

Osteoporosis or bone disease of horses is considered to belong to the same group as Rickets and Osteomalacia. Some authors are of opinion that the pathological process is identical with the lesions in Rickets and Osteomalacia, and that Osteoporosis is only unusually pronounced in horses where it is easily recognized as "big head". This disease is also considered to be connected with the metabolism of phosphates. As a support of this view a number of analyses were brought forward to show that not the want of phosphates is so much the cause of this disease as the unproportional low ratio of the phosphoric oxide to the lime. This conclusion is decidedly erroneous as far as Osteoporosis in South Africa is concerned. The foodstuffs analysed were not samples of the material with which the horses were fed. The observations in South Africa clearly indicated that an agency of a different character, in the nature of an infection, plays a role in this disease, and the loss of phosphates in the bones, which seemed to have been proved to be present, must also be considered not to be the primary but the secondary lesions. It is indeed nothing else but a sequel, the disease being a productive inflammatory process in the bone tissue, when new, non-calcified tissue is formed; this replaces the old tissue which disappears and the salts present are resorbed and probably eliminated.

It is not justifiable to compare the South African Osteoporosis of horses with Rickets and Osteomalacia of cattle. They are altogether different diseases, but there is a disease noticed in horses in Europe with similar symptoms, also called Osteoporosis. This disease is found in horses fed exclusively on bran, and for this reason is frequently alluded to as "bran disease". In this case the ailment seems to be connected with the foodstuffs in as much as bran contains a large amount of phosphoric oxide, the proportion being one part of phosphoric oxide to 0.09 parts of lime, but *here again it would appear that it is not the deficiency but the surplus of phosphates which causes the disease.*

Pica or Abnormal Craving in Cattle and its Sequel.—We understand, under the name of "Pica", a disease in cattle of a chronic nature, accompanied with disturbances in the digestive organs and nervous system. The animals first show a lack of appetite and suppressed rumination and occasionally constipation. Then they do not touch their usual food and develop an abnormal craving for indigestible matter, such as mortar, stones, wood, ropes, pieces of cloth, etc. In the early stages the cattle lick the walls and mangers and on rare occasions their neighbouring animals. In advanced stages this craving lasts almost constantly during day and night, when out of the stable such animals scrape away the ground and, contrary to their usual habit, will eat coarse grass and the young shoots of trees and shrubs. As soon as the abnormal appetite has developed, the animals lose in condition and waste away to a mere skeleton.

This disease is principally noted in heifers, cows, and young animals. It is of an enzootic nature, stationary in certain areas, and appearing principally in the winter. In certain localities it is present every year, whereas in others it occurs only occasionally, when conditions are not favourable for the growing of foodstuffs.

This disease was noted on the "Johannisberger Heide", situated in the moorlands of eastern Prussia, which had lately been improved by the so-called amelioration works, viz., a system of draining.

The Prussian Government appointed a commission to investigate into the cause of the disease, particularly as it had been stated that the amelioration work was responsible. The investigations showed that the disease was known in that particular locality before the area had been ameliorated, that it attacked principally young cattle which were fed with hay of the pasture, and that cattle which were fed on hay from sandy and loamy soils did not develop the disease.

The commission considered it their first duty to investigate whether the hay alone was the cause of the disease in question.

Accordingly two series of experiments were made, one in the neighbourhood of the particular area where the disease existed and the other one in Berlin.

At the first place (Johannisberger Heide) six calves were selected, born in the moorlands, and six from a healthy area. Three calves of each lot were placed in one group (I) and the same number in a second group (II). Group II exclusively received hay from a healthy area and group I a mixture of hays from the moorlands.

In Berlin also six calves from the "Johannisberger Heide" and six calves from healthy areas were placed into two groups numbered I and II.

A third group of two animals (one of which came from the moorlands) acted as controls. Group I of the Berlin lot received hay from the unimproved moorlands, and group II from the ameliorated lands. The control animals were fed on hay obtained in the neighbourhood of Berlin.

The results were as follows:—In the first instance all calves began to improve, including those which were fed on the moorland hay. This lasted for some weeks and then differences were noted. The animals which were fed on the moorland hay began to lose their appetite and condition and became infested with lice; at the same time they commenced gnawing and licking losing flesh rapidly, and some died. The first animal in the "Johannisberger Heide" died ten weeks after the beginning of the experiments, having lost in weight during this period from 112 to 90 kilo. In the post-mortem examination the following lesions were noted:—Wasting of muscles and big glands (liver, etc.), *wasting of the cortex of the long bones and abnormal brittleness, excessive anæmia.*

The corresponding control calf (a calf from the moorlands, but fed on normal hay) improved in the meantime from 70 to 102 kilo.

The remaining five calves, fed with moorland hay, offered a miserable aspect. They were gnawing wood, ate old paper and cloth. They were weighed and showed that no increase had occurred since the start of the experiment.

The control animals, viz., those fed on normal hay, had gained an increase of 25 per cent. in weight. All these control animals looked healthy, had sleek hair, and were free from all diseases. Subsequently, of the calves fed on moorland hay in the "Johannisberger Heide", three more animals died, and they showed similar lesions to those already described.

In Berlin it was also noted that all animals improved in condition at the commencement and in those of group I this improvement was more marked than in those of group II. About six weeks after the experiment started the first symptoms of Pica were noticed, but they disappeared later and the animals improved.

When inspected about four months after the start of the experiment the animals of group I showed an increase of 13 to 37 per cent. in weight.

The animals of group II, however, started to lose again in condition, and, when inspected, only showed an increase of 4.5 per cent. in their weight. They were all poor, had bad coats, did not feed well, and were gnawing pieces of leather and rope; they also ate paper and cloth when put in front of them. Subsequently some animals died, again showing the symptoms already referred to.

The control animals, fed in Berlin with normal hay, improved considerably, the one 41 per cent. and the other 61 per cent. of its initial weight. They never showed any illness.

In the course of the experiments eight quantitative analyses were made, but there was no difference in the digestibility of the organic food material in the unhealthy hay as compared with the healthy hay.

A chemical analysis of the hay showed a decrease in sodium, and accordingly there was a relative increase of potassium; *there was also a reduction in lime.*

The metabolism experiments showed that the decreased amount of sodium in the hay did not cover the want of the animal's system; more sodium was excreted by the urine than taken in by the foodstuffs, and at the same time a considerable amount of potassium salts were retained.

The retention of both limesalts and phosphoric acid was much less than would have been in the case of a normal growth of the bones.

These facts having been ascertained, it was thought to prevent and cure the disease by the administration of lime and phosphoric acid, and also with iron salts. *The results were negative.* The compensation of the abnormal composition of the hay ash by the addition of the "wanting" material did not remove the ill-effect of the moorland hay.

