

what the troughs are for, finish their ration in a few seconds, and can be driven on by the native herd-boy. The process goes very quickly and, properly managed, it can be conducted very cheaply. Daily feeding of bonemeal is recommended as most effective. The "lick method," i.e. mixing the bonemeal with salt and offering *ad libitum*, involves less trouble, but is more expensive in bonemeal, and is not recommended owing to the fact that the craving for salt and bonemeal do not run parallel. If the mixture is adjusted according to the estimated salt requirements, some animals may get too little bonemeal and others too much. If this method is used, however, the mixture should contain excess of bonemeal; two or more parts of bonemeal to one part of salt, varying according to the degree of phosphorus deficiency prevalent over the farm. Other methods of administration of bonemeal are now being worked out.

SECTION 1.—DEFINITION AND HISTORICAL NOTES.

Definition.

Lamsiekte, also known as gal-lamsiekte, and spelt lam-sikte in the earliest records, is a fatal disease of cattle, rarely of other animals, caused by the ingestion of carcass debris containing a toxin produced by an anaerobic toxicogenic saprophyte and characterized by symptoms of paralysis and paresis, principally of the locomotor system, but in many cases also of the muscles of mastication and deglutition.

There are no gross pathognomonic anatomical changes, and unless clinical observations are made during life, diagnosis is difficult. Pathological lesions of fatty degeneration and Zenker's degeneration are common in the muscles.

Lamsiekte may now be described as *Parabotulismus bovis*.

Occurrence.

Lamsiekte has been recorded as causing serious losses in the South Cape Province, being most severe in the coastal districts south of East London; on the Cape plateau, where it is popularly believed to have originated; in the western Transvaal, in the Bloemhof, Wolmaransstad, and Lichtenburg Districts; in the Orange Free State, in the Boshof and Hoopstad Districts; over the greater part of Griqualand West; and in many parts of Bechuanaland. Occasional outbreaks, however, have been reported or suspected at one time or another from nearly every area of the Union, and since the disease is liable to occur wherever *Phosphorus deficiency* of the soil and the presence of *Parabotulinus bovis* coincide, no region can be regarded as safe. Certain uninvestigated conditions of climate and soil, however, seem to affect the survival of an introduced infection, and the occurrence of the disease is not so universal as is the phosphorus deficiency of the pastures of South Africa.

Historical Notes.

Lamsiekte is not a new disease in South Africa, but is noted as far back as historical records of the sub-continent go. Le Vaillant, in his "Travels into the Interior Part of Africa," in the years 1780-1785, referring to Cape Colony, writes on page 78 (English translation, second edition, London, 1796):—

"It is then also that the season most dangerous for these animals commences; and when they are subject to four destructive maladies which occasion dreadful devastation among the herds. The first, called

at the Cape *lam-sikte*, is a real palsy which comes on all of a sudden; and though fat, and to appearance in perfect health, these animals are obliged to remain in a lying posture, and they generally die in fifteen days. As soon as the distemper appears, those which are still free from the infection are sent out of the country; and as there is no remedy for this plague, the planters destroy those attacked by it; and this they do with the more readiness, as they have no aversion to eat the diseased flesh; above all, they make no difficulty in giving it to their slaves and Hottentots, who in their taste are still less delicate.”

Referring to what we now know as “osteophagia,” a definite and practically essential link in the complex chain of causation of lam-sikte, and now known to be due to phosphorus deficiency of the pasture, Le Vaillant (p. 290) writes as follows:—

“This quality of the herbs may be easily distinguished, and I employed a particular method to discover it. After I had arrived in any canton, I judged of the harshness of the grass upon which they had been fed, by their dispersing themselves all over my camp and eagerly searching for the bones left by my dogs, in order to ease their teeth. As these bones were of a calcareous nature, by gnawing them they blunted the force of that irritation and acidity which tormented them. We, therefore, never threw our bones into the fire. When these were wanting, dry wood or stones supplied their place; and when they could not find these resources they even gnawed each other’s horns. When the pastures were good, this ceremony never took place.”

