INVESTIGATIONS INTO THE DISEASE
LAMZIEKTE OF CATTLE.

By Dr. WALTER FREI.

1. Clinical Symptoms.

The first sign of the disease is stiffness of the fore-legs, but there are exceptional cases which begin immediately with paralysis, the gait of the animal is slow and stiff, the fore-legs being carefully put on the ground and the step being shorter than normal, especially in its posterior half. In some cases the elbows are turned outwards and in others the legs are crossed in walking. As a rule the animals avoid moving, and prefer to lie on the ground.

In many instances this stiffness is the only symptom of the disease, and it is not yet certain whether such cases have to be considered as cases of lamziekte or of stiffziekte.

The cause of this phenomenon in both fore and hind legs may be:

1. Nervous (central or peripheral).
2. In the muscles.
3. In the bones (marrow, substantia spongiosa).
4. In the articulations (inflammation).
5. In the laminae of the hoof (laminitis, supposed only to be present in stiffziekte).

In the majority of cases the most striking symptom which follows or accompanies the stiffness is the paralysis of the hinder part of the body. In some patients, however, a stiffness of the hind-legs is antecedent to the paralysis. The animal walks very slowly, crossing the hind-legs, and placing them further under the body when standing still. It lies very often and rises clumsily.

In contradistinction to the above-mentioned cases, others have been observed; here no stiffness, either of fore or hind legs, occurred, and the disease began suddenly with paralysis. In such a case the animal drops at once while moving, and is unable to stand, or an animal apparently healthy in the evening is found on the following morning to be down and unable to rise.

The animals are found lying on the ground as if resting, the hind-legs being placed to one side, and the sternum resting on the fore-legs. They are unable to rise or to stand when lifted; sometimes they rise to the fore-quarters, but the hind-quarters cannot be moved. The latter are either completely motionless and without sensibility, so that pinching or stabbing with a knife point does not produce a reflex movement either of the cutaneous or of the skeletal muscles, or they can be moved at will, the legs themselves being unaffected, and the lesion being in the sacral or lumbar regions.

In very severe cases and in all those which end in death, there is before death a complete paralysis including also the fore-quarters. The animal is not even able to keep itself on the sternum, it lies stretched out and the legs are-
motionless or stiff, or may show spasmodic extensions. Sometimes also the dorsal muscles of the neck are contracted, so that the head is stretched straight out or pulled backwards and stiff, and when forcibly bent forwards it returns immediately to the original position.

These latter symptoms are evidently signs of nervous (motor) excitement, and are probably caused by the meningitis, showing that meningitis follows the intestinal affection (see later).

These two different classes of symptoms, excitement and paralysis, may have the following causal (anatomical) origin:—

(1) Both may be cerebral.
(2) Both may be spinal.
(3) One may be cerebral and the other spinal.

The paralysis of the hind-quarters is probably of a spinal (lumbar) origin, whilst the excitation of motor nerves may be caused by inflammation of the brain.

The general appearance of the animal is not, or only slightly, altered in the first stage of the disease, i.e. that of stiffness. The head is free, the sensibility normal, the nose moist, and the eyes normal; in most instances the back is arched.

Once the animal is paralysed, the general appearance is altered; the animal refuses food, appears dull and uninterested, and takes no notice of the settling of flies. The eyelids are swollen and conjunctiva is injected; signs of pain may be observed, such as grinding of the teeth, and looking backwards towards the abdomen.

Some cases are known where the general appearance of the paralysed animal was not altered until the day preceding death and sometimes not even then. They were eating and drinking to the last moment and no trouble of the sensorium could be noticed in spite of the affection of the brain which was revealed by post-mortem.

The alterations in the general appearance do not always adequately express the affection of the brain.

The temperature as a rule is not abnormally high, and lamziekte is not a febrile disease. In cases of complete paralysis (or usually twenty-four hours before death), the temperature is even sub-normal, 95° F, or less. The temperatures observed in various stages of lamziekte acquired under natural conditions, vary from 95° to 105° F. In one case of paralysis, however, a temperature of 109° was observed a few hours before death.

The temperatures of the herd of cattle running under natural conditions were found to oscillate between 95° and 105°, 95° being exceptional. A temperature of 101° was noticed comparatively often. It has frequently been observed that even in one and the same animal variations of the temperature amounting to 6° or 8° may occur within forty-eight hours, and also during many days in succession.

Another animal, which after death showed anatomical lesions of lamziekte, showed during life no symptoms of this disease except fever.

As a consequence of being recumbent for a long period, decubitis makes its appearance in paralysed animals, especially on the sternum, shoulders, and hips. First the hair falls off, then the skin and subcutaneous tissue become necrotic, and subsequently infection produces a suppurating wound.

The heart, as anatomical examination shows, may be affected; the pulse is therefore altered. The number of pulsations observed in the different stages of lamziekte in several cattle varied from 40 to 126 a minute.
In all cases but one, where clinically a frequent pulse was observed, an affection of the heart (epicarditis or endocarditis) was found at the post-mortem examination.

The blood seems not to be affected; it is normal in colour and coagulates normally.