It was then thought that if the constituents of the ash have any significance, they should act on the growth of the vegetation. An experiment was then undertaken to manure a piece of land with sodium manure (Chili saltpetre) and some success was apparent. Further, it was thought that only certain plants may cause the ill-effects, and it was shown that clover grown on the moorlands had *no ill-effects.*

Subsequently, investigations were made to see whether the ill-effects of the hay were apparent at certain periods in the growth of the grass, and whether by any specific preparation the hay could be rendered harmless.

Three tests were made:—

- (1) Hay treated with boiling water.
- (2) Hay treated with cold water, but alkalized by ammonia.
- (3) Hay treated with cold water rendered slightly sour by hydrochloric acid.

The idea was to remove any hypothetical poisons which may have been present in the hay.

Calves which were fed for five months with steamed hay had increased by 16.5 per cent. of their initial weight; after eight months, however, they sickened. It was thus shown that the steaming had some influence on the ill-effects. The hay treated with ammonia or hydrochloric acid was in no way influenced.

An excellent means to remove the ill-effect of the hay proved to be the overheating in the stack before it was sufficiently dry.

Mowburnt hay from one of the ameliorated lands was fed to calves for a period of four and a half months, at the end of which period they had improved by 32.9 per cent. of their weight, and the animals looked very healthy and were still in good condition after the lapse of another three months. Later it was found that the preparation of mowburnt hay must carefully be carried out in order to insure overheating.

Another result was the fact that cattle which were grazed on the pasture did not develop the disease, and increased during the five months of the experiment to 35.40 per cent. of their initial weight. Sick cattle also improved when placed on the pasture.

The administration of oatmeal, linseed, grass, clover, the addition of molasses, salt, etc., did not cause any improvement in sick cattle.

The fact that the grass, when eaten green off the pasture, did not cause the disease, and the fact that the grass of the same pasture when turned into hay did cause it, led to further investigations as to a reason for this peculiar phenomenon. It was not likely that the process of the preparation of hay would be responsible for the disease, but there was an apparent difference in the grass eaten on the pasture and the grass eaten as hay. The latter belonged to that stage in the plant life when it was in flower and mature, whereas the former belonged to the earlier stages. It is possible that plants, which are harmless in the immature stage, contain noxious constituents when ripe. The experiment was carried out in the following manner:— Instead of the usual two cuttings of the grass, three were made and turned into hay. The first cut was made so early in autumn that a fourth cutting could grow which, however, being frost-bitten, could not be cut. *The first cut did not produce any disease; the second and third cuttings produced the disease.*

A further experiment was made with the “aftermath” of an ameliorated moorland. Three animals which were fed only developed abnormal “licking and gnawing.”

The last experiment was made with clover hay grown on the moorland and fed to calves. Control calves received hay off the same area. The result was that at the conclusion of the experiment the clover-fed animals had increased by 5 per cent. above the weight of the grass-fed animals. The effect of the grass collected that year (1905) was not so noxious as in the previous years of the experiment (1902, 1903, and 1904).

The results of the above-mentioned experiments, which were undertaken by Ostertag and Zunst, can be summarized as follows:—

1. The hay of the moorlands of the "Johannisberger Heide" can produce the disease "Pica" in cattle.

2. The hay of the improved lands (ameliorated) shows this effect in a higher degree than that of not ameliorated pasture lands.

3. The "disease-causing effect" of the hay on one and the same pasture varies in various years.

4. The Pica caused by the hay has to be considered as an intoxication, which affects the feeding and the metabolism, particularly that of the blood and bone formation, and is further characterized by the abnormal craving.

5. The nature of the poison or of the poisons has not been established, but since the hay causes the disease only after it has been fed over a long period, it must be concluded that the poison is contained in the hay in but small quantities, and that its effect must be of a cumulative nature. It is possible that more than one kind of poisons is present, because the feeding with "aftermath" only caused abnormal craving without loss of condition.

6. Horses could eat the hay which proved so bad for cattle without any danger.

7. Steaming destroys the "disease-causing principle" to some extent.

8. Mowburnt hay of an infected area does not produce the disease.

9. Hay cut before the grasses have flowered did not cause the disease, but the second and third cuttings did so.

10. Hay off a portion of land manured with "Chili saltpetre" was less dangerous.

11. Grazing on the pasture does not cause the disease.

12. Clover hay grown on the affected moorlands does not produce the disease.

13. Animals suffering from "Pica" improve when placed on to the pasture, provided the disease is not too far advanced. Medicines and nourishing foodstuffs had no effect.

14. The administration of salt and calcium phosphates to the food in no way removed the ill-effects of the hay.

CONCLUSIONS AND COMPARISONS.

The observations and experiments quoted in this paper enable us to form the following conclusions and comparisons.

1. There exist several diseases affecting the skeletal system, in which a deficiency of phosphates of lime is accepted, but these diseases do not seem to be identical either in the same or different species of animals.

2. It has been proved that by feeding certain animals with foodstuffs poor in phosphates and lime, a disease of the skeletal system can be produced both in young and adult animals.

3. It has, however, not been proved that the absence of such salts in foodstuffs produce the disease known as Rickets, Osteomalacia and Osteoporosis.

4. It has been shown that even the substances of which a deficiency is considered to be the cause of the disease can produce a

disease when given in excess (phosphoric oxide), and it has further been noted that various causes must be responsible for the condition known as Osteomalacia and Rickets.

5. These diseases must be considered to be due to a cause directly or indirectly affecting the bony tissue whereby the metabolism of these parts becomes so affected that the lime-salts are cast out as waste products.

6. Rachitic and osteomalatic affections present themselves by general symptoms of malnutrition and indigestion, by swelling of the joints, particularly those of the distal ends of the legs, by deformation, by stunted growth, by brittleness of the bones, and by softening of the bony tissue.

7. Some of the disturbances of the metabolism of the skeletal system are preceded and accompanied by nervous symptoms such as a depraved appetite and abnormal cravings (Osteomalacia and Pica).

8. Comparing the descriptions of Rickets and Osteomalacia with the form of stiffness in cattle described by Hutcheon, a certain resemblance can be noted.

9. It has been shown with certainty that at least one of these diseases (with abnormal craving as the main symptom, and characterized by the stunted growth of the animals) is due to toxic substances in the vegetation of certain soils, the toxin being of a cumulative nature, and its presence in the herbage being dependent on climatical conditions (Pica).

10. The disease Lamziekte as described by Hutcheon, has in common with Osteomalacia only the symptoms of depraved appetite and abnormal craving, and none of the symptoms pointing to lesions on the skeletal system.

