- Heartworms (Paroncherca spp.) are filarial parasites
- Found vascular system (heart, vena cava)
- Associated with cardiac rupture in imported rufous-crested bustards
- Prominent myocardial degeneration and fibrosis associated with microfilaria (histopathology)
- In other avian species heartworm associated with weight loss.
- Treatment is levamisole
- See: Journal of Zoo Wildl. Med. 1995: 26; 590-586

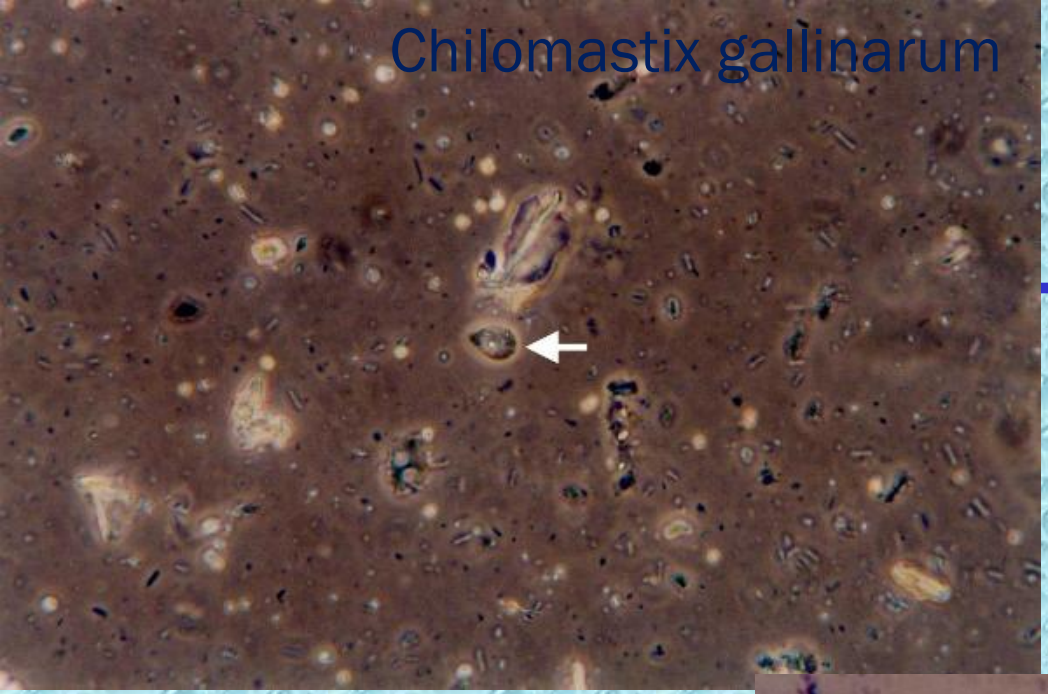


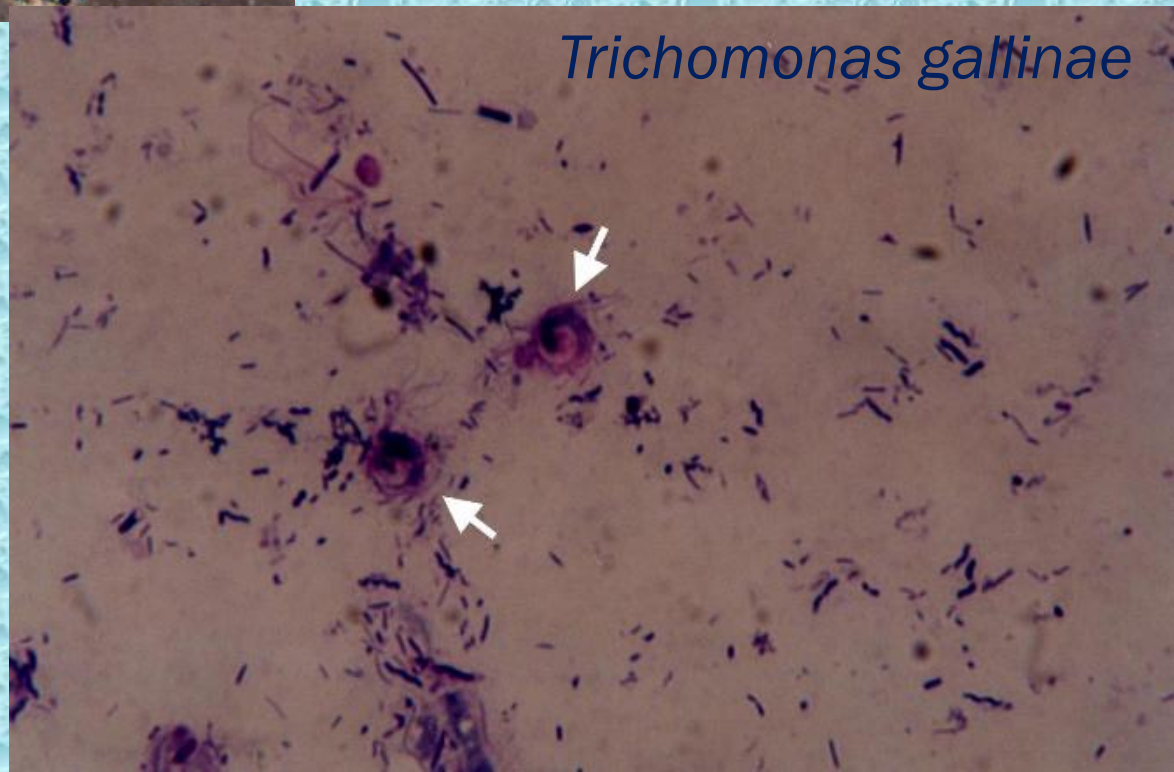




# Protozoal Infections

- **Intestinal protozoa**
- Survey at NARC -protozoa detected in fecal samples from 43% birds;
- *Trichomonas gallinarum* (30%) no clinical signs
- *Chilomastix gallinarum* (11%) no clinical signs
- *Lophomonas* spp.(1%) no clinical signs
- *Giardia* spp. (1%) associated with diarrhoea
- *T.*and *C. gallinarum* normal flora of lower GIT
- See: Avian Pathol 1998: 28; 94-97