The famous naturalist, Dr. Lichtenstein, in his “Travels in Southern Africa in the years 1803, 1804, 1805, and 1806,” referring to Rietvalley, near St. Helena Bay, writes (p. 49, English translation by Anne Plumtree, London, 1812):—

“ . . . the valley has the advantage of being a spot where a disease among cattle called the *lam-ziekte* (a sort of murrain of the most pernicious kind, since the animal infected with it becomes entirely lame) is wholly unknown. At the time when this disease prevails in any part, cattle are sent hither even from a distance to remain until the danger of infection is over, and this brings sometimes a good deal of money to the purses of the inhabitants.”

Referring to Goudinie (p. 148), in recording his “Journey from Roodezond to Swellendam and along the Southern Coast to Algoa Bay,” Lichtenstein writes:—

“We here, for the first time, saw a cow afflicted with the *lam-ziekte*. She had for a whole month lain entirely on the ground, excepting when sometimes by the assistance of the men she was raised for a short time, but she could neither rise or stand without assistance. It appears to be a disease of the nerves, particularly of those of the buttocks and hinder legs; but neither the cause of, or remedies for it, are yet well understood.

“When as in the present case the disease continues a long time, and the animal is always able to eat, great hopes may be entertained of an entire recovery.”

The collection of documents “Belangrijke Historische Dokumenten over Zuid-Afrika” contains a diary of a commission appointed by the Government of the time of Commissioner Van der Mist to visit certain farmers and induce them to improve their flocks by using woolled sheep rams or, as they are called in the report, “Spanish Sheep.” The commission consisted of three members, who started

their travels on the 25th September, 1805, from Koeberg, in the present district of Wellington, and travelled along the Vogel Valley (Paarl) to the Tulbagh District, along the Witzenberg Mountains to the present District of Worcester, then along the Hex River and Laken Valley to the Ceres District; thence through Karroport, over the Doorn, Dwyka, and Ongeluk Rivers, into the present District of Sutherland; into the Calvinia District over Hantam's Berg to the Bokkenveld and Kobie Mountains, across the Doorn into the present District of Clanwilliam, along Oliphants and Twenty-four Rivers into Piquetberg; thence to the present District of Malmesbury and back to starting point. On this journey the presence of lamziekte was noted amongst the cattle of fifteen different owners, ten of the farms being situated in the territory of the Twenty-four Rivers.

In the "Settler's Guide to the Cape of Good Hope and Natal," published by Stamford, London, 1858, a paper by a certain Mr. Reitz on the various cattle diseases contains the statement:—

"Lam-sickness; no one knows the cause or seat of, and yet it is one of the oldest and most destructive cattle diseases we have."

A quarter of a century later the disease came under the notice of Hutcheon, Colonial Veterinary Surgeon in the Cape, and his professional observations are recorded in his official reports. In his report for 1884 he records it at Barkly West, on the elevated Kaap plateau, at Griquatown, and elsewhere. At Douglas he was informed by a Mr. Wright of an old Griqua, whose age was estimated at nearly one hundred years, and who had said that lamsiekte prevailed as an epizootic over the whole territory of Griqualand West three times during his lifetime, each time during an exceptional drought.

In later reports Hutcheon records the disease in various other parts of the Cape Colony, chiefly on "dry porous soils such as the calcareous and siliceous which have little power of retaining moisture"; and as "most prevalent in dry seasons." He emphasizes the association of the disease with "intense craving for bones and all kinds of animal matter which the stock on such pastures manifest." He regards both lamsiekte and styfsiekte, a disease which is now known to be etiologically and clinically distinct, as due to deficiency of phosphates in the food, and correctly maintains that both can be prevented by feeding bran, bonemeal, or other food rich in phosphorus.