11. It has been proved that the toxic principle contained in the hay which caused "Pica" was dependent on the growth of the grass and on certain climatical conditions; and that the toxic principle could be destroyed by treating the hay in various ways.

12. It has been proved that the supply of salts and phosphates to prevent Pica due to vegetable poisoning was of no avail.

13. It appears that the symptoms of abnormal craving can be considered to be indicative of some intoxication with vegetable matters not yet known, and it appears further that several kinds of toxins exist which are responsible for the lesions of Osteomalacia and Pica.

14. The suggestion by Robinson that the Stijfziekte he observed in the coastal districts of the Cape was connected with the sour condition of the veld in that part of the country, finds an analogy in the causes of Osteomalacia and Pica.

15. The fact that cattle show Lamziekte only after they have been for some time, even many months, on a reputed Lamziekte area before they begin to develop the abnormal craving, and still later the disease, has a certain resemblance to results with the feeding experiments of hay made in Germany in connection with Pica.

16. Leaving the pathology out of the question, there is no other disease which, in its etiological features, has so many points of resemblance to Lamziekte as the Pica which was studied in Germany by Ostertag and Zuntz.

PART III.

THE STIJFZIEKTES OF SOUTH AFRICA.

THE term "stijfziekte" is frequently used in South Africa to designate certain symptoms and conditions in cattle, the nature of which is indicated by the name itself. It describes an affection of the locomotory organs, embracing almost anything interfering with the normal movement of the limbs up to a complete paralysis. Experience has shown that there are quite a number of ailments in cattle which may affect the movement of the animal, and it depends very much on the opinion of the observer whether he calls such an impaired action "stijf" or "lam." Accordingly, the very names may become misleading in the description of the disease which, perhaps, has nothing to do with those forms of stijfziekte under consideration. This fact has been brought home to me since I started my personal investigations and inquiries.

Some years ago the disease in cattle known as "Three Days' Sickness" swept through South Africa. It was generally called stijfziekte and lamziekte, and these names would have been quite appropriate if they had not already been used for other diseases. The symptoms of stiffness and subsequent apparent paralysis were so much pronounced that, to the inexperienced, they caused much alarm. At that time, when speaking of stijfziekte and lamziekte, one had to make clear at the very outset what disease was meant, the new one or any of the old ones known by that name. In the course of my recent investigations but little reference was made to the "Three Days' Sickness" except that it was mentioned in some instances still to exist in certain parts of South Africa, a statement which, however, I was not able to verify. Most of the farmers had already forgotten its previous invasion and the anxiety it caused then. Accordingly, when speaking about stijfziekte and lamziekte, the diseases were meant which, before and after that time, were known under these names. In Bechuanaland, and also in the Orange Free State, the name "gal-lamziekte" is frequently used. It applies to a severe form of lamziekte. It does not designate a new disease, although a good many farmers are inclined to believe that the old lamziekte now shows new and more severe symptoms, and is of a more virulent nature. From the notes on this disease by Dr. Hutcheon (which he mentioned as gall-sickness) we will notice that he was familiar with it at an early date. In his later reports he does not distinguish it any longer and also calls it lamziekte. In using the terms stijfziekte and lamziekte it will be necessary to give the symptoms and lesions found in the diseases they designate. I am at present more particularly dealing with stijfziekte, and shall leave lamziekte for a future consideration. At the present time there appear to be at least three diseases which go under the name of stijfziekte, into the nature of which I will now enter.

I.—THE STIJFZIEKTE CAUSED BY THE STIJFZIEKTE BOSCHJE (*Crotolaria burkeana*). THE CROTALISM OF CATTLE.

This stijfziekte was described by me in the first number of the *Union Agricultural Journal* as a laminitis of cattle, the seat of the affection having principally been found in the horn-forming membrane of the hoofs. As a result of an acute inflammatory process the animal walks on its heels, with the forelegs placed forward and the hindlegs put underneath the abdomen, with the back arched, and showing much pain, like a foundered horse when forced to walk. As a result of this, the toes of the hoofs lose touch with the ground and begin to turn up, the digits separate and grow out. Frequently the skin around the coronary pedal joints forms a groove, the axis of the distal end of the foot which, from the fetlock to the hoof, should run in a straight line, having a distinct kink. Underneath the coronet in the wall of the hoofs a shallow groove or a ring is formed, narrow in front and gradually broadening out on the side as it progresses downwards. There may be a succession of grooves or rings. In the course of events the toes of all four feet turn up and grow out; sometimes this is more prominent in the hindlegs and on other occasions more in the forelegs. The hoofs of the hindlegs seem to remain abnormal much longer than those of the forelegs. The grown-out horn may disappear as a result of being worked down through walking, when the hoofs again take to their former shape and the gait of the animal becomes normal once more. This does not always take place, and even when the hoofs are sawn or cut down the gait does not of necessity regain its normal action: the hoofs remain turned up and the toes commence to grow out again.

During the acute state of the attack, the hoofs are warm, they appear more bright in colour, and when normally of white horn they may even have a reddish hue. When percussed with a stone the animal shows pain by withdrawing the foot. The alternate lifting of the feet and changing of the weight of the body from one side to the other are the first symptoms, and such animals show the characteristic laminitic walk. In this stage the animals frequently lay down, they lag behind the rest of the herd, and begin to lose in condition. Later the pain in the feet seems to lessen, the animals still walk stiffly, and after the hoofs have grown out there seems to be not so much pain, but there is mechanical interference of the long toes with the gait, the animal lifting the legs somewhat abnormally, and a peculiar rattling noise becomes audible, particularly when the animals are driven on, due to the sound of the dry horn touching the ground.

This disease does not, as a rule, lead directly to death, but indirectly, inasmuch as some of the animals through not being able to walk about lose so rapidly in condition that later on they are unable to rise, and die of debility or starvation.

The *cause* of this stiff-sickness has been experimentally established by feeding of the so-called "stijfziekte boschje" (*Crotolaria burkeana*). These experiments were made in two different places in the Transvaal, viz., in Barberton and in Zeerust. It is true that in some cases large quantities of the plant were fed. This was done to make sure that the plant was poisonous, but also, when smaller quantities of

the herb were used after the lapse of only a few days, the disease developed. Accordingly there is no doubt that the stijfziekte which I described above is caused by the plant *Crotalaria burkeana*.

The article in question in the *Agricultural Journal* dealing with this stijfziekte drew the attention of many farmers to this plant. They were on the lookout for it; some found it, and in sufficient quantities, so that its presence would account for the presence of the disease on their farms. Some, however, found it in such rare numbers that it could hardly be connected with the disease, and a good many more did not find it at all and yet they had a disease, stiff-sickness, amongst their cattle, and sometimes even in an alarming manner.

