THE

JOURNAL OF

COMPARATIVE PATHOLOGY

AND

THERAPEUTICS.

VOL. XXI.—NO. 3. SEPTEMBER 30, 1908. PRICE 2s. 6d.

LAMZIEKTE ON THE KAAP PLATEAU.

By J. SPREULL, M.R.C.V.S., Koopmansfontein, Cape Colony.

THE investigation work of Mr Thos. Bowhill, F.R.C.V.S., late of Grahamstown, in connection with what is known as coast lamziekte, or, as he termed it, pasteurellosis, has recently been largely confirmed by Mr Wm. Robertson, M.R.C.V.S., his successor at the Veterinary Laboratory, Grahamstown. In the following remarks I wish to set forth the results of my recent work at Koopmansfontein, which so largely confirm the work of these investigators, and in addition to place on record descriptions of the disease as it is met with in this area in cattle and goats. The variations of type in the symptoms during life and the lesions at the necropsy appear to me to be sufficient excuse for this contribution to the literature on the subject, and I trust that it will be useful, if only for comparison with the disease as seen on the coast.

In my Annual Report for 1906 I tried as fully as I could then to describe how my change of headquarters from Somerset East to Grahamstown had brought me into contact with a new disease, which up till then I had barely seen. Referring to it, I wrote, "It is this disease, which in its apparently hydra-headed forms led Dr Edington to his 'correlation of diseases' theory, and to the erroneous conclusion that horse-sickness, bloodlung, veldsickness, gallsickness (by which he generally meant lamziekte), and heartwater were one and the same disease. Until my arrival in Grahamstown I had had almost no experience of this disease (pasteurellosis), and I was for a time unsuspicious of it, until at the almost inevitable *post-mortem* I found the same type of lesions and the same infection running through cases

which I had supposed to be widely different in their causation and pathology." My transfer to Koopmansfontein seems to have brought me face to face again with the same malady but in slightly altered phases; it has, moreover, given me opportunities of investigating such cases bacteriologically and by means of experimental inoculation, and so extended my acquaintance with this type of disease as to confirm me still further in the views then expressed.

Let us first consider lamziekte as affecting bovines.

Nomenclature.—The Boers have given to this disease the name "lamziekte," which is descriptive enough as far as the paralytic form is concerned. The name is sanctioned in the Cape Colony by long usage, for in some parts the disease has been known for about forty years. It does not, however, aptly fit the throat form, nor even the very acute lung and bowel forms, in which paralysis only appears when the animal is *in extremis*. According to its type, it is frequently called also bloodlung, imapunga, veldsickness, gallsickness, and also heartwater (the latter quite wrongly). Very frequently also acute cases are mistaken for some severe form of vegetable poisoning.

Lamziekte must not be confounded with stiffziekte, or stiffsickness, a disease affecting the bones and joints chiefly of the fore legs, and causing bony enlargements around the fetlocks and coronets and deformity of the hoofs. I am aware that cases of chronic lamziekte in this area are often called stiffsickness from the peculiarity of the animal's gait, but I prefer to reserve this term for the bone disease proper, and to distinguish true lamziekte, which is not a bone disease, by the fact that in it no bony enlargements ever take place. During my stay at Koopmansfontein no case of stiffsickness has come under my notice. I am also informed that cases of stiffsickness do not develop into lamziekte nor *vice versa*, whilst it is a common thing for chronic cases of lamziekte to recover more or less completely and die later from the acute form.

Distribution.—Lamziekte in one or other of its forms enjoys a very wide distribution in this Colony. It is certain that not all of the diseases called lamziekte are really due to this infection, but, on the other hand, some others masquerading under different names will be found to be identical as regards their causal organism; for instance, to my certain knowledge acute cases, both in cattle and goats, are frequently called gallsickness. It is the difficulty of its recognition with any certainty which has made it nearly impossible for anyone to get at its areas of distribution. To do so I have laid my colleagues under contribution with only moderate success. It is commonest and most severe in the coastal districts of the Eastern Province, but is probably to be met with within restricted areas in nearly every district from Cape Town to East London. Generally it is confined to sour veld areas, but lately it has also invaded karroo veld in the Cradock district. It is also present in some parts of the adjoining districts of Somerset East and Bedford. In Alexandria, Albany, Bathurst, Peddie, and Victoria East it is well known and dreaded accordingly. Mr Freer has seen it on the sour veld of Uitenhage and Humansdorp districts, and Mr Dixon in the districts adjoining East London. Mr Hutchence has met with it "in three areas on the high sour veld of the upper slopes of the Drakensberg (Griqualand East) facing S.-W.," but his description and the lack of mortality in his outbreaks make

its identity uncertain, though his cases do bear some resemblance to paralytic lamziekte. It is well known over nearly the whole of Griqualand West and many parts of Bechuanaland as far north as the Langeberg, and along the south bank of the Orange River as far west as Upington.

