

Lamsiekte of Sheep in South West Africa.*

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Not much has been published about natural lamsiekte in sheep, and, when studying the literature, above all the standard work of Theiler and his co-workers (1927), one must become convinced that the paratuberculosis in sheep does not play any economic rôle in the Union of South Africa. Though the South African authors have established the high susceptibility of sheep to the lamsiekte toxin, they state that, contrary to reports received from farmers, no veterinary records show that sheep or goats contracted the disease naturally. They state further that the only reason why sheep do not contract lamsiekte naturally is that they do not develop osteophagia to any appreciable extent, and so do not ingest the toxin.

We veterinary surgeons in South West Africa have been forced to alter our former opinion regarding the non-existence of osteophagia and natural lamsiekte in sheep. This disease has become a very important factor in the sheep industry of South West Africa, especially in its southern part. To prevent a wrong impression, I must state before going into further details, that the great majority of the S.W.A. stock consists of fat-tailed sheep (africanders and karakuls), and that my observations regarding lamsiekte are more or less limited to these types. It appears that the merino does not often contract natural lamsiekte.

The losses sustained by the farmers are by no means trifling. It is in my opinion a low estimate to state that in the lamsiekte areas about 3 per cent. of the ewes die of the disease. I know farms which lost 10 per cent. within a year, and I have received reports putting the percentage of deaths from this cause as high as 50%. Naturally these figures can not be proved scientifically, but I wish to stress the fact that, whenever I was called in by farmers to investigate the cause of a disease resulting in remarkable losses amongst their sheep, and for which they found no other name than lamsiekte, or the so much abused term "galsiekte," I had to diagnose lamsiekte.

I am not the only veterinary surgeon who diagnosed the natural lamsiekte in South West African sheep. In 1921, O. Henning, at that time Veterinary Officer of Keetmanshoop, wrote to me:—"Moreover, also the bone-eaters amongst the sheep contract lamsiekte and they are even more susceptible than cattle and succumb quickly." Dr. G. Schmid who relieved me not long ago and toured the eastern parts of

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the districts of Rehoboth and Gibeon, writes in his official report to the Senior Veterinary Officer :—" I fully confirm the opinion of Dr. S. that lamsiekte is in fact the most important cause of all the numerous deaths amongst sheep and cattle in this area."

It is evident that lamsiekte does not occur in all parts of the country. I have observed that the lamsiekte area of cattle is also that of sheep, with one exception, viz. that an area largely covered with small-leafy bush provides better food for the sheep and therefore postpones the onset of phosphorus deficiency in them. On the other hand, an area like the Kalahari, with its sand-dunes, is most dangerous for sheep. One finds there a strong growth of grass, hardly palatable for sheep, and but few bushes. The soil there is also deficient in phosphorus to a very large degree as stated by Schultze (1907).

The lamsiekte season of cattle and that of sheep generally coincide, but some small variations, due to the different kind of pasture, as mentioned above, may happen. South West Africa has practically no winter rains. The first cases of lamsiekte are observed during August and the losses increase as the progressing drought reaches its culminating point in December or January, when the summer rains usually commence.

As in cattle, the great majority of sheep affected with natural lamsiekte are females. During a heavy drought ewes may die also from starvation, but these cases can easily be distinguished from those caused by lamsiekte, as lamsiekte sheep are very often in a fairly good condition.

As regards cattle, we know that phosphorus deficiency in the pasture leads to osteophagia or allotriophagia. In South West Africa both these forms of depraved appetite considered as factors in the production of lamsiekte, are also shown by sheep and goats. This, however, is not so much noticed in the neighbourhood of the homestead or the watering place, when the thirsty sheep are hurrying to the water, or are being driven to their resting place shortly afterwards. It is in the veld generally that the grazing sheep pick up portions of carcasses with which they try to satisfy their depraved appetite. This is my own observation, but there is no reason to distrust similar observations made by farmers. That osteophagia in sheep occurs also in Australia, is reported by Seddon (1926). He writes :—" It occurs at times that cattle and even sheep develop the habit of chewing and even eating the carcasses."