Chilomastix gallinarum


Trichomonas gallinae

# Protozoal Infections

- Oropharyngeal protozoa
- Survey at NARC -protozoa detected in OP samples from 180 normal birds and 20 birds with clinical signs
- *Endolimax* spp., *Entamoeba gallinarum*, *Acanthamoeba* spp., *Coccidia* spp. isolated from normal birds
- *Trichomonas gallinae* and *Entamoeba anatis* associated with clinical disease
- Protozoal disease assoc with secondary bacterial infection (*Pseudomonas* spp. and *Staphylococcus* spp.)
- See: Avian Pathol 1998: 27; 526-530

# Diseases of Bustards

- Viral
- Bacterial
- Fungal
- Parasites
- Multifactorial

# Fatty liver syndrome

- FLS  is an accumulation of XS lipid in hepatocytes than would normally be expected.  As fat levels increase, liver function decreases.  Severe fatty liver results in liver failure and death.

**Cause**

- Occurs when hepatic lipoprotein production impaired.
- Nutritional causes (protein malnutrition, choline deficiency) lead to inhibition of lipid transport.

**Epidemiology**

- Important problem in early years of NARC (1993-1995).
- Historical prevalence of 47% (n=72) in HB.

# Fatty liver syndrome

**Clinical signs**

- Depression, sudden death, aphagia, recumbancy, paralysis, reduced growth, increased flock mortality/morbidity.

**Diagnosis**

- Endoscopy (biopsy), blood chemistry (L Gluc, H AST, L TP, L Alb), radiography.
- PM finding - friable haemorrhagic liver.
- Confirm by histopathology

**Treatment**

- Well balanced protein diet with adequate methionine and choline levels.

# Fatty Liver Disease

- FLKS (in poultry)
  - High energy diet, positive energy balance & restricted exercise
  - Fat deposition in liver & elsewhere
  - High temperatures, low exercise levels, reduced heat loss compound the problem

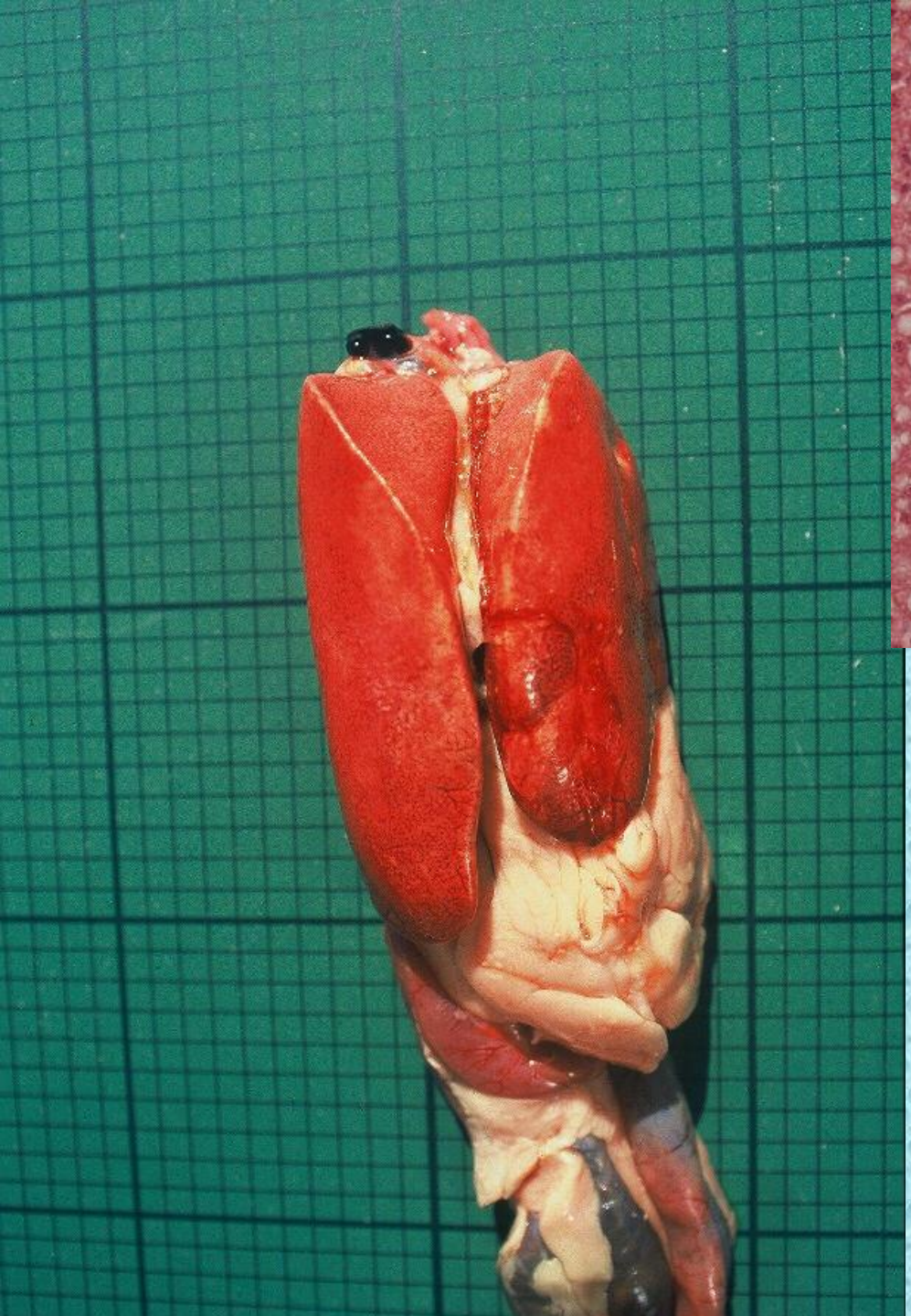
- Clinical signs (other species)
  - Depression
  - Sudden death
  - Reduced growth
  - Lethargy
  - Aphagia
  - Recumbency
  - Sometimes paralysis