This fact was brought to my notice from answers in the query sheet issued for the purpose of information concerning lamziekte in cattle, by correspondence and verbal discussions with farmers, and it forced me to the conclusion that as a cause of stijfziekte there must be other plants responsible, and that not all stijfziektes are identical. It is possible that even other causes than plants may play a role.

Personal observations convinced me of this fact when visits were made into the various parts of South Africa inquiring into the nature of the disease under discussion.

II.—STIJFZIEKTE NOT CAUSED BY CROTALARIA, FREQUENTLY COMPLICATED WITH JOINT LESIONS.

In this form symptoms similar to those found in the acute laminitis as caused by crotalaria may be present. The animal walks exactly in the same way, with arched back, taking short steps, keeping the fore feet forward and placing the hind feet underneath the abdomen; the digits separate and grow out, and they may even grow out to some length. Rings appear underneath the coronary band and the horn seems to be brighter in colour. Although in these respects the disease resembles crotalism very closely, so far I have not yet seen a case where a turning up of the digits has taken place and is so pronounced as in the former disease. The hoofs seem simply to grow longer and to broaden out toward the toes, but the toes remain in touch with the ground, and after the attack is over the abnormal length is worked off by walking, normal conditions returning. Frequently, however, an animal is marked forever by the abnormal length of the toes and by the turning in of the walls on the side. It seems also that in this second form of stiff-sickness the animals do not go down so much on their heels as in crotalism, which accounts for the toes not turning up.

The animals in the acute stage also lie down frequently, lag behind, and lose in condition.

The symptoms in the hoofs pointing to the seat of the attacks may be present alone, frequently, however, there are other lesions present pointing to an affection of the distal joints and particularly the fetlock joint seems to be the one most affected. Examining it from the front, it seems enlarged, and on both sides, inside and outside, between the bone and the flexor tendon, a growth of the bone bulges out like wind-galls in the horse. These tumours are hard, and are nothing else than a diffuse thickening of the lateral portion of the distal end of the bone.

Attention must be drawn here to the fact that in young animals, about one to two years old, principally in oxen and more particularly in animals crossbred from Africander with imported bulls, and very noticeable in the Friesland breed, prominent joints and thick ends of the canonbones are noticeable. In stiff-sickness, however, this swelling is so to say exaggerated and can be recognized without difficulty in the Africander breed where the bones are not so heavy and the joints not so thick.

In some cases the thickening can lead to an abnormal form and shape of the joints, the coronary joint underneath may also be involved and the whole distal end of the foot looks abnormally thick.

These joint lesions may or may not be accompanied by abnormal growth of the hoofs. Their presence causes stiffness and lameness similar to the laminitic form of stiff-sickness or crotalism, but usually placed on the limb, when pain is felt and the animal strives to relieve the leg as quickly as possible. Hence the short and quick steps. So far the post-mortem examinations have shown that the joint itself is not affected, it is the lower end of the canonbone, the epiphyses, which are enlarged to a hard and solid diffuse tumour. Cutting into it did not show any softness—it was hard, and the same resistance was felt as when an attempt was made to cut normal bones with a knife. Not only thick joints can result from this disease, but also abnormal position of the legs can be noticed. Some animals become pronouncedly knock-kneed, so much so that in walking the knee-joint of the one touches that of the other leg, or in the hindlegs the hocks may turn inwards and the feet outwards; in other cases the distal ends of the legs, from the fetlocks downwards are turned in an outward direction. Other joints, viz., knee-joint and hock may be enlarged, and sometimes the canonbones show thickening. Bending of the canon-bone can be noticed and as a result of this an abnormal gait develops.

The animals so affected show unthriftiness, they are stunted in their growth, remain small, have a rough coat, and they develop a big abdomen.

This disease affects young stock, heifers and tollies, and cows with a calf at foot. It begins to show itself particularly soon after calving, viz., within a month. When the calves are taken away and the cows are not milked they seem to recover more quickly. The same animal may at the next subsequent calving show the disease again. It has been stated that oxen used in transport and harnessed in the plough are not at all or at least much less susceptible to stiff-sickness. Also old animals develop this disease sometimes, although the younger ones are generally admitted to be those principally affected.

I have seen this form of stijfziekte in cows, heifers, and tollies in Bechuanaland, in heifers of the Western Transvaal, in calves of the Mafeking district, in cows on a farm in the district of Bloemfontein, in young heifers near Stellenbosch, in cows and oxen up to four years of age in Grahamstown, and lately in Middelburg in the high veld, Transvaal. I am also informed that it is present in Natal, and the description given to me, at least seems to support this view.

From two places in the high veld cows suffering from the acute stage (before any changes in hoofs or joints had resulted) were

brought to the Onderstepoort laboratories, where they were kept for the purpose of close observation. These animals showed the characteristic gait. They were both stiff, viz., had the foundered walk and both had lost in condition. Soon after their arrival they seemed to improve, they put on flesh and after the sojourn of a few weeks they had recovered. They now walked freely and showed no pain. The disease in the high veld of the Transvaal was noticed by the Government veterinary officers and brought to my notice in the districts of Middelburg, Lydenburg, Carolina, Ermelo, on some farms in Standerton, and also in the district of Pretoria. It is likely to exist in many other districts.

An interesting observation was made that the disease is only found on certain farms and not on others. Moving of the cattle from such farms to healthy farms stops the spread of the disease and helps to arrest the development in those already affected. It has also been stated that on such farms where the disease is rather bad, an unusual craving for bones and refuse on the ash-heap is shown, and that notwithstanding a liberal supply of bonemeal and salt it is still met with.

On some farms a few cases were noted during the last five or six years, but owing to their scarcity no notice was taken of the disease. During the last two years and perhaps more so during the last year, losses have become more frequent and some farmers are inclined to connect this fact with shortage of the rainfall or with the absence of well-distributed rains during this period.

Up to the present the information as to the nature of the pasture of the farms on which the disease has been found was uniformly given as that of sour veld, and it is stated that it is not found on sweet veld; a movement from the former to the latter is considered of beneficial influence. Although the sour veld is the dominant character of the high veld, yet the disease is not met with on all farms and it is a curious fact that in one and the same neighbourhood of farms adjoining each other, with apparently the same grass, one farm is badly infected with the disease and another is absolutely free. Farms in valleys do not seem to be infected. Some of the farms on which the disease was noted were carefully examined by the farmers themselves, by the Government veterinary officers, and by the Botanist, Mr. Burt-Davy. *Crotalaria burkeana* could not be found and this fact brings home the conclusion that the disease must be due to other causes. The question naturally arises whether the two forms of stijfziekte, the foot and the joint form, as described now under the title of the second form of stiff-sickness, are identical, or whether here also separate causes are responsible for the different lesions. At the present time I am inclined to consider both to be due to the same cause. My reason for this opinion is the fact that both forms are usually met together on one and the same farm, and occasionally in one and the same animal. It seems to me, however, that in female animals the hoofs are the seat of predilection whereas in tollies more frequently the distal ends of the canonbones are affected.