I think that it is certainly to be met with in the Orange River Colony districts adjoining Kimberley, and likewise in the Western Transvaal. It is not known in Rhodesia, but possibly it exists in Natal, where the so-called "gallsickness" is very prevalent.

Cause.—Since acquiring sufficient knowledge of the disease to enable me to search for it aright, I have been able to recover with unvarying success the same organism from all cases of lamziekte here, and have cultivated it on artificial media in the incubator and found it virulent on inoculation into healthy animals. This organism conforms in all respects except in its active motility with Lignières's description of the pasteurella, that is to say, it is a cocco-bacillus, appearing under the microscope in the form of a tiny bipolar, stained at each end and clear in the centre, polymorphic, sporeless, and preferably ærobic; it does not stain by Gram's method, grows well on gelatin, agar, and beef broth, and forms on naturally acid potato a growth which is invisible to the naked eye; it does not liquefy gelatin nor coagulate milk, and during its growth forms neither acid nor indol. Its cultures have a characteristically sour and very disagreeable smell. It is generally associated with other organisms, such as bacillus coli and staphylococci, and much labour is often entailed in the endeavour to isolate it in a state of purity. When successful my cultures conformed to the above description in all cases.

Exciting Causes.—Although there no longer seems to be good reason for doubting the specific nature of this disease, there are certain exciting causes which seem to play an important rôle in its production. The disease is met with sporadically all the year round, but its season of greatest prevalence is from September to the end of January, depending in this area very much upon the amount of the rainfall. Lamziekte is always prevalent in the hottest weather when the rainfall is desultory and insufficient, and cases keep coming on until good rains fall and put the veld in good condition. This year, the rains having almost entirely failed, has brought on a crop of cases in February, whilst last year the good rains in January almost made the disease to cease. All are agreed that lamziekte is most prevalent when the grass shoots up and again withers away under the weather conditions above indicated. In much the same way burnt veld is believed to be very dangerous as a producer of lamziekte, and probably this is because the young grass shooting up without the protection of last year's old tussocks and bush is more prone to wilting than where the veld is not burned, since evaporation from the surface of the soil itself must be greater under such conditions.

Many incline to blame the water supply, and it is said that cattle which drink at a large river will seldom or never contract the disease. This appears to me easy of explanation when we turn to the germ as the actual and only cause necessary for its production. A large river, even if polluted in its bed during the dry season, would by dilution when in flood wash itself clean and so prove innocuous to cattle drinking its waters, whereas a very small one would be more

dangerous; and the water of dams and boreholes, unless protected from all sources of contamination by animal excreta, would tend to become more and more a centre of infection. In my opinion this is actually what occurs, for a farm once infected seems always to remain so. There is danger also I consider in allowing cattle to drink at pans upon an infected farm. Mr Robertson's recent investigations have shown that the infective germ of lamziekte flourishes in the water of dams upon infected farms along the coast, and we have been able to confirm his results in this area.

The disease seems to be neither contagious nor infectious in the ordinary sense. The virus doubtless escapes from the economy of the sick animal in its fæces and other discharges, and remains in the soil or water, just as in anthrax or blackquarter.

On all farms upon which this infection exists the cattle and small stock evince a craving for bones. On the other hand, on farms free from lamziekte this peculiarity is not manifest to anything like the same degree. Every cattle owner knows how the favourite cow will run away with one's washing, but this is a playful pastime compared to the zeal with which cattle on infected farms furbish in the ash heaps and roam around the farmhouse or native huts in search of bones, etc. This craving almost amounts to disease—it has been noticed in other parts of the world (Argentine Republic) where forms of this malady prevail—and may with some truth be reckoned as the first symptom, or, at any rate, as a sign that something is wrong in the animal's economy. Where a liberal lick of bonemeal is supplied it will be readily seen after a time how this craving ceases, but under the worst weather conditions (or, shall I say, period of greatest infection?) it reappears in some cases even in cattle dosed by the mouth as frequently as are our cattle at Koopmansfontein. Nor is it bones alone which are picked and chewed; stones and preserved meat or milk tins serve almost as well, but as they do not break into pieces they are generally dropped after being subjected to vigorous chewing for as much as half an hour on end. Cattle will also at times pick up and swallow loose manure from the surface of the kraal.

I shall now pass on to describe the three commonest forms in which this disease has been recognised in this Colony, dwelling more particularly, however, upon the paralytic variety, which is the one most prevalent in this area.