In 1895, experiments were actually carried out to prove this, under the supervision of Mr. Borthwick, on a farm Witte Clay Rug, renowned for lamsiekte. Two lots of cattle were grazed under parallel conditions, one lot receiving a liberal allowance of bonemeal and the other not. Of the twenty-three controls, ten contracted lamsiekte, while of thirty bone-fed cattle none developed the disease. The experiment was considered a complete success and regarded as proving that lamsiekte was due to deficiency of phosphates. This conclusion received much support from the contemporary soil analyses of Juritz, Cape Government Analyst, which revealed a wide-spread deficiency of phosphorus in the soils of the Colony, and Juritz himself favoured Hutcheon's views. At no time was the toxicogenic saprophyte, now known to be the direct cause of the disease, ever suspected, although Hutcheon must have been very near suspecting something of the sort when he wrote in 1894, "There can be very little doubt, however, that in the case of the acute form of lamsiekte there must be some other immediate and exciting agent which causes the sudden

development of this form of the complaint, and which causes the nervous prostration and effusion into the cranial cavity of the medulla oblongata which so quickly follows." The last sentence, it may be added, does not correctly represent the truth. Effusion into the cranial cavity is by no means characteristic of the disease. There are indeed no characteristic lesions at all.

The last notes of Hutcheon are contained in the *Agricultural Journal of the Cape of Good Hope*, 1903, and since they are in reply to a controversy, may be considered to represent his latest views. They are of especial interest as crystallizing the authoritative veterinary standpoint in 1903, and may therefore be quoted:—

"Lamziekte is a form of paralysis due to an effusion of a clear serous fluid into the membranes covering the brain and spinal cord, associated with a highly congested condition of the bones of the vertebrae, and of the articular extremities or joint ends of the long bones of the limbs, principally of the larger ones, with softening of their cancellated tissue."

"In lamziekte it will be observed that some cases are very acute and rapid in their course, the patient becoming comatose within from ten to twenty hours, and death in some instances occurring within twenty-four hours. In other cases the patients may last for days, and even weeks, during which they may eat, drink, and ruminant and die merely from exposure or exhaustion."

"There is no essential difference, however, between the acute and the more protracted cases."

"In the acute cases the effusion of the fluid takes place into the membranes surrounding the brain and the upper portion of the spinal cord—the medulla oblongata—which is rapidly followed by complete paralysis, coma, and death. The quantity of the fluid surrounding the medulla is sometimes so great that when the membrane is pierced the serous fluid rises up like a spring. In the protracted cases the fluid is found principally in the membranes surrounding the spinal cord in the dorsal and lumbar regions, producing paralysis of the hind extremities only, and rarely producing any disturbance of the brain."

"With respect to the cause of this serous effusion into the membranes surrounding the brain and spinal cord, I think there can be little doubt that it is intimately connected with the softened and highly congested condition of the bones of the vertebrae, which is invariably present."

"The long bones of the limbs are affected in the same manner as in styfsiekte, the only difference being that in styfsiekte it is the lower bones of the limb which are most severely affected, whereas in lamziekte it is the higher and larger bones, such as the humerus and radius of the fore leg and the femur and tibia of the hind leg, which are most severely affected."

So much for the nature of the disease.

Dr. Hutcheon continues:—

"From what I have written it will be evident that I regard lamziekte and styfsiekte as simply different phases of the same disease, arising from the same primary cause, a deficiency of phosphates in the vegetation of the particular farm or district. It may be difficult to explain satisfactorily why in certain animals and in certain localities the lower bones of the fore limbs should be the principal bones of the skeleton affected, while, in other instances, the bones

of the vertebrae and the upper and larger bones of the limbs are most seriously involved. It is very probable that, although the vegetation of the different districts where this disease prevails agrees in this one particular—that it is more or less deficient in phosphates—the vegetation of each district may differ considerably in other respects, the one from the other. In our experience we have found that a beast which is growing and improving in condition appears to be much more liable to contract lamziekte than one which is simply maintaining the same uniform condition. The latter are more subject to styfziekte.”

“Further, the most acute and rapidly fatal cases of lamziekte are generally met with in young animals in good condition. From these and other observations I arrive at the opinion that when food is deficient in one essential constituent, the balance of the system would be the more readily upset the more abundant the other constituents are. Acute lamziekte would therefore be most prevalent where the vegetation was luxuriant, and chronic lamziekte and styfziekte most prevalent when the vegetation was dry and not so abundant. This may account for the fact that the disease is more prevalent on certain portions of the same farm than on others.”