A definite decision will only be possible once the cause is known and the disease can be produced experimentally.

Non-identity of this Disease with Lamziekte.

This second form of stijfziekte is, to my mind, undoubtedly identical with one that has been described by the late Dr. Hutcheon, whose notes I have given in detail. In comparing my observations with his there is practically no difference as far as the joint form is concerned. Concerning the affection of the feet, we notice that Hutcheon does not draw the attention to the turning up of the toes.

I do not now think that Hutcheon ever saw the form I described in the first number of the *Union Agricultural Journal*, and which is caused by crotalaria, and I must admit that the form which I have described just now, and which I consider identical with Hutcheon's, was never before brought to my notice. It only came to my knowledge subsequent to the publication of my first article, as a result of visiting many farms in connection with lamziekte, and as a result of correspondence with Government veterinary officers and farmers. The reason for the misunderstanding is an obvious one. In certain stages both diseases resemble each other so much that a differential diagnosis is not always possible without actually going into all the conditions under which they are met with.

This second form of stijfziekte which for convenience' sake, until we have a better name deducted from the cause, I shall call the complicated form, is considered by Hutcheon to be identical with lamziekte, viz., to be one form of lamziekte, and the reasons for this view are clearly exposed in his own words in the first part of this article. The main reason perhaps is the fact that Hutcheon, in his investigations, met this disease practically on all such farms where lamziekte was known to exist at one or another time; if both were not together at the same time the stijfziekte was usually the first to be noticed, and on such farms the craving for bones was usually very noticeable, and subsequently the lamziekte was reported to appear or to have appeared.

In my own investigations I met this second or complicated form of the disease for the first time in the lamziekte area, and on a farm on which the latter disease was reported to be very prevalent. Subsequently it was reported on farms on which there was never any lamziekte at all, and finally it has been found to be prevalent in the Eastern Transvaal, in the high veld, and in Natal, on farms where there is no history of lamziekte. This fact, therefore, warrants the conclusion that the second or complicated form of stijfziekte is not connected with lamziekte.

Cause of the Disease.

The conclusions Dr. Hutcheon had arrived at was that this form of stijfziekte, like that of lamziekte, was due to the want of phosphates in the foodstuffs. From Dr. Hutcheon's description of the disease, as well as from mine, it will be seen that this second form of stijfziekte resembles, to a certain extent, the diseases rickets and osteomalacia in cattle, and Hutcheon, indeed, identified the latter with stiff-sickness. In order to explain the swellings of the epiphyses, the deformation of the joints, the thickening of the bones, and the distortions of the limbs themselves, a softening of the bony substance may be accepted, during which period the weight of the body may lead to these deformations,

and it may be that whilst the acute process is proceeding there is a deficiency of phosphates of lime in the altered parts. If stiff-sickness is identical with osteomalacia, then the want of the mineral salts is not limited to the affected bones alone, but the whole skeleton must show a shortage. Accordingly we expect that an analysis of the bones of stijfziekte animals will give us this information, and a comparison with the analysis of healthy bones will show differences. For comparisons take the results of analysis of the bones of animals suffering from crotalism are repeated here. The analyses were undertaken by Messrs. McCrae (complicated form), Ingle (crotalism and normal, Nos. 543 and 569), and Vipond [normal (three) and debility.]

Extract of Analytical Results.

No. of Animal.	Disease.	PHOSPHORIC OXIDE TO ASH.	LIME IN BONES.	PHOSPHORIC OXIDE TO LIME.		LIME IN HEALTHY AND DISEASED.		PHOSPHORIC OXIDE IN HEALTHY AND DISEASED.	
		Percentage of phosphoric oxide in 100 parts of ash.	Percentage of lime present in 100 parts of ash.	Ratio.		Ratio.		Ratio.	
				Phosphoric oxide.	Lime.	Nor-mal.	Di-seased.	Nor-mal.	Di-seased.
2020 2022 2023	Joint form.....	% 39·93	% 56·68	1	1·453				
		41·55	56·35	1	1·356				
		41·50	57·47	1	1·385				
	Mean.....	$\frac{122\cdot98}{=40\cdot99}$	$\frac{170\cdot50}{=56\cdot83}$			1	1·05	1	0·97
563 568	Crotalism.....	41·42	55·85	1	1·348				
		40·85	55·76	1	1·365				
		Mean.....	$\frac{82\cdot27}{=41\cdot13}$	$\frac{111\cdot61}{=55\cdot80}$			1	1·03	1
543 639 3	Normal.....	41·5	55·21	1	1·330				
		44·6	52·85	1	1·183				
		40·2	54·17	1	1·341				
	Mean.....	$\frac{126\cdot3}{=42\cdot1}$	$\frac{162\cdot23}{=54\cdot08}$						
4	Debility.....	39·8	53·78	1	1·351	1	0·99	1	0·95

The comparison of the analyses of the bones of three healthy cattle show the minimal percentage of phosphoric oxide in 100 parts of ash to be 40·2 per cent., and the maximum 44·6 per cent.; the analyses of bones of stijfziekte animals (complicated form) varies from 39·93 per cent. to 41·55 per cent; the difference between the two minimums is 0·27 per cent., in favour of healthy bones, and the difference of the two maximums is 3·05 per cent., also in favour of the healthy

bones. The average in stijfziekte bones would be 40.99 per cent., and in healthy bones 42.1 per cent., and the difference would be 1.11 per cent. There is, accordingly, a slight difference in favour of the healthy bones, but, taking the two minimum and averages into consideration, the discrepancies only amount to just over 1 per cent.

Concerning the percentage of lime present in the ash of the bones the comparison of the analyses of the bones of three healthy cattle shows a maximum of 55.21 per cent., and a minimum of 54.17 per cent., or a variation of 1.04 per cent. In the diseased bones the maximum amounted to 57.47 per cent. and the minimum to 56.35 per cent., or a variation of 1.12 per cent.

Of further interest to us will be the ratio of phosphoric oxide to lime. This ratio in healthy bones in three animals reads as follows: 1 : 1.330, 1 : 1.183, and 1 : 1.341, and in three diseased bones 1 : 1.453, 1 : 1.356, and 1 : 1.385. Accordingly, there is a slight discrepancy between the two, there being less phosphates or more lime in the diseased bones as compared to the healthy bones.