Œdematous or Throat Form.—This form is met with in some of our coastal districts. I have not yet met with it in Griqualand West, and it must be rare, for in response to numerous inquiries I have only been informed of two such outbreaks. In February 1906 I saw this form of the disease in a cow on a farm in the Peddie district. The outbreak was at first thought to be anthrax, but microscopic examination and the *post-mortem* lesions quite negatived this. Five cattle had died before my arrival. The sick cow which I examined had extreme difficulty in breathing, owing to an œdematous swelling of the throat and head; her tongue also was swollen, and lolled from the mouth, which was full of frothy saliva, froth also escaping from the nostrils. There was also fever, rapid wasting, and diarrhœa. Out of the five which died two had no external swellings, and were probably thoracic in type, but the other three showed symptoms identical with this cow. Cattle of all ages were affected except small

calves, and four sick animals recovered. Deaths took place in from one to three days, but cases which recovered were sick for five days or more. Mr Bowhill saw this form also in this country, and recognised it as a form of "pasteurellosis," whilst Mr Dixon also says he has seen it in his area.

Thoracic Form.—Of this I have had two cases at Koopmansfontein, and I held a *post-mortem* examination upon another at Blikfontein. It nearly coincides with the type of case which I have seen in the Eastern Coastal districts. In this form the *post-mortem* lesions do not differ greatly from those seen in the paralytic types except in so far as the lungs are concerned. These are acutely congested, with early red hepatisation in their dependent areas, more or less interlobular œdema, often with infarcts and ecchymoses under the pleura, the lesions being more congestive or hæmorrhagic in the peracute cases and more inflammatory in those in which death is a little longer delayed. They resemble acute pleura-pneumonia contagiosa, but the changes are more uniform in age and the pleura is much less affected. The abomasal and bowel lesions may be quite as well marked as in the paralytic form, but are generally not so severe. This is the form described by Mr Bowhill as pasteurellosis bovis, and known along the coast as bloodlung or imapunga. Quite recently it has been described by Mr Robertson as pneumo-enteritis or pasteurellosis bovis. The symptoms manifested during life are those of congestion of the lungs, and paralysis does not appear until the animal is *in extremis*. I can form no opinion as to why the thoracic form should be the more prevalent on the coast and the paralytic form the usual one here. It may be that in this area the altitude, diminished rainfall, and severity of the frosts in winter make the germ less virulent here. It is the form most commonly reproduced by intravenous inoculation with cultures of the organism, and cases of this type occurring through natural infection are the most violent and fatal.

In touching briefly upon the above forms of pasteurellosis my object has been simply to associate them with the next one, which by so many of our farmers is considered something entirely apart—a different disease, in fact, to that found along the coast.

Paralytic Form.—As already explained, my work here has been almost wholly concerned with this form, and consequently I shall describe it more fully, though I consider it less formidable and in some respects less important than the thoracic or coastal form.

The paralytic form varies greatly in its severity. For convenience it may be divided into acute and chronic.

Symptoms.—Even in the *acute* form the symptoms may come on rather slowly, and at first may be unnoticed. Sometimes the first warning is a cessation or great diminution of the milk supply in cows. In other cases the milk is secreted even after all the symptoms are advanced and typical; the amount, however, is probably much reduced. Should the stoppage of the milk warn one and the cow be examined, her temperature, pulse, respiration, and excretions are probably quite normal, and no sign of disease is manifest until the stiffness of the gait comes on. Again, I have seen acute cases, with rapidly developing stiffness and inability to walk more than a few yards without lying down, show no other signs of discomfort, and

rumination go on for some time as if in health. The acute cases are all fatal, and the earlier the animal goes down the shorter is the attack. In those peracute cases in which the animal dies within twenty-four hours to three days the onset of the symptoms is very rapid. These are stiffness (described later), loss of appetite, cessation of rumination, depression of the body temperature, stupor, grinding of the teeth, salivation, frequent eructations and swallowing movements, and paralysis manifested by the animal going down within a few hours and refusing to rise again. If lifted it will simply hang in a sling and refuse to support its weight. Sensibility of the skin is not lost, and both hind and fore legs can be moved at will though incapable of bearing weight.