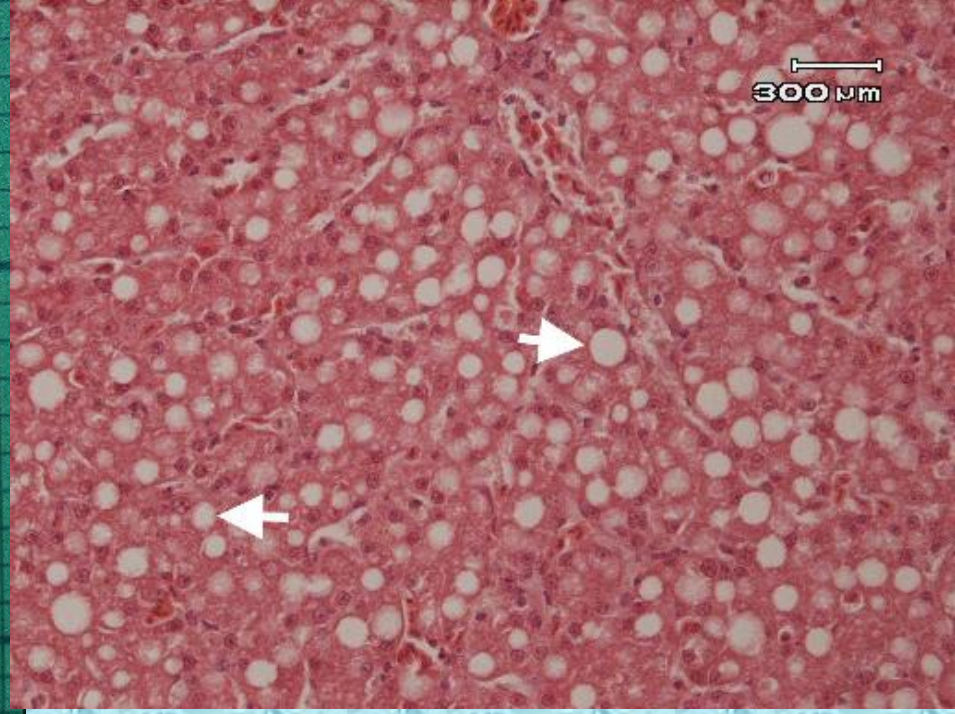
# Fatty liver syndrome

- **FLS in Bustards**
- In HB associated with diet (irregular + quality), condition, and housing (stocking density), capture, transport and translocation.
- Prevention - reduce transport/capture stress, Vit E/Se supplementation of diet and preventive treatment of water before capture, avoid heat stress.
- Further work to determine dietary components (Choline, biotin, Vit E, Se) is warranted.
- See: Avian Pathol. 1997: 26; 19-31

# Fatty Liver Disease

- Can be reversible, but pigment (ceroid) accumulation, fibrosis & nodular hyperplasia
- In poultry tx of FLKS with
  - Oral or injectable biotin
  - Oral choline, inositol, vitamin E / selenium & vitamin B12
- Tx in psittacines involves
  - Dietary correction (reducing fat in the diet)
  - Supplementing diet (vitamins, amino-acid & trace minerals)
  - Vit E a useful additive to prevent free-radical damage to hepatic cell membranes
  - Trace minerals considered important as catalysts
  - Lactulose considered useful in reducing blood ammonia levels






Marked vacuolation in fatty liver

# Capture Paresia

- Known by a spectrum of names including;
- Muscular dystrophy, capture disease, capture myopathy, degenerative polymyopathy, overstraining disease, white muscle disease, leg paralysis, muscle necrosis, idiopathic muscle necrosis, exertional rhabdomyolysis, stress myopathy, transit myopathy, diffuse muscular degeneration, and white muscle stress syndrome
- Common condition in early days of NARC accounting for 2-3% of morbidity in one survey.

# Capture Paresia

**Definition**

- Paresis is defined as "*slight or incomplete paralysis, and includes animals that can make purposeful attempts to rise without being able to do so, those that are able to rise with assistance, those that are able to rise and walk with major difficulty including frequent falling, and those able to stand and walk without assistance, but with slight errors*."