“It is quite possible also that there are certain plants which, when eaten largely by cattle, may have a tendency to act as an exciting cause in hastening the development of the disease, just as certain poisonous plants, when eaten by a perfectly healthy animal, have a tendency to cause an effusion of serous fluid into the membranes of the brain and spinal cord, producing rapid coma and sudden death. But the main cause of the prevalence of this disease is undoubtedly due to a deficiency of phosphates in the food, and it disappears when that deficiency is supplied.”

These views of Hutcheon, however, failed to find general acceptance, and in analysing the published articles of the next decade the stumbling blocks may be grouped as follows:—

- (1) How can deficiency of phosphates explain the fact that the incidence of the disease is independent of the food supply in the sense that some animals may be *recovering* from the disease at the very time and in the very paddock that other members of the same herd are *sickening* of it? If something is missing, how can an animal recover without that something being supplied? Countless observations by numerous observers, including competent veterinarians, established beyond doubt the fact that animals recovered from the disease under precisely the same conditions as those under which they contracted it.
- (2) How can the fact be explained that of two similar farms separated only by wire fencing lamziekte might be rife on one and unknown on the other? This fact had been established so definitely that the trend of opinion swung towards the idea of an infectious origin amongst one group of observers and a poisonous plant amongst another; both of which might, of course, exist upon one farm and not on the adjoining one, although conditions of soil and climate were the same.
- (3) Numerous farmers reported that they had given a bonemeal lick regularly to their cattle without preventing lamziekte. Others reported that bonemeal reduced the incidence of the disease. The opponents of the deficiency theory

retorted that arsenic administration reduced the incidence of worm infection, but that arsenic deficiency was certainly not the cause of worms; that epsom-salt relieved constipation, but that a costive interior argued no deficiency of magnesium.

- (4) The lesions described by earlier observers did not always correspond with later records, in which the most characteristic feature was the complete absence of pathognomonic anatomical changes. Unless an animal was examined clinically during life, no reliance could be placed upon a diagnosis of lamsiekte. Doubt were cast upon the identity of various conditions described as lamsiekte, and particular difficulties were encountered in accepting the view that *styfsiekte*, for the commonest form of which Hutcheon's explanation of origin is probably correct, and *lamsiekte*, for which his explanation is now known to be wrong, were merely varying forms of the same disease.

Various alternative theories were therefore put forward. Spreull and Robertson (*Journal of Comparative Pathology and Therapeutics*, Vol. 21, No. 3, 1908, and *Agricultural Journal*, Cape of Good Hope, Vol. 31, 1907) described a pasteurilla as producing lamsiekte, or a disease commonly confounded with lamsiekte, and their work received such acceptance that this organism is referred to in Lehmann and Neumann's "Bakteriologische Diagnostik" as the actual cause of the disease.

Keeling Roberts, Veterinary Bacteriologist in the Orange Free State, followed up this work, and in the Sixth Annual Report of the Agricultural Department, 1909-1910, records attempts to isolate their bacterium from the intestines of sick cattle. His cultures in most cases were not pure, but in eight injection experiments with them he claimed a definite diagnosis of death from lamsiekte in one case and a doubtful diagnosis in another. He also dosed cattle with small portions of alimentary tract and contents; and with liver, kidney, spinal cord, and brain. In some cases apparently positive results were obtained. Roberts himself, however, found it difficult to reconcile the production of what he considered lamsiekte, by intravenous injections of cultures which failed to produce the disease when administered per os, and the production of the disease by oral administration of viscera from which he failed to isolate the suspected organism.

According to the pasteurilla theory the disease would be an infection caused by the growth of bacteria in the intestinal canal, but since the deductions of its supporters were not in accordance with the current epizootic knowledge of lamsiekte, other workers disputed the identity of the disease produced by bacterial cultures, and explained the results obtained by oral administration of carcass remains as due to a non-specific toxæmia. In reviewing the protocols of Roberts in the light of present-day knowledge, there can be no doubt that some of his positive cases were genuine lamsiekte, and due to a specific intoxication not suspected by him.