From the commencement of the attack the temperature falls gradually from normal to 97° F., or even below this; shortly before death it sometimes cannot be registered on the ordinary clinical thermometer. There frequently is considerable excitability at the outset of an attack, but later a sort of stupor ensues, in which the animal takes no notice of surrounding objects, and shows signs of headache by frequently shifting the position of the head and an inclination to tuck it round into the flank or pillow it on any convenient support. The respiration and pulse are accelerated only during the period of excitability and in those cases in which the lungs afterwards show the more extensive disease changes. · In some cases the bowels appear relaxed at first, but later the passage of fæces is in abeyance, yet no real constipation exists. In the same way the urine, which always looks normal, may not be voided for long intervals. The sinking of the temperature is manifested by coldness of the skin of the body and limbs, and frequently the coat stares. The animal dies poisoned by the toxins produced by the specific microbe. In the cases which survive the first three days the animal remains *in decubitus*, and may sit up as if in health; but rumination is suspended, appetite for food practically gone, and prehension, mastication, and deglutition performed with great difficulty. The animal maintains a sour and indifferent attitude to all one's ministrations, and death ensues sooner or later from exhaustion.

Although the acute form is generally fatal in from twenty-four hours to three days, it sometimes does not terminate in death until as late as the seventh to ninth days. These latter cases do not differ on *post-mortem* from the more acute, and there is no real difference. The animal remains in a state of more or less complete stupor during the whole time, and though it may sit up fairly well, and even resent human interference, still there appears to be little return of appetite and small power to masticate or swallow the food. It is this loss of function in the prehensile power of the tongue and muscles of mastication which so completely destroys the chances of such animals recovering. To look at them one would say that, given a little time, with nursing and good food, their strength would return and the paralysis be overcome. In none of my cases has this happened. A little forage or lucerne pushed into the mouth would frequently remain for hours in the same position, the animal making slow chewing movements and futile attempts with its tongue to draw the wisp completely into its mouth. Some of the animals would drink fairly well, although slowly, and these received milk, gruel,

bran, and slops, but only a very little of such food was partaken of. When we attempted to drench with such fluids, swallowing difficulties were soon manifested, and the *post-mortem* lesions in the lungs showed that portions of the drench had been inhaled, and that our attentions had only aggravated the poor animal's sad plight. The few cases which did manage to swallow ate so little and in such a half-hearted manner that they could get no good from the food even if it were digested, which it certainly was not. I am aware that cattle do occasionally recover and get up after lying for weeks, but the only case we have had here which did so I prefer to place amongst the chronic cases, inasmuch as prehension, mastication, deglutition, and rumination were never in any way impaired.

The *chronic* form which we have met with here has manifested itself also with different degrees of intensity. Some cases showed only slight stiffness during progression, nearly always most manifest in the forequarters. These cases would last a few days only, and leave the animal apparently little the worse. In more severe cases the gait was characteristic; whilst the knees seemed closer together, as if hobbled, the feet would be thrown forward and outward with a laboured movement faintly suggestive of the goose step. Viewed from the side the knees were very hollow in front ("calf-kneed"), and there was a sinking of the forequarters from side to side when the weight was placed on each limb. The movements of the hind legs were much less noticeable, but sometimes there was a very slight arching of the back and the feet were thrown well forward, similar to a case of founder in the horse. Others showed slight crossing or plaiting of the hind legs, with a tendency to draw the feet a little backwards before planting them on the ground during progression. A calf which took sick when nine days old showed more stiffness in the hind than in the forequarters. In the mildest cases the time limits of the attacks were hard to fix, the animals making a gradual return to health. Some were stiff for a week only, others for more than a month, and these lost condition greatly. One went down on the thirteenth day, was lifted daily with slings, and finally got up and made a gradual recovery after being down twelve days. Such severe cases, even when recovered and putting on condition, do not always regain their wonted freedom of movement, and can be picked out from the herd for a long time afterwards by the peculiarity of their gait.

Post-mortem Lesions.—If the carcase is skinned off soon after death there is seldom any subcutaneous extravasation, but an injection of the blood vessels is often noticeable.

The lungs are sometimes quite healthy, but generally there is considerable congestion of both, with a few patches of early hepatisation in the lower lobes, and there may be a few infarcts. Where the lungs are congested there is often a little straw-coloured watery effusion in the chest cavity, and frequently also a lymphy œdema present in the mediastinal spaces. The posterior mediastinal and other lymph glands of the chest are always more or less enlarged, œdematous, inflamed, or hæmorrhagic, their condition being generally in sympathy with the state of the lungs themselves. The thymus gland is sometimes dotted all over with tiny blood spots under its pleural covering. Of course from the above remarks I exclude all

cases in which lesions have been produced in the lungs through careless or inconsiderate drenching at a time when paralysis of the muscles of deglutition made such a proceeding quite indefensible.

The heart will be found dilated and its cavities full of softly clotted blood. There are generally petechiæ externally, especially along the interventricular grooves, and internally under the endocardium of both ventricles, but more especially the left. Sometimes these hæmorrhages are only external, sometimes only internal to the heart. The heart sac is usually normal, but may be inflamed and contain a little watery effusion.