The slight discrepancies may be accounted for when we compare the above analysis with that of a beast which died of debility and which, in other respects, was healthy. In this case the percentage of phosphoric oxide in 100 parts of ash found to be 39.8, or less than was found to be the minimum in stijfziekte bones.

If we remember that stijfziekte in advanced cases causes loss of condition in the animals, due to malnutrition, then we understand the discrepancies in the analysis without accusing the disease of being responsible. A greater discrepancy may, however, be found in the bones actually affected, and this point requires further investigation. Comparing our results with the notes concerning the pica (or licking) disease in Eastern Prussia, we find that in this disease a slight reduction in the quantity of lime salts was also noticed. It was then thought that the supply of these salts in the food would prevent the disease, but it did not, thus showing that the want of the salts was not the cause of the disease. Comparing furthermore the results of our investigations with the information collected about rickets and osteomalacia, we come to a similar conclusion that the disease cannot be due to the want of phosphates in the foodstuffs, but very likely to some toxine acting on the bony tissue and more particularly on the bone-forming membrane. The inner layers of the periosteal membrane of the pedal bone, as well as that of the metacarpal and metatarsal bones are probably chiefly attacked. These being the parts on which the animal's weight is immediately bearing, we understand that they suffer most, and the response to the injury is an inflammatory process with symptoms of laminitis and periostitis. In crocotalism we have experimental proof that a certain plant, *Crotalaria burkeana*, can produce a definite disease so similar in many respects to the one under discussion. This demonstrates the action of a poison, and we have no difficulty in understanding that the second form of stiff-sickness is also caused by some vegetable poisoning. So far, we have not been successful in finding that plant. A number of suspects have been experimented with, all belonging to the family of Leguminosae, but no definite results have, as yet, been obtained, although some animals developed a stiff-sickness of a passing nature not sufficiently severe to call it stijfziekte. I feel confident that this question will be solved by experiments, but there

need not only be one plant—there may be more. We are justified in thinking that such plants are not always poisonous, but only at certain times and under peculiar conditions of temperature, humidity, and soil. In this respect we have made a very interesting observation; the plant commonly known as ragwort (or, in Natal, as Dan's Cabbage) *Senecio latifolia*, proved, in the experiments of Robertson in Grahamstown, to be very poisonous for cattle and horses; after a comparatively short time a small quantity killed the animals which were fed on it. In Mooi River, however, under the supervision of Mr. Webb, Government Veterinary Surgeon, large quantities of the same plant fed to cattle and horses proved to be harmless. This difference might be due to the influence of different environments on the plant itself.

The vegetable poison theory is in no way contradictory to our experience of the disease. The fact that young animals are mostly attacked would mean that the tissues in question in this stage are more susceptible than in later stages. The same may apply to young cows in calf. In addition, such animals eat a much larger amount of food than oxen, and, owing to depraved appetite, sometimes eat unusual foodstuffs. Working animals being less subject would mean that in these the toxins would probably not be absorbed by the susceptible tissues, being rendered harmless in the blood before they reach it. If we take into consideration the fact that the disease is one of the sour veld, then we have practically an indirect indication of its being connected with the flora. When discussing the cause of lamziekte the explanation given there may also prove, *mutatis mutandis*, to be the correct one to apply to stijfziekte.

III.—THE LAMZIEKTE FORM OF STIJFZIEKTE.

The third form of stijfziekte may be considered to be but a slight form of lamziekte.

The disease is characterized by a peculiar gait, the upper parts of the legs, the shoulders, being affected and not the lower ones. Accordingly, there is not so much stiffness as there is interference with a free movement of the shoulder. The animals decidedly walk with shorter steps and there seems to be pain as a result of movement of the limbs. It is principally the forequarters that are affected, and there is no deformation in the hoofs or joints nor is there any swelling of the limbs.

The stiffness may last only a few days, a few weeks, or may be protracted for some months. It is noted on farms where there is no stijfziekte, as described before, and it is frequently the only form of lamziekte met with. It is usually the first symptom of lamziekte and frequently remains the only symptom, the disease taking a mild course and ending in recovery.

CONCLUSIONS.

1. Four forms of stijfziekte in cattle exist, namely—

- (1) The foot or laminitic form (crotalism); (2) the foot and joint (the complicated form); (3) the one described by Robinson which I am not yet exactly able to bring to book; and (4) the lamziekte form.

2. The foot or laminitic form is caused by eating a certain plant *Crotalaria burkeana*, the toxic principle of which seems to affect the

laminæ of the hoof whereby symptoms are produced resembling those in foundered horses. The disease may be called crotalism.

3. In the foot and joint, or the complicated form of stijfziekte, the seat of the lesions is either in the hoofs or in the metatarsal or metacarpal bones. In these a diffuse periostitis is present, and the disease may therefore also be called the periostitic form of stijfziekte.

4. The cause of this second form is not yet known; the idea that a want of phosphates in the food is the cause of the trouble cannot be upheld, although it is possible that there is a disturbance in the metabolism of the phosphorus owing to the existence of an inflammatory process caused by the toxins.

5. The slight discrepancies in the amount of phosphates in healthy and sick bones must be looked upon to be a sequel of the disease.

6. The periostitic form resembles, to a certain extent, that of rickets, inasmuch as young growing animals are attacked, but it differs from it in the actual pathological changes as defined under the heading of that disease.

7. It resembles more nearly osteomalacia, and may prove to belong to this group of diseases; the etiology of osteomalacia has, however, not yet been satisfactorily cleared up.

8. The symptoms of depraved appetite, such as abnormal craving, must be considered as part of the disease, similarly to the case of pica, the disturbed appetite may be conducive to a further development of the disease.

9. Comparisons of the second or complicated form of stijfziekte with crotalism, osteomalacia, and pica allow of the interpretation that it is also due to a vegetable poison.

10. Stiff-sickness also forms a symptom of lamziekte and is frequently mistaken with the stijfziekte described above.

TREATMENT.