In 1909, Walker commenced observations on the transmission of lamsiekte by drenching or injecting ingesta and emulsified organs of sick animals into healthy animals. (Second Report of the Director of Veterinary Research, 1912.) Information from farmers revealed the opinion that the disease was contracted from carcasses, skins, and contents of the alimentary tract of animals which had died of lamsiekte,

and in his records Walker differentiates between material obtained from fresh lamsiekte carcasses and material obtained after putrefaction. He undoubtedly produced cases resembling lamsiekte by oral dosing of the latter (putrefying intestinal tissue), but he was puzzled by the lack of uniformity in his experiments and was not prepared to diagnose the disease definitely as such. The few cases in which the symptoms suggested lamsiekte were more guardedly considered as a non-specific toxæmia. Walker also carried out experiments designed to test the pasteurilla theory, and conducted a number of feeding trials on various suspected plants, all with negative results. His attempts to transmit the disease from the living sick animal to the healthy animal by injection of blood, subarachnoidal fluid, bone-marrow, brain, mucus, gelatinous exudates, faeces, milk, mesenteric glands, all yielded negative results. This work was taken as supporting the conclusion that lamsiekte was not an inoculable disease, and not infectious in the ordinary sense of the term.

In 1911-1912, Mitchell (Second Report of Director of Veterinary Research, 1912) carried out experiments similar to those of Walker, inoculating or drenching body fluids, organ emulsions, rumenal contents, and other carcass material derived from lamsiekte cases into healthy cattle. The experiments of particular interest from the point of view of the present day are those in which crushed bones in various stages of putrefaction produced symptoms resembling those of lamsiekte. At least two of the cases must be identified as lamsiekte, although diagnosed by Mitchell at the time as due to "absorption of septic material in the bones fed." This was a natural diagnosis in view of the similar cases previously recorded by Roberts and Walker, and the enigma of lamsiekte passed unsolved.

Like Walker, Mitchell also failed to produce the disease by injection or oral administration of *fresh* material from lamsiekte cases, although every source of possible infectivity was tried. He also carried out a "muzzling experiment," in which the animals were unable to pick up food, but were still able to come into contact with the soil and vegetation by means of the tongue, through the mesh of the muzzle, and were still able to snuffle at each other. Taking all Mitchell's evidence together, it was concluded that the disease was neither infectious nor contagious, but that in some way the mouth was the channel of entry of the causal agent.

This strengthened the view that the cause might be a poisonous plant. Burt-Davy and Theiler (Second Report of the Director of Veterinary Research, 1912) travelled over a large number of farms, the former classifying suspects according to geographical distribution and the latter subsequently undertaking feeding experiments with such as were common to lamsiekte areas. The results were all negative, and Theiler discarded the poisonous plant theory in favour of what he termed the "Accumulative Poison Theory," which he considered could be made to cover most of the known facts in connexion with the disease. In this he was partially influenced by the work of Ostertag and Zuntz upon the disease "lecksucht" in East Prussia. The theory predicted the production in the grass, under certain climatic and telluric conditions, of a toxin which accumulated in the muscles, but which under certain physiological conditions could be destroyed or got rid of. The only historical interest which this theory now has is its emphasis upon the idea of a *toxin* as cause of the

disease, the toxin being now known to be produced in carcass débris by an anaerobic toxicogenic saprophyte, and not occurring, as Theiler then supposed, in the grass.

The various views prevalent in 1912 are fully discussed by Theiler in his Second Report of the Director of Veterinary Research, and serve to indicate expert opinion of that date.

The following year the "Vitamine Deficiency" theory was propounded by Stead in South Africa (*Journal of the Department of Agriculture*, 1913), and by Funk in Europe ("Die Vitamine," C. Funk, 1914, publ. Bergmann).

This theory was dismissed by Theiler, Green, and Viljoen the following year (Report of the Director of Veterinary Research, 1915).