There is frequently a little yellow, watery fluid in the abdominal cavity; it may be slightly blood-stained, but in my cases it has never been abundant and the peritoneum looks normal. The paunch and reticulum are healthy; in one or two cases only have I noticed congestion of the papillæ after scraping off the epithelium. Small splinters of bone are constantly found amongst the contents of the paunch. The manyplies is also normal as a rule, though in some cases I have seen the leaves injected in places. In my cases, which have always had plenty of water supplied to them during illness and generally a purgative drench given at the outset, there has been no impaction of this stomach, and I can hardly believe that such a lesion is common in this disease, but incline to think that where present it is due to the cattle having gone down far from water and died without being supplied with it. Many farmers also do not clearly understand the difference between the normal dry state of this stomach's contents and impaction proper. The abomasum or fourth stomach, on the other hand, is almost invariably inflamed, sometimes very severely, its mucous membrane a little swollen and showing erosions or early ulceration, chiefly along the free margin of its folds. In my cases the pylorus has been usually less inflamed than the body (fundus) of the stomach.

The small bowels are more or less inflamed throughout, their mucous membrane being swollen and much catarrhal mucus in their canal. Zebra stripings are frequent, and Peyer's patches may be but are not always prominent. In the most severe cases hæmorrhages take place into the bowel wall, and the ingesta are blood-stained, but less extensively than in anthrax. There is often a highly congested and varicose state of the mesenteric blood vessels, with bleeding into and staining of the fat. It will thus be seen that the lesions of paralytic lamziekte are mainly those of an acute gastro-enteritis. The cæcum, colon, and rectum are more or less inflamed, but in my cases always much less severely than the small bowel and fourth stomach. The lymph glands of the abdominal cavity, and especially those situated in the mesenteries, are almost invariably hæmorrhagic, inflamed, œdematous, and swollen, their condition corresponding with that portion of the bowel which they serve.

The spleen is more often normal than not, but may be a little swollen and the pulp slightly softened.

The liver is always congested; the gall-bladder is normal or may have the blood vessels of its mucous membrane highly injected, but the bile is clear and normal, even though it fill the bag completely, as indeed it generally does.

The kidneys are congested and very little altered to the eye. They

may have hæmorrhages into their cortex, and the apices are sometimes very yellow. The urine looks normal and the bladder also, but sometimes its mucous membrane is congested or even inflamed in cattle which have been long paralysed.

The muscular system looks unaltered to the naked eye.

The fluid in the spinal cavity is frequently increased in amount, but always clear and watery. The pia-mater of the brain and medulla is congested, often acutely, but on the cord this covering is normal, even in the lumbar region. The brain and cord look otherwise normal.

The mucous membrane lining the whole nasal chambers is frequently acutely congested or inflamed and of a deep blue or purple colour.

The bones, joints, and marrow look quite healthy, but the cancellated bodies of the vertebræ are often blood-stained.

Susceptibility.—A strange feature about this disease is the severity which it manifests in the female sex. Pregnant cows when near to calving are the greatest sufferers, and the disease in them generally takes on the acute form. Next to these in susceptibility come cows which are giving milk. The disease is less frequent in young growing cattle, and still less in calves and oxen. Still, the latter are susceptible, even when drawing transport regularly. A local transport rider told me that during six months in 1906, when his one span of oxen were working almost daily, he lost three oxen from lamziekte, whilst a fourth sickened and recovered. Bulls are by many considered naturally insusceptible, but this is not the case. On the coast the thoracic form is very common in young cattle and calves. So far as we know, no breed is exempt or even enjoys comparative immunity from attack.

Plurality of Attacks.—The power of resistance to this disease seems to be greatly impaired by one attack, and perhaps there is good ground for saying that animals once affected do not really recover. Doubtless our experience is rather too limited to allow of our coming to such a hard and fast conclusion, but in this experiment so far such a verdict might be given as regards the control (non-bone-fed) animals at least. An exception might be made in the case of the small calf, and possibly that of very young animals generally. In this respect I may mention that I had three two-years-old heifers and a young cow sent to me by the Messrs Radloff which up till now have remained healthy. Possibly the change of veld, even of infected veld, may have acted beneficially, and all were, however, receiving bonemeal here. Let us examine how our apparently recovered animals have fared.

Ox (property of G. Emslie).—Sick for a few days about 1st December 1906, but did not go down. Sick again on 8th to 14th January 1907, and died. Ox contracted both attacks when pulling in transport waggon and had been very much in the yoke previous to his attacks.