As the lungs show the state of oedema at the autopsy, alterations in respiration are naturally seen during life—at least, shortly before death. The number of respirations per minute is sometimes as much as 120 (climatic influences to be taken into consideration). Respiration is as a rule regular, but irregular and jerky respiration has been observed, especially shortly before death. The nose may be either moist or dry, variations in this respect being sometimes seen in the same animal.

In most cases there is discharge from the nostrils, either serous and colourless or mucous, or mucous and frothy, and sometimes there is even a white froth escaping from both nostrils. This latter symptom is a consequence of the oedema of the lungs, while the former symptoms are due to the inflammation of the mucous membrane of the nose (catarrhal).

I have also observed patients, the respirations of which were accompanied by a groaning noise, caused either by a collection of froth from the lungs in the larynx, or by a paralysis of the soft palate. The tongue and muscles of mastication of these animals were paralysed, and there was abundant salivation.

The symptoms in connection with the digestive organs are the following: The appetite is reduced in the majority of cases according to the views of farmers. Especially when the animal is paralysed and death is approaching food is refused, even when there is no paralysis of the tongue or pharynx. The animal is so dull and comatose that it takes no notice of food placed before it, and even when the food is placed directly into the mouth it does not masticate at all, or only slowly and interruptedly.

In other instances the animal eats until shortly before death, and it may happen that the desire to eat is present in spite of inability to masticate or swallow, on account of paralysis of the tongue and pharynx. In these cases the animal eats and masticates, but cannot swallow properly, and therefore long strings of badly masticated forage can be pulled out of the mouth and oesophagus. I have seen cattle lying on their sides with the head stretched out on account of spasm of the neck muscles, and yet these animals were seeking food and trying to get hold of it by stretching the head and tongue as far as possible.

The appetite depends upon the intensity of the cerebral affection, and therefore, is no absolute indication of the degree of gastro-enteritis.

The mucosa of the mouth is sometimes pinkish and hot, and the mouth contains a considerable amount of saliva which may also be running from the mouth. This may be due to

(a) an inflammatory super-production; or
(b) interference with the act of deglutition (tongue evidently paralysed in a few cases); or
(c) both of these together.

Increase of salivation has been noticed even when the pharynx was not paralysed and the animal was feeding, but also when the pharynx was paralysed.

The process of mastication, sometimes normal and sometimes very slow, has in several instances been noticed to be irregular and accompanied by pronounced grinding of the teeth, perhaps owing to defective conformation.

In other cases the paralysis extends to the tongue and muscles of mastication, so that if the mouth is open and the tongue pulled out, it remains hanging with the lower jaw drooping.
Rumination, like the appetite, seems to persist in certain cases, even when the animal is paralysed, until shortly before death, but in other cases it is interrupted.

The movement of the intestines, i.e. rumen and small and large intestines, seems not to be altered greatly in the stage of stiffness. During the paralytic stage these organs may be either normally active or paralysed. Tympanitis is seldom present, but when it occurs it indicates paralysis of the rumen, and usually the ordinary contractions are slow and cannot be heard or felt. Less evident intestinal regurgitations are not necessarily accompanied by constipation, which would be a sign of paralysis of the large intestines. These sounds may be diminished although defaecation occurs normally.

In the majority of cases the intestines act in a normal manner until the end, but in other instances diminished sounds in the abdomen, and less frequent contractions were noticeable in the paralytic stage, or on the other hand, they were absolutely normal during the first few days of paralysis, and became slower and less distinct when death approached.

The activity of the bowels and the intensity of the regurgitations are, therefore, not of prognostic value in either direction, although the intestines are undoubtedly affected in every instance.

Defaecation is usually practically normal and the faeces are as a rule very little altered, but in a few cases there was a real constipation or a distinct diarrhoea, and in one of the latter cases blood corpuscles were seen in the faeces microscopically.

In two instances, both of which ended fatally, the faeces were covered with flakes and strings of white mucus, although otherwise normal.

Nothing pathological has been reported regarding the kidneys, nor did I myself ever meet with clinical symptoms pointing to any abnormal condition of the kidneys themselves or of the urine.

Among the sexual organs only the udder is affected, showing a sudden remarkable decrease in milk production, a phenomenon which some farmers consider to be an infallible symptom indicating the beginning of lamziekte.

The symptoms due to the affection of the nervous system, and especially of the brain, may be divided into two groups:

(1) Symptoms of excitation.
(2) Symptoms of depression, including paralysis.

The former symptoms (exciting) may be absent, but the depression stage has never been found to be absent. It not rarely occurs that both classes of symptoms occur simultaneously in one and the same animal.

Among the symptoms of excitation are:
(a) Cramps of the hind legs.
(b) Spasmodic twitchings of the muscles of mastication.
(c) Tetanic contractions of the extensor muscles of the neck.
(d) Running round in circles (mal lamziekte).

The symptoms of depression are the following:
(a) Dullness and indifference.
(b) Diminished sensibility of the skin.
(c) Irregular and abnormal feeding.
(d) Paralysis of the pharynx and tongue, i.e. inability to swallow, and the entire musculature, the digestive tract (tympanitis, decreased regurgitation, constipation).
(e) Paralysis of the skeletal muscles of both fore and hind legs.