In 1914-1915, Hedinger advanced the view that lamsiekte was a sarcosporidiosis (Pathological Investigations into Lamsiekte, E. Hedinger, Government Printer, Pretoria, 1915), a theory combated by Theiler and by Viljoen.

In 1915, Theiler also considered the remote possibility of the disease being caused by an ultra-visible virus, non-inoculable but transmitted in some way through the ingestion of insects by the cattle. Amongst other conceivable vectors, large numbers of blow-fly pupae (*Pycnosoma* species) were noted around veld carcasses of animals which had died from lamsiekte, and some of these were collected, sent to Onderstepoort, and there dosed to healthy cattle. In successive experiments with different consignments, six animals were drenched, each with from 600 to 900 pupae. Two animals died as the result of the dosing, and of these, one showed symptoms which fitted a diagnosis of lamsiekte. The symptoms of the other were less characteristic and a diagnosis of "toxaemia" was made. The carcass of the heifer whose death suggested lamsiekte was then placed in a paddock at Onderstepoort and blow-fly pupae reared upon it for further experiment. Seven animals were dosed with large quantities of these pupae, but none developed any symptoms of disease. This result was not in accordance with the idea that lamsiekte might be a specific disease transmitted by blow-fly pupae ingested by cattle, and no conclusions could therefore be drawn. The apparent production of lamsiekte in one case, however, was not lost sight of, and, taken in connexion with the earlier results of Keeling Roberts, Walker, and Mitchell, at least established the fact that a carcass may produce toxic substances which, when forcibly administered to cattle, may produce a disease that resembles lamsiekte. Four years later it was established by Theiler that naturally grazing cattle which display *depraved appetite* do in fact naturally pick up carcass débris from the veld, and that this débris, when collected and drenched experimentally, may produce typical lamsiekte.

Carcass débris, if found to be toxic, was also found capable of generating further toxin after inoculation into fresh tissue and various media suitable for bacterial growth. Not all putrefying carcass débris on a lamsiekte farm was found to be toxic, and indeed not even all carcasses of animals which had died of lamsiekte. The ordinary putrefactive bacteria could therefore not be incriminated, and the infection of the carcass débris must be specific. Further work by Theiler and his colleagues showed that the organism was an anaerobic saprophyte, Robinson finally identifying it as a member of what may be termed the "parabotulinus" group.

The observation that only cattle suffering from "pica" or "abnormal appetite" ever picked up material containing toxin, and the subsequent proof that this pica was specifically due to phosphorus deficiency of the natural pasture, allowed all the earlier observations to be brought into line. It is interesting to note that all the earlier theories of causation held *part* of the truth, but not one the *whole* truth. Hutcheon was right in predicting phosphorus deficiency as causally connected with the disease, but wrong in considering it the direct cause. It is only of etiological significance in so far as it induces an abnormal craving in the cattle, making them ingest toxic carcass débris which they would otherwise shun. Hutcheon and Borthwick were right in maintaining that the feeding of bonemeal prevented the disease; if fed in *sufficient amounts* (very large in some cases) it prevented and cured pica, and so prevented ingestion of toxin. The same result, in so far as prevention of disease is concerned, would have been achieved by burying or burning all carcass débris capable of serving as substrate for toxin production. The opponents of the idea of prevention by bonemeal feeding were also right since certain cattle develop habitual pica incurable by phosphorus feeding, while some others require amounts so high that they never came into practical consideration.

The various exponents of the "infection theory" were right in predicating a bacterium, but wrong in predicating a pathogenic one; the organism being a saprophyte which does not infect the living animal, but does infect the dead one. The believers in the various "intoxication theories" were right in so far as they explained the symptoms of lamsiekte as due to poisoning, but wrong in predicating toxins of vegetable origin, the toxin being of bacterial and the substrate generally of animal origin.

The credit of grasping the truth as a coherent whole in 1919 belongs to Theiler, and of establishing it in all its bearings, to him and the co-workers of his staff over the ensuing few years.

SECTION 2.—CAUSE OF THE DISEASE AND EXPERIMENTS UPON ITS PRODUCTION IN THE LAMSIKTE AREA.

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