Ox 6.—Sick here 15th September 1907, down on thirteenth day, rose after twelve days and recovered. Again sick 31st January 1908, went down 12th March, and is still down at date of writing (17th March).

Heifer 58.—Sick for five weeks from 28th October 1907, calved

12th November at the height of her illness, and did not go down. She picked up nicely again in condition, but her gait was still quite noticeably amiss when on 7th March she again fell sick, and died on the 11th from an acute attack.

Cow 91.—Sick 1st to 8th November 1907, only mildly stiff. Sick 3rd January 1908, and died within twenty-four hours. She had appeared quite recovered in the interval.

Cow 30.—Once sick at Secretaris (Radloff's farm). Sick here 2nd December 1907, and died within twenty-four hours.

Ox 76.—Three times sick at Secretaris (Radloff's farm). Sick here 21st to 25th April 1907. Again sick 17th to 23rd May 1907, when he died from a fifth attack.

Cow 33.—Sick 24th December 1907, and with slight remissions up to date. Had lost greatly in condition, but recently began to improve in her gait and put on flesh again.

Ox 95.—Sick for three weeks from 24th January 1908, but only very mildly; appeared recovered, but sickened again on 25th February, and died within thirty-six hours.

None of the above animals received any bonemeal whilst in my hands. The other mild cases which appear to have recovered are too recent to be worthy of inclusion here.

Treatment.—I feel very reluctant to waste time by describing treatments none of which, to my mind, appeared to have availed anything. Acute cases die in spite of any drugs given, and chronic cases undoubtedly recover without them, nay, even in spite of them perhaps. The following is a list of the drugs which have been tried singly or in combination with others: Epsom salts, Glauber salts, podophyllin, gamboge, calomel, salicylic acid, colchicum, cyllin, sodii hyposulph., arsenic, arrhenal, nux vomica, and also acetic acid (the latter as a substitute for the Boer remedy, vinegar). Besides these, artificial serum, normal serum, and weak iodine solutions have been given hypodermically. Perhaps the best and most reasonable thing to give is a purgative, for it has a marked action in clearing out germs from the inside of the intestinal canal. The best advice I can offer is: Don't kill your animal by treatment, never give a drench to an animal incapable of swallowing it, bed your paralysed cow down comfortably, prop her up with sacks filled with earth, lift her frequently, and make her change sides two or three times daily. Give her lucerne or green barley if she will eat it and plenty of water to drink. Pin your faith on methods of prevention, and you will save time, worry, and cattle enough to recoup yourself amply for all the expense entailed.

Seeing that the flesh is not harmful I think that the "baas" (master) might very judiciously at times cut the throat of those cases which from the outset look certain to be fatal, and so make the best salvage upon a fat cow which otherwise is but a free gift to the natives.

I would strongly recommend also that all recovered (?) cases be fattened off and sold, for there is no doubt that such animals are very prone to further attacks. Possibly, also, through course of time this weeding out of the most susceptible cattle would cause the herd to become gradually more resistant.

Prevention.—Convinced as I am that the complaint known here

as lamziekte is not a bone disease, it seems strange that we should turn to the administration of sterilised bonemeal in the form of a lick as our foremost and best tried means of prevention. As already mentioned, cattle well supplied with this lick scarcely manifest any depravity of appetite; but, more remarkable still, they do not contract lamziekte in anything like the same proportion of cases, and probably when they are attacked the disease is less severe (but here my experience is too brief to warrant any dogmatic assertion on the last point). Empirical practice by supplying an apparent want has found a preventive remedy, but it is left for science to solve the enigma of just how it acts. Bonemeal is evidently a good tonic, its phosphates are a necessity to the food ration, and, should these be proven deficient in the grasses and bush of the varied areas in which this disease abounds, the benefits accruing from its administration will be further explained in the results, which are that the stock are thus kept in a state of exuberant health. Further explanation than this I am at present unable to give. The method has its drawbacks, the chief of which is the expense entailed; and, looking at it theoretically, I fear there is a wastage, and hope that some day the one thing needful which seems to be contained in the bonemeal will be separated from the vast bulk of other matter, and made capable of administration in a cheaper and even more convenient form. Meanwhile there is always the hope that bacteriology may come to the rescue, as it has done in so many other specific diseases of late.

Bonemeal fails in another great respect—it does not remove the cause. To this end there are other methods which suggest themselves for prevention, and which if tried thoroughly may in time be even more successful. I refer to the possibility of avoiding all contamination of dams, wells, and boreholes, by fencing them in and running the water into troughs for the supply of the farm stock. These troughs would of course have to be thoroughly cleansed at intervals.