# Capture Paresia

Reported in;

Greater and lesser flamingos

Secretary bird

Ostriches, emus and rhea

Bar-headed godwits

Sandhill cranes

Whooping cranes

Canada geese

Wild and domestic turkeys

East African crowned cranes

Houbara, kori and rufous-crested bustards

# Capture Paresia

**Pathogenesis**

- Anaerobic metabolism during intense muscular activity.
- Lactic acid produced in muscle causes local and systemic acidosis resulting in the lesions and clinical signs of paresia
- Low pH in tissues results in cell lysis, releasing intracellular enzymes including creatine kinase (CK), lactate dehydrogenase (LDH) and aspartate aminotransferase (AST) to the blood
- Elevated concentrations of CK and AST in plasma reflect damage to skeletal and cardiac muscle. Elevation of CK is a sensitive and specific index of muscle damage in birds.

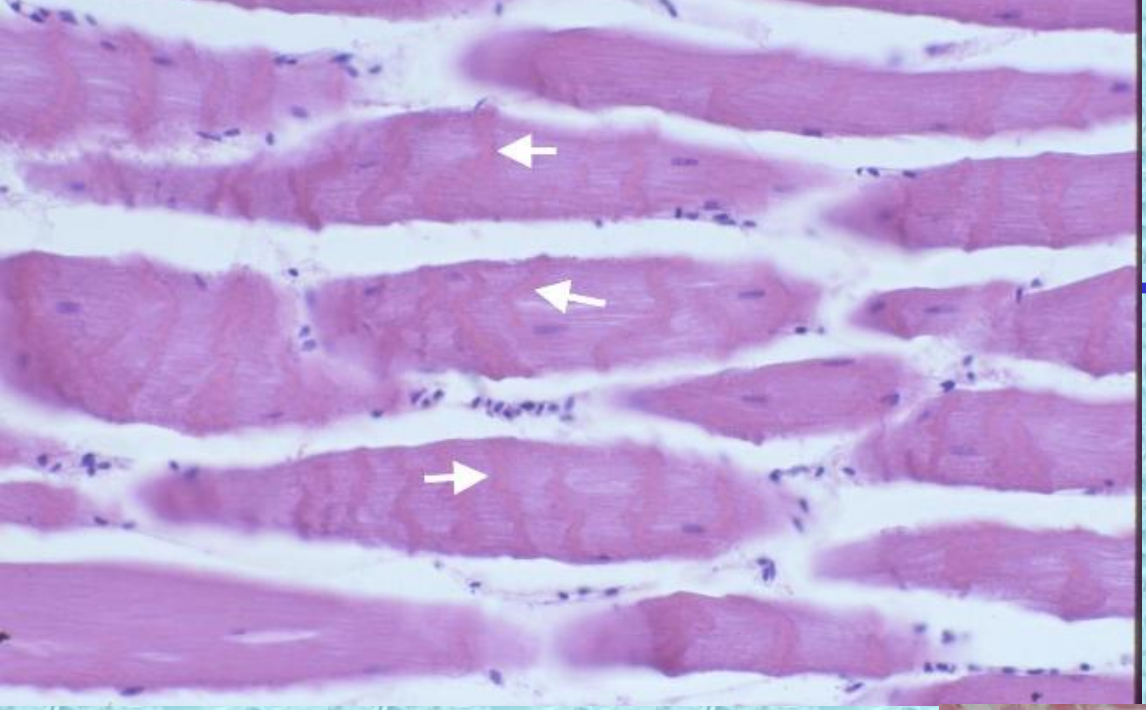
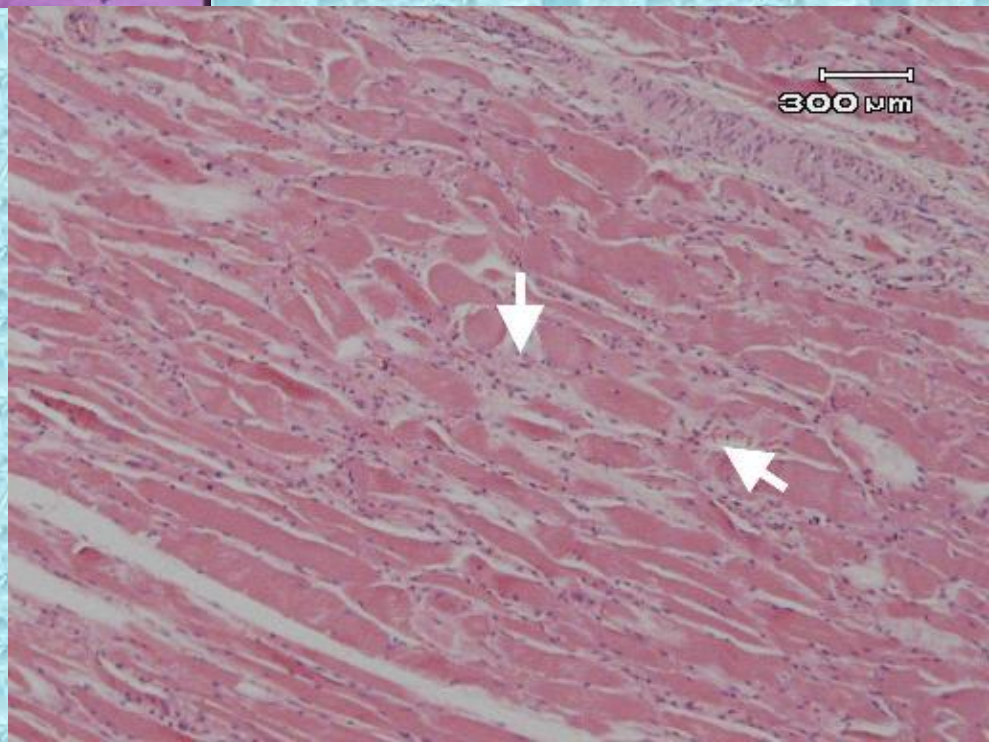


**Figure 20.14.** (above) Hypercontraction (arrows) of muscle fibres of a houbara bustard that died in the early stages of capture myopathy (H & E stain) (Photo credit CVRL).