Notwithstanding these observations, it is a fact that sheep do not seem to relish the protracted chewing of bones as do cattle. One must therefore look for another proof of osteophagia or allotriophagia, and this proof is furnished by the finding of **foreign bodies** in the stomach of sheep which either died of suspected lamsiekte or were killed when showing clinical symptoms of the disease. I

examined the stomach contents of 14 known lamsiekte sheep, and found in the rumen and reticulum of 8 of them the following foreign bodies :—Pieces of bones (in 4 cases), of sinews and skins, feathers, small stones, pupae shells of blow-fly, conglomerated horsehair and pieces of tissue which I considered to be parts of foetal membranes. The last-mentioned finding is in my opinion worthy of being specially commented upon. I have found parts of after-births as stomach contents in three cases out of fourteen. For this reason I made it a rule to draw the attention of sheep farmers to the dangerous possibilities of decaying after-births. My opinion became strengthened when the manager of a large merino farm in my area informed me that, when searching for the cause of losses amongst his sheep, he came to similar conclusions. This man had not received any warning in this respect, but immediately took practical steps, and by isolating the lambing ewes, and repeatedly changing the lambing camps, he claims to have achieved complete success.

This communication was of great value to me for another reason. It threw some light upon the question as to whether the merino contracts natural lamsiekte. So far all farmers had alleged that this was not the case. I have never succeeded in establishing lamsiekte in the merino sheep, whereas in the same area africander sheep and their crosses, as well as karakuls, were suffering heavily. As regards the occurrence of lamsiekte in pure-bred black-head persians, nothing can be said at present. If there is a difference in susceptibility between the fat-tailed sheep and the merino, the reason for this is not easy to find. Has the merino, reflecting as it does the artificial product of a type decided upon a century ago, partly lost its natural instinct of helping itself ?

The symptoms of natural lamsiekte in sheep are very similar to those seen in cattle, and my observations correspond with those of Theiler and his co-workers who drenched some sheep with pycnosoma larvae and pupae. Some points peculiar to the sheep may be mentioned.

A conspicuous nervous excitement, seen at the beginning of the disease is shown by frequent wagging of the tail and urinating. One of the first symptoms, the disturbance of the locomotor system, is very often accompanied by an impaired muscular action of the neck. Cattle also suffer from this paresis of the neck, but the specific anatomical structure of the sheep's neck makes this more marked. Most sheep suffering from the first stages of lamsiekte therefore offer a typical picture and can be recognised from a good distance (See Fig. 1). The attitude is like that of a lively riding horse, which is reined in by the rider. The hindquarters are moved under the body, the neck seems to be bent forward, and the head is lifted, the animal exerting its last strength to do so.

With the increasing paresis of the muscles, the neck becomes totally paralysed, and at last the head is seen doubled back on the flank.

In acute cases affection of the muscles of deglutition and mastication, paresis of the tongue and also salivation are frequently observed. The salivation is often shown as a distinct foam at the mouth. The Dutch farmers speak of this as "nat bek." When these symptoms manifest themselves on the head of a small animal such as a sheep, they are not so easily recognizable and I must confess that at first I overlooked them. The temperature of many sick sheep examined was always within normal limits.

The course of natural lamsiekte in sheep is as far as I could observe, more acute than in cattle. The great majority of the cases may be considered as acute and subacute. Chronic cases with subsequent recovery I have not encountered.