I believe that dipping of the cattle has so far given no indication of efficacy in checking the ravages of this disease in the coastal districts.

Changing of the grazing area, the old time-honoured method of prevention in former days, becomes less and less possible as the country becomes more settled; it is also one of the most prolific means of spreading germ infections, and has done incalculable harm in the past. Most farmers are agreed that during the late war the movements of stock, and transport oxen more especially, were responsible for the infection of many hitherto clean farms in this area. One farmer, writing to me, says that his farm was clean until he lent grazing to a neighbour's badly infected herd, with the result that the infected herd benefited at once by the change of veld and left off dying, whilst his own cattle began to die off at the rate of four and five a day.

Although the flesh may be eaten with impunity, I strongly recommend destruction of the viscera and all ingesta and excreta, preferably by fire.

Investigation Work.—Although I knew that a good deal of experimental work had been done already with this disease, I thought it best upon my arrival here to proceed with this part of the investigation, just as if I were working with a new complaint about which

nothing was known. As nearly all of my cases of lamziekte assumed the paralytic form, this procedure seemed all the more justifiable. Experiments were undertaken, therefore, on many different lines, such as (1) drenching, and (2) inoculation, both being performed with (*a*) materials obtained from dead animals and (*b*) artificial cultures, the last being by far the most important and successful work accomplished.

Drenching.—(*a*) With ingesta (pens mest). Several goats were drenched with the fluid contents of the paunch, fourth stomach, small bowel, or cæcum, or even with scrapings of the mucous linings of these organs, but all remained apparently unaffected.

(*b*) With blood. Three goats were drenched, each with from 12 to 16 ozs. of fresh blood from three different sick cattle, with no result so far as could be seen.

(*c*) With marrow. One goat received 6 ozs. from the humerus of a sick ox. This goat developed a fever reaction from the fourth to the eleventh days, which had probably nothing to do with the drenching.

(*d*) With artificial cultures. One goat received by the mouth 70 cc. of an eight-days-old nearly pure broth culture of the pasteurella, and on the following day 55 cc. of a similar culture eleven days old, without developing any consequent sickness.

Inoculation with Body Fluids.—(*a*) Blood taken from sick cattle and injected in huge quantities into both cattle and goats either under the skin or into the veins proved harmless. Two control cattle (*i.e.*, those receiving no bone-meal) were used as the subjects of these experiments. Long afterwards both these cattle contracted lamziekte from the natural infection. A guinea-pig also resisted blood inoculation.

(*b*) Lymph-gland emulsion or expressed fluid proved innocuous; it was used upon six goats from four different sick animals. In my hands also it has so far failed to kill guinea-pigs by intraperitoneal injection.

(*c*) Spinal fluid was injected into the vein of a cow, a goat, and a sheep without result.

(*d*) Peritoneal effusion. A dose of 10 cc., fresh and warm, injected into the vein of a goat proved negative.

Inoculation with Artificial Cultures.—As already stated, the motile bipolar organism has been recovered from the tissues of all our later cases. Very frequently it cannot be picked up from the lymphatic glands of the chest or abdomen, but so far I have never failed to cultivate it from the submucosa of the abomasum and small or large bowel. From such sites the resultant cultures are generally very much mixed. The following cases have yielded cultures of this organism—Emslie's Ox, Nos. 56, 76, 9, 62, 62's calf, 91, and 105. Our initial failures can easily be accounted for by errors of technique and want of knowledge of the habitat of the organism within the animal system. Having once recovered the organism and brought it to purity, it is an easy matter to keep hold of the strain and pass it through the different experimental animals. In our hands it has proved virulent for an ox, goats, sheep, guinea-pigs, and a rabbit. It has generally been used intravenously, but in increased doses will kill also by injection under the skin. Old cultures contain a very

virulent toxin, which upon injection produces the same train of symptoms as the live organism. These experiments are much too numerous to detail, but two sample cases will be given by way of elucidation. It will be seen that the paralytic type of the disease has not been reproduced by inoculation, but that the *post-mortem* lesions in these cases resemble very closely the thoracic and gastro-enteric forms as seen more particularly in the coastal districts.

Ox No. 76 died from his fifth attack of lamziekte on 23rd May 1907, after an illness lasting seven days. A nearly pure culture of the motile bipolar organism was obtained direct from one of its mesenteric glands, and 4 cc. of this broth was injected into goat No. 18 at 6 P.M. Next morning the goat was dead; it had lived only eleven hours. The autopsy was held at once, and revealed watery effusions in the chest, heart sac, and abdomen; congestion, emphysema, and marked interlobular œdema in both lungs; lymphy jelly in the mediastinal spaces and around the heart sac; acute inflammation of the fourth stomach, with small incipient ulcers at the pylorus; small bowels congested or patchily inflamed, large bowels even more so. All the lymphatic glands enlarged, inflamed, and œdematous. Liver and kidneys congested; spleen small. Pia-mater of brain congested; mucous lining of the nasal chambers of a deep purple-red colour.