**Figure 20.15.** (right) A more advanced stage of capture myopathy than 20.14, in a houbara bustard showing subacute necrosis (arrows) of muscle fibres (H & E stain) (Photo credit CVRL).



# Capture Paresia

## Clinical signs and history

- A number of factors are considered to predispose birds to CP including;
  - strenuous pursuit during capture operations.
  - prolonged handling.
  - translocation.
  - poor transport conditions.
  - possible vitamin E and selenium deficiencies.
  - intercurrent disease.
  - hot weather.
  - cramping of the limbs.

# Capture Paresia

**Clinical signs**

- depression.
- limb paresia or paralysis.
- hock-sitting.
- lateral or sternal recumbency.
- death during or after capture, handling or translocation.

- Acute death occurs in many cases and is thought to be caused by myocardial necrosis and trauma, while necrosis of the muscles of the thighs and flank causes limb paralysis.

# Capture Paresia

**Treatment**

- The primary goal is to control shock and hyperthermia;
  - Intravenous and oral sodium bicarbonate to correct acidosis.
  - Fluid therapy to restore blood pressure and volume.
  - Parenteral Vitamin E and selenium and multiple vitamins.
  - Corticosteroids.
  - Cool the bird if it is hyperthermic.
  - Cardiac and respiratory stimulants.
- Support affected birds in slings, physiotherapy.

# Capture Paresia

**Prevention**

- Recommendations for minimising the problem include;
- Supplemental vitamin E and selenium prior to capture.
- Capture birds on days that have acceptable environmental conditions.
- Keep handling times and struggling to a minimum and avoid hyperthermia.
- Transport in well-ventilated containers.
- Conditioning and training groups of animals can reduce the mortality associated with older exertionally dependent methods of capture.

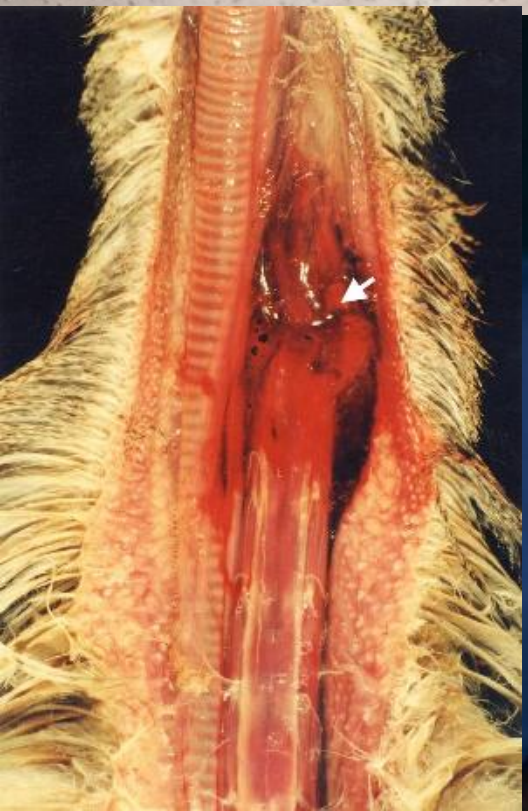
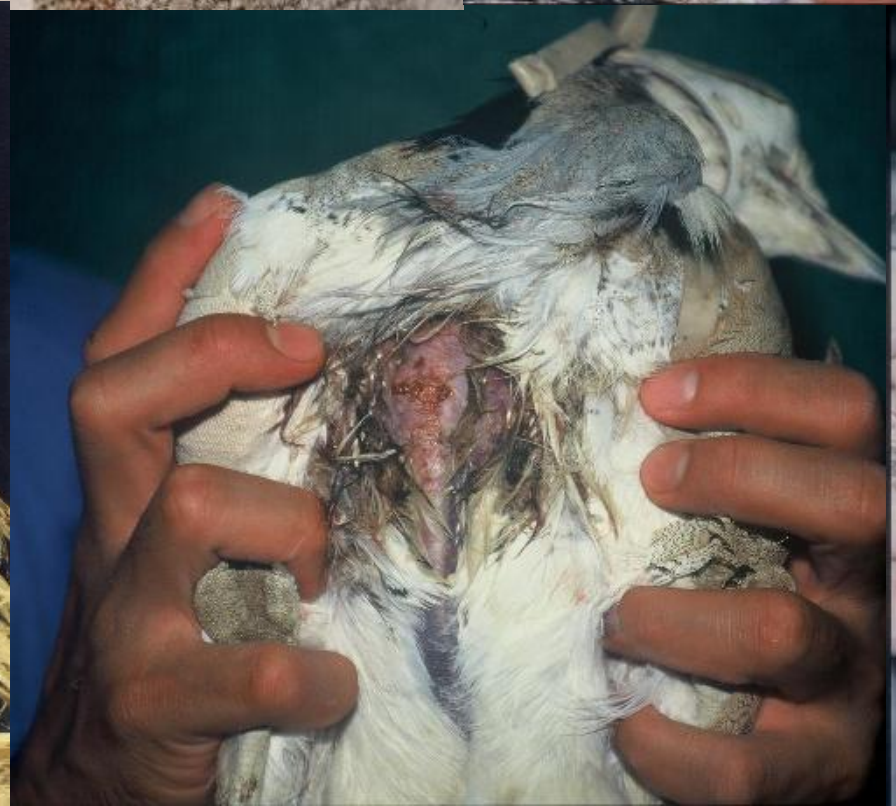
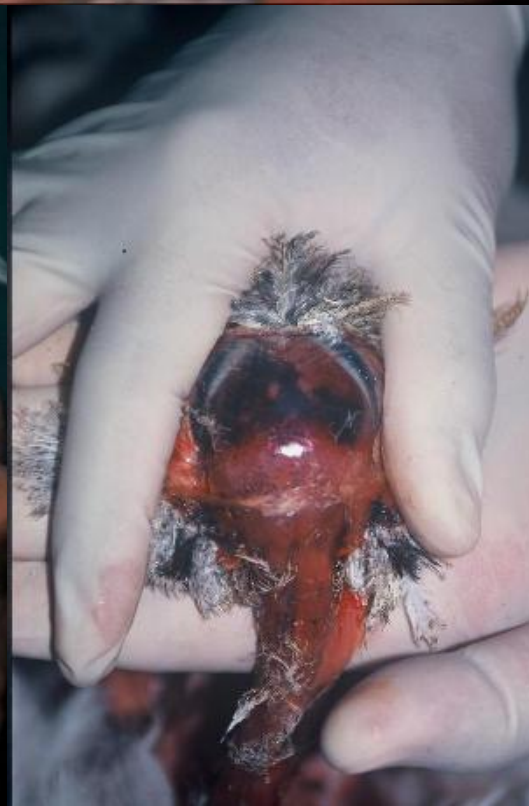
# Lead toxicosis