The macroscopical pathological anatomical changes in the natural lamsiekte of sheep are indeed very similar to those in cattle. Only some few points may be mentioned here. The finding of foreign bodies in the rumen and reticulum has already been recorded above. The impaction of the omasum is often very pronounced. We know that this condition, though only a symptom of disturbed digestion, has given rise to the terms "droë geilsiekte" or "droë galsiekte," used by the South African farmers to signify a specific disease. De Kock (1928), is doubtful whether the above-termed disease, "characterized by an extremely rapid course, e.g. sudden death and impaction of the omasum," can be regarded as a disease *sui generis*. In S.W.A., when farmers reported to me the occurrence of droë-galsiekte or droë-geilsiekte amongst their small stock, and I had an opportunity to investigate, I was able to diagnose lamsiekte.

In the great majority of post-mortems made on lamsiekte sheep, I found the pericardial fluid increased. In fresh cases the liquid was usually as clear as water or of pale yellow colour. I have estimated the amount to be 10 to 50 ccm. or even more. Similar findings have been recorded at Onderstepoort in cattle as well as in sheep, but it seems to me that sheep show this pericardial liquid more regularly and to a greater extent. It may be that sheep are generally more prone to hydropericardium than other animals. From many cases of acute lamsiekte in sheep, blood-smears have been taken and examined by me. I was not able to detect any microorganisms in them.

In regard to the **prevention** of lamsiekte in sheep I have given the usual advice to feed bone-meal (with certain modifications) and to remove the toxin producing material. I am pleased that my diagnosis—lamsiekte in sheep—has been confirmed by reliable farmers who sent me favourable records about successful results obtained by the

feeding of bone-meal or precipitated calcium phosphate. Some time must certainly pass before the average farmer will fully recognise the benefits of regular and early feeding of phosphorus containing supplements.

So far I have confined myself to lamsiekte in sheep. But I may mention that in S.W.A. in areas with severe lamsiekte, goats also die of the disease. These animals, though just as susceptible to the parobotulism toxin as sheep, contract the disease in a minor degree and very often display the chronic form. The cause for this difference I ascribe to the fact that the goat feeds mainly on bushes and the branches of trees which are out of reach of sheep.

Literature.

- DE KOCK, G., (1928). Diseases of Sheep in Relation to the Pasture under South African Conditions. *Jl. S. Afr. Vet. Med. Assn.* I (2). 29.
- SCHULTZE, L., (1907). *Aus Namaland und Kalahari.* 592.
- SEDDON, H. R., (1926). Botulism in Cattle from Eating Rabbit Carcasses. *Agr. Gaz. of N.S.W.* July.
- THEILER, A., (1927). Lamsiekte (Parobotulism) in Cattle in South Africa. *11th and 12th Rpts. Dir. Vet. Ed. Res. Part II.* U. S. Africa.

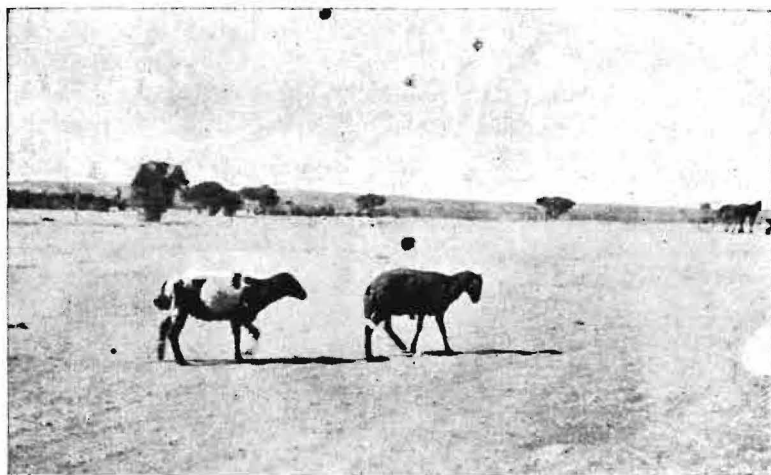


Fig. 1,

Afrikaner Sheep (on right) shows typical attitude of acute lamsiekte. There was already paralysis of tongue. District Gibeon, S.W.A.