Four months afterwards, when this strain of virus had been passed successively through three guinea-pigs, a rabbit, a goat, and another guinea-pig, it was injected in the dose of 100 cc. into the vein of ox No. 80, and produced his death in three and a half days. The injection was made at 6.15 P.M., and although the culture was only seven days old the very depressing and powerful nature of its toxin was soon evident. At 11 P.M. his temperature had fallen from 103·2° at the time of injection to 95·8°. He showed marked rigors, and stood with his back arched and a dull expression; his movements were all over stiff, the surface of the body cold, and there was frequent passage of very fluid fæces. Next day he was very dull and stiff, refused all food, purging continued till noon, and thereafter slimy mucus streaked with blood was passed; he also salivated a little, and passed blood with his urine. The body temperature ranged between 98·4° to 101·2°. Next day he looked much worse and showed respiratory trouble; ate nothing, passed no fæces; temperature 96·4°, A.M., 101°, P.M.; very much depressed all day. On the third day the symptoms of pneumonia, depression, and debility became more and more pronounced; he lay a great deal, but could rise when wanted. He died at 8 A.M. on the fourth day, after struggling for some time on one side. The necropsy revealed the following lesions: There was much extravasation and injection of blood vessels under the skin. A little effusion in heart sac; heart externally showed a few roughened patches of early pericarditis; fat stained red, and a few ecchymoses; left ventricular endocardium was one mass of deep blue-black ecchymoses, right showed a few at its apex only; blood fluid in all its cavities. Chest contained at least a pint of straw-coloured effusion on each side; lungs showed much interlobular œdema and emphysema also in places. The lower lobes especially were pneumonic, not all of same red colour and consistence. There was much bloody extravasation under

the pleura, especially in upper parts of lung; tracheal mucous membrane red and inflamed. Lymphatic glands of chest swollen, inflamed, and œdematous. Spleen, liver, and kidneys little altered. Only a little effusion in abdomen. First three stomachs normal; fourth inflamed, its mucous membrane swollen, red or bluish-red in colour, and with one incipient ulcer. Small bowel acutely inflamed, its mucous membrane a little swollen, and all blood vessels injected; large bowel much less so, and worst at cæcum. Mesenteries injected and blood-stained; all lymphatic glands swollen, œdematous, inflamed, and pinkish in colour. Spinal fluid much increased, spouted briskly when tapped; cord normal throughout, but inner covering of brain highly injected and ventricles full of fluid. The lining membrane of the nasal chambers was bluish-red and congested. Mouth and throat normal; bones, marrow, and joints normal.

Many other strains of virus have been tested by means of experimental inoculations into goats and sheep, death ensuing more or less rapidly, according to the age and purity of the cultures used. The *post-mortem* appearances are almost identical with the two above described, and vary a little only according as the germ exerts its virulence mostly upon the thoracic or upon the abdominal viscera. The micro-organism is readily picked up again from their tissues, and maintains its virulence for fresh animals.

THE ETIOLOGY AND PROPHYLAXIS OF EQUINE OSTEOPOROSIS.

By HAROLD B. ELLIOT, M.R.C.V.S., Hilo, Hawaii.

IN a previous article contributed to this *Journal*[1] I discussed this disease from a strictly clinical standpoint. Although nearly ten years have elapsed since then, and the disease has been described by several careful and scientific observers in various and far apart countries, and despite much excellent laboratory research, the solution of the etiological problem involved is so little advanced that this method of treatment is still the only possible one. In this article the theory of infective causation was, for the first time, argued *in extenso* and logically presented, and because it has been frequently quoted in current veterinary literature it seems desirable, in the light of a more mature experience, to correct and amplify some of the statements and opinions expressed therein.

Osteoporosis is of almost world-wide distribution, is the source of a large annual mortality, and inflicts severe economic losses in those areas in which it is prevalent. The consultant is exposed to an apparently insuperable difficulty, and is compelled to the most distasteful task of admitting his inability to advise successful measures either of prophylaxis or therapeutic treatment. It is, therefore, very necessary that every hint, every fragment, however small, of knowledge should be published and made common property.

"The result of my clinical observations has convinced me that the theory of the dietetic origin of osteoporosis is erroneous, and founded

[1] Vol. XII., Part 4, p. 300.