- Outbreak of lead toxicosis in HB associated with lead paint used in aviaries. Bustards ingest foreign material.
- Clinical signs - weight loss, weakness, depression, green diarrhoea, polyuria, anorexia, regurgitation, proventricular impaction, leg paralysis, ataxia, wing droop, head tremors.
- Confirmed by lead analysis (normal range <1mg/kg wet weight). In this outbreak lead levels of 47 mg/kg in the kidney and 397.5 mg/kg in the feces provided positive confirmation.
- See: Vet. Rec. 1995: 137; 193-194.

# Traumatic injuries

- Wing-tip trauma, keel trauma and other soft-tissue traumatic injuries are the most commonly diagnosed medical conditions of captive bustards.
- 50.7% of total causes of morbidity in captive adult bustards are soft-tissue trauma.
- 23.5% of deaths in captive adult HB and 57.1% of deaths of captive adult RCB caused by traumatic injuries
- Trauma is usually self-inflicted, but in some species there is intra-specific aggression (KB, RCB, WBB, BB).



# Traumatic injuries

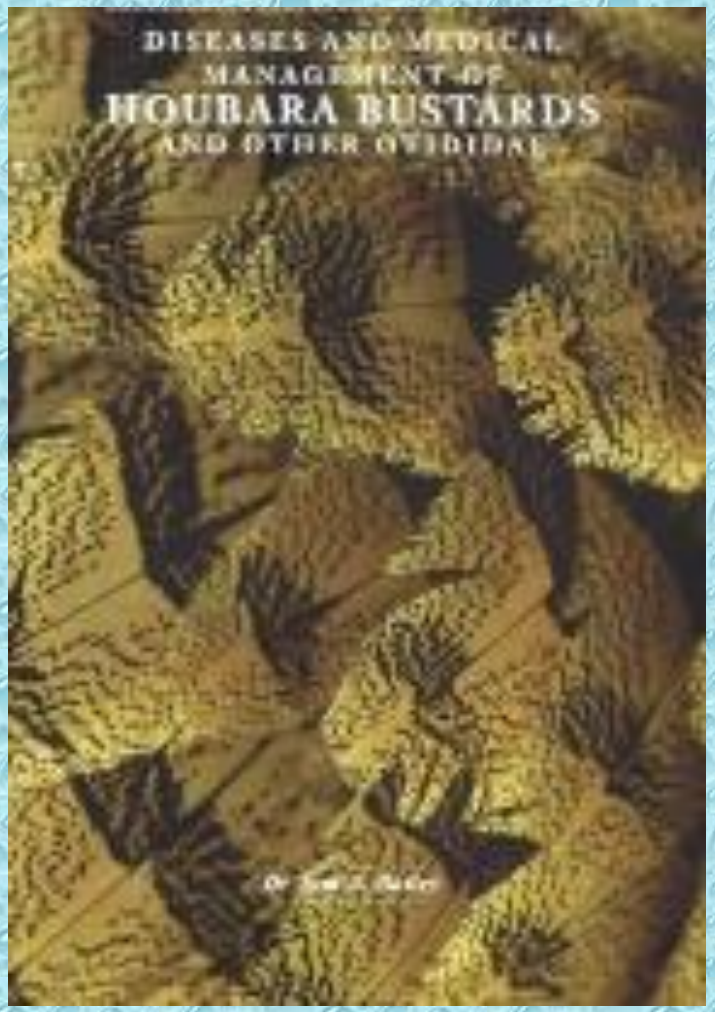
- Adult and juvenile bustards are “trauma susceptible”. Minimise trauma by;
- 1. Modifying behaviour by taming nervous individuals or housing such birds in naturalistic pens with cover.
- 2. Enclosure design using padding or shade-cloth to reduce potential danger zones.
- 3. Pinioning and feather cutting.
- 4. Consider genetics (migratory behaviour in captive setting).
- 4. Chemical modification of behaviour (future).