No specific treatment is yet known, but there are many in use by farmers. Judging from the similarity of stijfziekte to laminitis in horses, the same treatment may be applied, providing the attack is an acute one, namely, freely bleeding from the jugular vein or a good clearing out of the bowels with a purgative. Bleeding in an advanced stage, that is to say when the disease has become chronic, will do harm. As long as the lesions are not too severe, exercise of the animal can be recommended; swimming may prove advantageous. If the pain in the feet is great, relief may be obtained by letting the animals stand in water. In all cases a change of food should immediately take place by substituting green mealies, green barley, or similar crop, or by turning the animals into sweet grass. Cows in milk should no longer be milked and their calves should be taken away. When the disease attacks a number of cattle in the herd, then it is time to change pasture, namely, to trek from the sour veld into the sweet veld. We do not know of any lick which will with certainty keep away the disease, but frequent administrations of sterilized bone meal mixed with salts is still to be recommended. Experiments are still in progress with this disease, and perhaps the suggestion which I will bring forward under the treatment of gal-lamziekte may also apply to the stijfziekte.

PART IV.

LAMZIEKTE OR GAL-LAMZIEKTE,

A good and complete description of this disease, both from a clinical and pathological anatomical point of view, was given by Spreull in the *Agricultural Journal of the Cape of Good Hope*, Vol. XXXII, No 5, under the title of "Lamziekte on the Kaap Plateau."

In the following description I have made use of his notes as well as those of my assistants, Dr. Frei (in the Report of the Government Veterinary Bacteriologist for the year 1908-09), Mr. Walker, and Mr. Mitchell, who during the last two years have had an extensive experience with lamziekte; some personal observations; and last, but not least, those of farmers have been utilized as well.

Four different clinical forms may be distinguished:—

1. *The Per-acute form.*—In history and course this form much resembles anthrax. Frequently the animal is to all appearances in good spirits when seen last; when in milk the cow is reported to have given the usual quantity at the last milking. She is then either found dead the next morning in the kraal or, when turned out, does not return in the evening with rest of the herd, and when looked for is found to be dead. In other instances the animal is noticed to be very ill indeed and dies soon afterwards.

A good number of smears of cattle with the above history were submitted for examination, and frequently the suspicion was expressed that death may have been due to anthrax. This suspicion was in some instances justified, since on many farms on which lamziekte is prevalent anthrax is also met with. The reverse order of things has also occurred. Animals suspected of having died of lamziekte were found on microscopical examination of the blood to have died of anthrax.

In the Eastern Province an acute disease ending in death is known to occur in cattle and is called "imapunga" or blacklung. There is considerable controversy about the nature of it. Some people consider it to be specific and in no way connected with any of the known ones; others are emphatic in their opinion that it is identical with anthrax; and still another group think it is a very acute form of lamziekte. Judging from the statements expressed that imapunga is most frequently, perhaps invariably, found on such places where the well-known lamziekte is also present, and that it is absent where that disease is absent, I am inclined to place it under "Per-acute Lamziekte."

The examination of blood smears gives negative results in per-acute lamziekte, and this fact, together with the history emanating from a lamziekte area, permit of the diagnosis. The post-mortem lesions in such cases reveal nothing characteristic, and this may also be utilized in the diagnosis.

2. *The Acute Form of Gal-lamziekte.*—This may last from two to three days to about a week, and in very rare cases for a longer period. The animal is noticed to lag behind when the herd is driven or returns

to the kraal, or when out grazing is found to isolate itself from its mates looking for some shady place or some other protection, and when such are near by, there it will lie down. Some farmers state that occasionally animals which are about to sicken keep to themselves before the symptoms of illness are actually seen.

When found walking and lagging behind, the animal—with its peculiar gait, a kind of unsteady walk—at once catches the eye of the examiner. The animal is generally described as walking heavily or waddling and as being stiff; the stiffness at the beginning may be so slightly pronounced that the impaired action may escape the inexperienced eye. When more distinctly pronounced it can be seen that the shoulders seem to be the part most affected and the action of moving the limbs appears to be impaired. The animal takes shorter steps, seems to lift its legs in an abnormal way as if it had difficulty in moving them; sometimes the walk is halting, and the shoulder blade seems to be more erect so that the top reaches over the withers or runs parallel to them, the withers appearing to be sunk in between the two blades. The back appears to be arched. It is only in rare cases that the elbows are noted to be turned out. There are all possible transitions in the severity of the symptoms from a slight, hardly noticeable, stiffness to an actual impossibility of walking. Animals thus attacked in a severe form frequently lie down or rather they seem to drop down contrary to the leisurely way in which a beast generally does when it wants a rest. They are unwilling to rise when forced to do so. When attempting to rise in the early stages of the disease they may succeed, but the animal will only walk a short distance—sometimes only a few yards—before it goes down again. Sometimes a beast keeps on going down and rising alternately. At a later period the animal still attempts to rise when forced, but can only lift its forequarters and then sinks back again to its former position. There is frequently a shivering of the muscles of the shoulder. In a still later stage the animal, although willing to rise, can no longer lift its forequarters, and finally gives up all attempts. It remains for some time in a normal position, viz., lying on the breastbone, supported by the two front legs doubled up underneath the body. When attempts are made to lift the animal on its legs it is found that it can no longer support the body, and sinks back immediately when no longer supported. In some instances the animal bends its head and neck back, the head resting on one side, and it remains in this position until death sets in. When, during life, attempts are made to stretch the head out it snaps back like a spring. In other instances the animal is unable to support itself in its resting position; it lies flat on one side and stretches all four legs out. It may be motionless or may show spasmodic cramps; the back may be stretched out straight or bent back. It is only rarely that the sensitiveness of the hindquarters disappears, and usually the animal reacts to pinching of the tail or pricking with a needle or knife in the legs. It can move the legs, but it cannot support its own weight.

Apart from stiffness the general appearance of the animal in the beginning of the disease is only slightly or in no way altered. The head is carried free, the eyes are clear, the ears are pricked, the muzzle is moist, the hair is smooth and normal. The animal may still feed and drink normally. It is only when the symptoms of paralysis become pronounced that a change for the worst takes place. The food

remains untouched and rumination ceases. The muzzle dries up and may even crack; a mucous discharge may be noticed from the nostrils and grinding of the teeth may be heard; an increase salivation may be seen; the saliva hangs around the lips and drips down in strings; the ears droop; the eyes appear sunken; and the coat stares.

The defæcations of the animal may appear normal or sometimes constipation may be present; in some instances diarrhœa was noted. The faeces, dry or watery, may be mixed with flakes of mucus or blood, dry faeces are also frequently covered with mucus.

Cows in milk soon show a decrease in the yield or even a complete stoppage, and experienced farmers informed me that the cessation of milk is frequently the first symptom of the approaching disease, and accordingly is considered to be alarming.