# Conclusions

- Cost of CBRPs and Rehabilitation projects are high
  - *Goal of these projects is to breed and release bustards that are not a health risk to themselves or to other animals that share the ecosystem*
- Avian disease risks in Middle East are high
- Investment in health management and veterinary research is an important component of any project

# Thanks

- Thanks to colleagues at :
  - NARC
  - DFH
  - IWC
  - ZSL
  - Who shared information with me over the years



كل ديك على مذبلته صيّاح

*Kull dīk 'alā mazbalatihi sayyāḥ*

***Every cock is a town crier on his own dung heap***

*Every cock will crow upon his own dunghill*

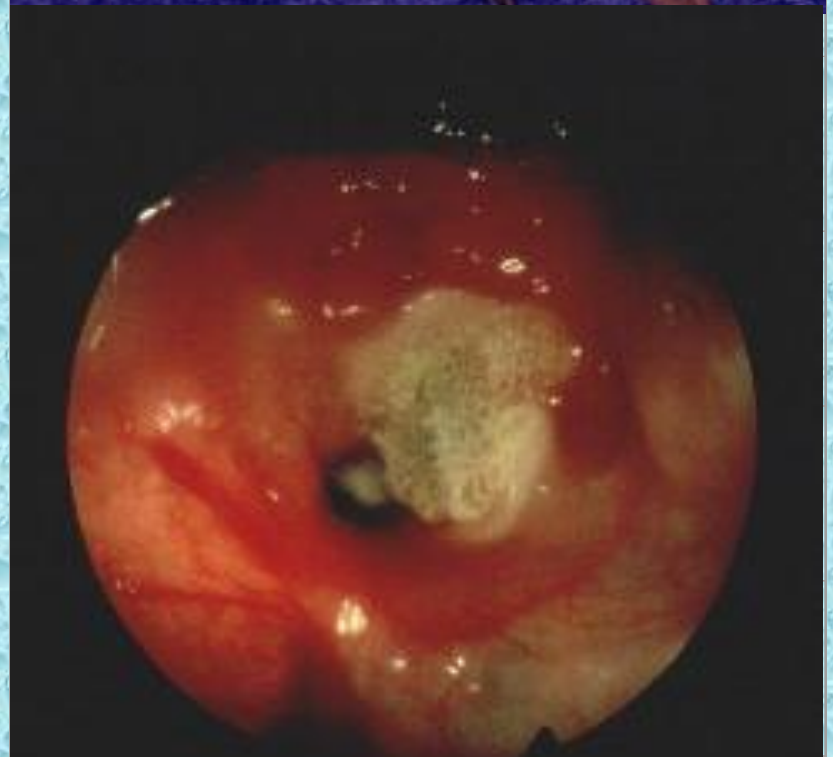
# Amyloidosis

- Background:
  - Amyloidosis = deposition of amyloid A, a fibrillar protein derived from immunoglobulins within the body
    - » Amyloid A = acute phase protein degradation product
    - » Amyloidosis denotes a pathological tissue change d/t amyloid protein deposition
  - Reported in many raptor species
    - » Considered an emerging disease in falcons in Middle East
      - Most cases large female gyrfalcons and gyr-hybrid falcons
      - Occasionally peregrines & sakers
      - Wild-caught gyrfalcons higher incidence compared to captive- bred gyrfalcons

# Amyloidosis

- Background:
  - Assoc with chronic inflammatory or infectious stimuli
    - e.g. aspergillosis in falcons or tuberculosis in waterfowl
    - Most falcon cases occur 3-6 mo after primary inflammatory process
- Reports of splenic amyloidosis in seagulls within 1 mo of entering captivity suggest maladaption stress plays a role in pathogenesis
- Amyloidosis in ducks assoc with *E. coli* endotoxin