From per-acute to acute stages there are transition forms. The symptoms of stiffness may be absent or at least overlooked. The paralysis is noticed to be the first symptom and the animal is unable to rise again. The picture of the disease, as described above, represents the majority of cases of acute gal-lamziekte, although all the symptoms given are not always present or appear exactly in the order enumerated. There are forms of acute gal-lamziekte with more alarming symptoms and lesions in addition to those already mentioned. These affect principally the masticatory and deglutitive organs. Apparently a paralysis of the pharynx and oesophagus are present, the animal desires to eat, it seizes the food and is still able to masticate, but is unable to swallow it. Such animals may die with a bolus of food in their mouth, and during life green-stained saliva hangs about the mouth. In other cases the tongue is paralysed, hanging out of the mouth, or when pulled out cannot be drawn in again. The paralysis can even cause the lower jaw to hang down, the animal being unable to close the mouth. When the head of such an animal is taken by the horns and shaken, the lower jaw will move easily and the knocking of the upper and lower molar teeth against each other can be heard. In some animals occasionally a stage of excitement is noticed. It has a wild look, shakes its head, and when molested is inclined to charge. When down it has still the same savage appearance and becomes restless when approached, although it is unable to rise. When completely paralysed and lying flat out on the ground the animal may show spasms of the neck and stretching out of the head.

Attention must be drawn to an important fact in all forms of lamziekte, viz., the absence of fever. In all cases where temperature records were kept this observation has been made, and it was even found that as a rule subnormal temperatures are recorded. This fact may be considered as pathognomonic. Attention must be drawn here to the observation that the normal temperature in healthy animals exposed to the sun and all outside conditions varies within wide limits. It is by no means rare to find these extremes to vary between 4 to 6 degrees from morning to evening. In lamziekte the tendency is not to vary so much, but to remain with the lower limit.

The acute cases nearly all end fatally, and with rare exceptions the earlier an animal goes down the sooner it dies.

3. *The Sub-Acute Form of Lamziekte.*—This is called by the farmers the old lamziekte of former years. As pointed out previously, the idea that in the former days only this form was known is not quite

correct, Hutcheon having mentioned the acute form in one of his earliest reports under the name of gall-sickness. It may be true that recently more acute cases were noted than sub-acute ones, and this may be explained by accepting that recently the best conditions for the development of the acute form have been existing. We shall understand this fully when the cause of the disease is explained. There are no different symptoms present in this form of the disease. They appear in the same order, but do not succeed each other so rapidly; they take a slower course and are not always so pronounced. The animal becomes paralysed in the back and cannot rise. It continues to feed, and if food is not supplied it drags itself in search of it, and finally all the grass in the neighbourhood becomes eaten off. When water and green foodstuff are supplied, such an animal can live for several weeks. It is to all outside appearances normal, except that it is unable to rise. When lifted up it can occasionally stand, but soon goes down again. With improvement the animal can sometimes stand for quite a long time on end. Frequently it happens that such an animal, after it has been down for weeks, will rise suddenly and walk away. Other animals weaken rapidly after they are once down, lose condition, their eyes sink, decubitus sets in, and they either succumb to debility or are killed to save them from further misery. In other cases after an animal has been down for some time and apparently showing only paralysis of the back, it will suddenly take a turn for the worse and die within a short time. Animals which recover may be stiff for months.

4. *The Stijfziekte form of Lamziekte.*—We might distinguish in lamziekte a stage of stiffness previous to paralysis and consider this form as lamziekte without paralysis. We are justified in doing so since the stiffness is the main symptom in this disease. The back is arched when walking. There is practically no other symptom present, the animals continue, as a rule, to feed, or when temporarily off feed soon resume their appetite again. Cows in milk will show a decrease in the production. The duration of the stiffness may be anything from a few days to a few weeks or months, and some animals do not regain their normal gait at all. They lose in condition and have difficulty in regaining it. This form may be called the mild form of lamziekte since, in the majority of cases, recovery is the rule. Dr. Hutcheon has also noticed this form, and it appears to me that he placed this form together with the second form of stijfziekte which I have separated above. His remarks about stijfziekte being present on all lamziekte farms is therefore correct as far as this form is concerned. Our experience has shown that on one and the same farm there may be lamziekte and stijfziekte present in all the forms we have described.

Post-mortem Lesions in Lamziekte.

There are no definite characteristic lesions found in this disease. It is therefore of little use to enter into details of the lesions which have been registered by the various investigators as their reports do not completely agree, a sign that there are no constant changes to be found.

It is generally admitted that an increased amount of liquid in the subdural space of the spinal cord was perhaps one of the most certain symptoms, but from reports at my disposal from veterinary surgeons who have given special attention to this point, this does not seem to be the case at all and it is even doubtful, when an increased amount of liquid is found, whether it is connected with the disease or not. Accurate measurements of the liquids found in healthy animals or in animals suffering from other diseases have shown that enormous differences exist, the quantity varying from 50 c.c. to 250 c.c. in animals of the same age. Accordingly we cannot place much reliance on the statement when the liquid is said to be increased in lamziekte. It has also been said that the spongy substance of the vertebrae and of the long bones of the limbs are generally more injected than in healthy animals and that the bone substance itself is softer than usual. None of the recent investigators support this statement, and they do not find any difference in the hardness of the bones in healthy or sick animals.

Changes in the digestive organs are perhaps most frequently found, but they are usually so little pronounced that they may escape the notice of the laymen. Considerable attention is given to the gall bladder by the layman. The veterinary surgeons' reports show that in some instances the gall bladder is filled with normal bile, rarely with thick turbid bile, but frequently nothing abnormal is found. Laymen also attach importance to the third stomach, which is usually reported to be very dry. The reports sent to me show that this is not always found, as it is frequently noticed to be soft and normal. More constant lesions are found in the intestines, but care must be exercised in identifying them with lamziekte, since the majority of animals have been treated with reasonable or unreasonable medicines as the case may be, and as the result of this hyperaemia and even inflammation of the mucous membrane of those parts may be produced.

In the fourth stomach the presence of a hyperaemia or inflammation may occasionally be found, the mucosa being swollen and diffusely reddened.

Most of the reports at my disposal dealing with animals not treated in any way show the presence of a slight muco-catarrhal enteritis, especially of the small intestines, which may or may not be accompanied by hæmorrhagic extravasations. This latter is occasionally very pronounced.

Full attention has also been given to the condition of the brain and its envelopes, and some observers noted the presence of an injection of these envelopes in a certain number of cases. But the reports are not uniform, a fact which shows that there must be differences in individual cases.

Attention may perhaps be drawn to the lesions of decubitus in animals lying for some time, when an infiltration of yellow liquid will be found in the subcutaneous tissue.

Microscopical examination of blood of a great number of cases have in no instance indicated the presence of any specific organism or even in any lesions which might be considered to be constant or typical of the disease. The same applies to the examination of the internal organs, including the brain and the spinal cord.