McKinney. *Amyloidosis in falconiformes. Proc Aust Chapter: AAV: 2002.*



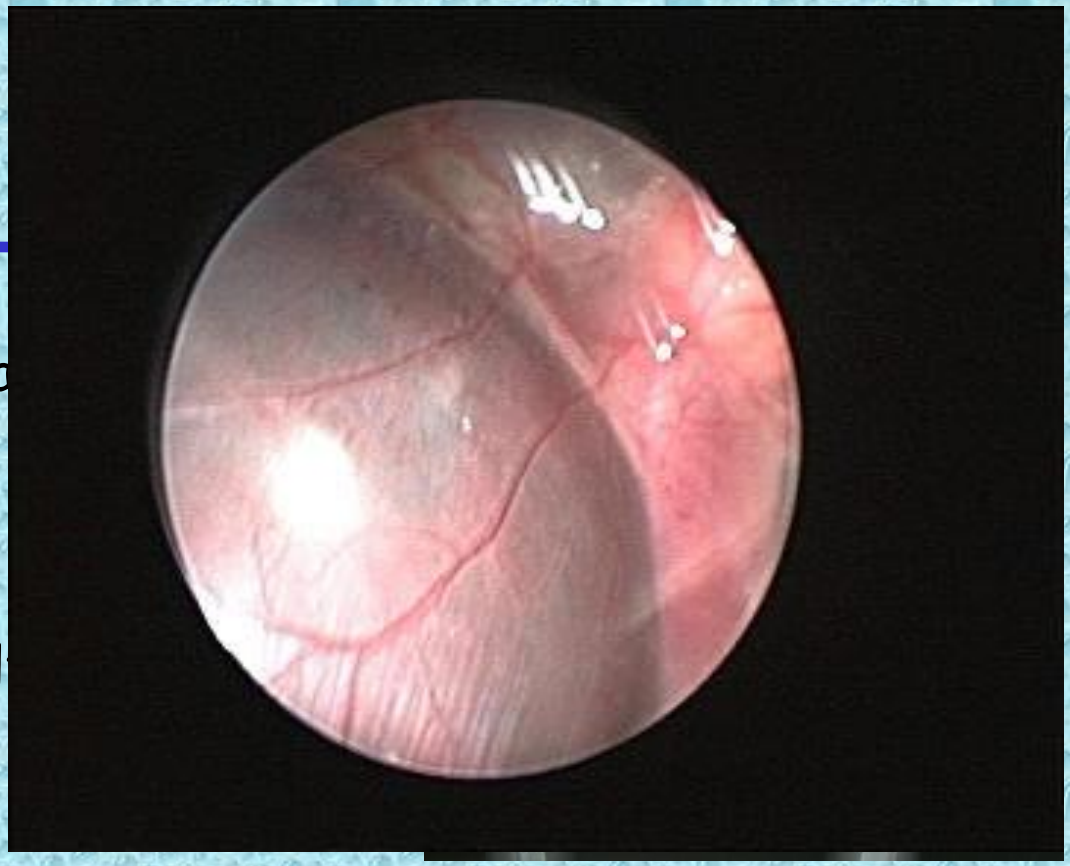
# Amyloidosis

- Background:
  - Amyloid insoluble & resistant to proteolysis
    - » Once deposited in tissues it cannot be eliminated, resulting in tissue destruction
  - Symptoms vary according to the organ (s) affected
    - » Sites of deposition include;
      - Liver
      - Kidney
      - Vascular walls of GIT (leading to protein loss & malabsorbtion)
      - Spleen

# Amyloidosis

## Clinical signs:

- Exercise intolerance
- Weight loss with norma
- Lime green urates
- In falcons mutes often terminal stages
- Abnormal feather moul
- Ascites



## Endoscopy:

- Green tinged swollen liver with waxy appearance & enlarged spleen

## Radiography:

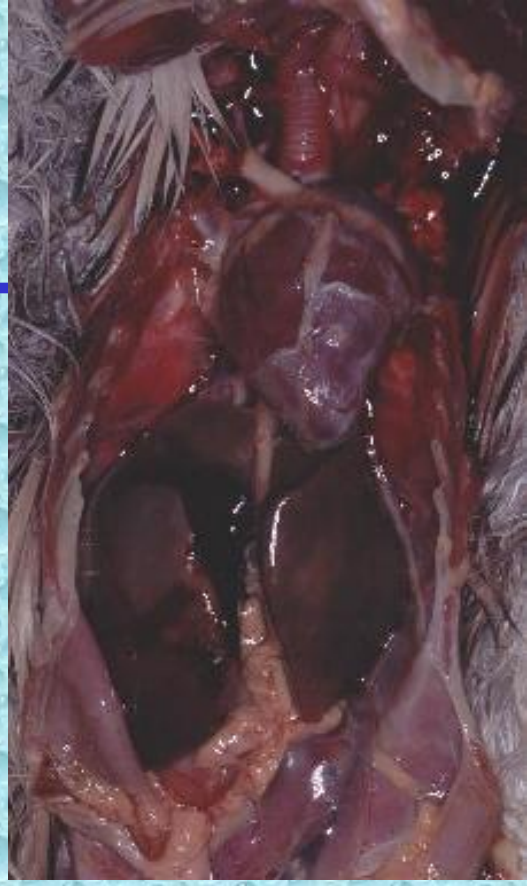
- Hepatomegaly & splenomegaly

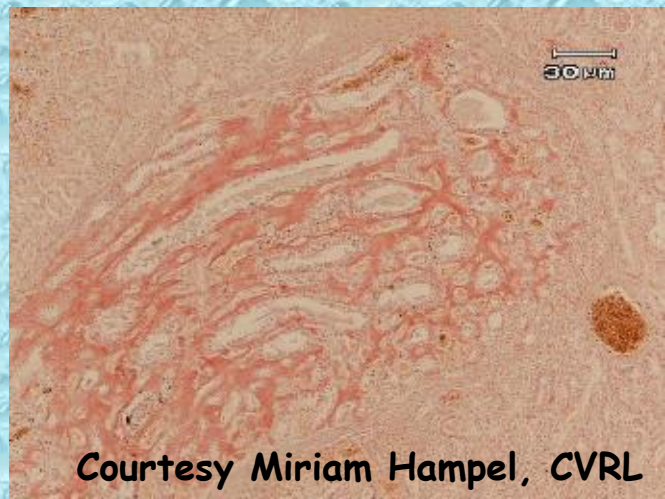
# Amyloidosis

## Diagnosis:

- Liver biopsy with special stains
- Histological demonstration of amorphous, eosinophillic amyloid
  - » On Congo red staining shows green birefringence under polarised light.
  - » Image shows marked interstitial amyloidosis btw collecting tubules
- ↑AST, GGT & bile acid
  - » 30% of amyloid cases BA & AST levels within normal ranges
  - » ↑ plasma iron and TP often seen in terminal amyloid cases when liver grossly enlarged

Hampel et al. *News about amyloidosis. Falco; 2003.*






Courtesy Miriam Hampel, CVRL

# Amyloidosis

- Treatment & prognosis:
  - Progressive amyloidosis ultimately fatal
  - Therapy to correct underlying infection
  - Colchicine (0.04mg/kg PO q12-24h)
  - Vitamin C therapy ?
  - Consider preventive colchicine in 'at risk' patients (e.g. chronic bumblefoot cases
  - DMSO used in humans but DMSO given at a dose recommended for dogs of 80mg/kg s.c 3 times /week used by McKinney but disappointing results