Complete paralysis of the limbs combined with stiffness of the neck may often be observed, and such cases are sometimes accompanied by rapid spasmodic masticatory movements.
What farmers call mal lamziekte is probably an indication of acute meningitis, and would therefore be a typical symptom of cerebral irritation. This form, which is seldom observed and which I personally never had an opportunity of investigating, is indicated by the peculiar behaviour of the animal, e.g. running round in circles, and always in the same direction.

I leave it an open question as to whether it is justifiable to class the paralysis of the hind-quarters with the symptoms of cerebral depression. The real cause of the paralysis may be in the spinal cord, and in favour of this conception is the fact that an animal may be completely paralysed in the hind-quarters without showing any signs of meningitis.

Course of the disease and termination.

Considering the succession of the symptoms, the disease might be divided into two stages.

(1) The stage of stiffness.

(2) The stage of paralysis.

This division, however, is merely clinical and supported by neither anatomical nor physiological facts.

According to farmers, the disease usually begins with stiffness in the shoulder and fore-legs, and accordingly with an irregular and uncertain gait; sometimes the hind-legs are also stiff. The animals lie down and do not stand; in fact they are unable to rise as paralysis of the hind-quarters has set in.

After this the majority of patients show, sooner or later, symptoms affecting the sensibility, namely dullness, indifference, and loss of appetite.

The duration of the stiffness ranges up to fourteen days, and the paralytic stage lasts as long as twenty days.

In fatal cases a state of complete paralysis lasting for a few hours, or even a few days, precedes death.

The following table shows the length of the various stages of lamziekte.

<table>
<thead>
<tr>
<th>Date of Post-mortem</th>
<th>Owner</th>
<th>Animal</th>
<th>Stiffness</th>
<th>Paralysis</th>
<th>Exitus</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 16/12/08</td>
<td>L. R.</td>
<td>Cow</td>
<td>Days</td>
<td>Days, About 90</td>
<td>Recovered</td>
</tr>
<tr>
<td>2 19/12/08</td>
<td>G. F. M.</td>
<td>Cow with calf</td>
<td>4</td>
<td>2</td>
<td>Died</td>
</tr>
<tr>
<td>3 21/12/08</td>
<td>F. V.</td>
<td>Heifer</td>
<td>0</td>
<td>1</td>
<td>Killed</td>
</tr>
<tr>
<td>4 24/12/08</td>
<td>K. V.</td>
<td>Cow</td>
<td>2</td>
<td>20</td>
<td>Died</td>
</tr>
<tr>
<td>5 27/12/08</td>
<td>S. A.</td>
<td>&quot;</td>
<td>7</td>
<td>&quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>6 28/12/08</td>
<td>J. B.</td>
<td>&quot;</td>
<td>5</td>
<td>3</td>
<td>&quot;</td>
</tr>
<tr>
<td>7 31/12/08</td>
<td>J. N. H.</td>
<td>&quot;</td>
<td>0</td>
<td>1</td>
<td>&quot;</td>
</tr>
<tr>
<td>8 3/1/09</td>
<td>J. N. H.</td>
<td>&quot;</td>
<td>4</td>
<td>1</td>
<td>&quot;</td>
</tr>
<tr>
<td>9 8/1/09</td>
<td>J. N. H.</td>
<td>Ox</td>
<td>1</td>
<td>6</td>
<td>&quot;</td>
</tr>
<tr>
<td>10 10/1/09</td>
<td>A. P. V.</td>
<td>&quot;</td>
<td>14</td>
<td>8</td>
<td>&quot;</td>
</tr>
<tr>
<td>11 11/1/09</td>
<td>F.</td>
<td>Cow</td>
<td>1</td>
<td>3</td>
<td>&quot;</td>
</tr>
<tr>
<td>12 14/1/09</td>
<td>Station</td>
<td>Cow with calf (15)</td>
<td>0</td>
<td>5</td>
<td>&quot;</td>
</tr>
<tr>
<td>13 14/1/09</td>
<td>&quot;</td>
<td>Calf (15)</td>
<td>0</td>
<td>0</td>
<td>&quot;</td>
</tr>
<tr>
<td>14 25/1/09</td>
<td>&quot;</td>
<td>Calf with calf (17)</td>
<td>0</td>
<td>7</td>
<td>&quot;</td>
</tr>
<tr>
<td>15 25/1/09</td>
<td>&quot;</td>
<td>Calf (17)</td>
<td>0</td>
<td>1</td>
<td>&quot;</td>
</tr>
<tr>
<td>16 25/1/09</td>
<td>&quot;</td>
<td>Cow (87)</td>
<td>0</td>
<td>10</td>
<td>&quot;</td>
</tr>
<tr>
<td>17 14/2/09</td>
<td>&quot;</td>
<td>Cow with calf (9)</td>
<td>0</td>
<td>3</td>
<td>&quot;</td>
</tr>
<tr>
<td>18 21/2/09</td>
<td>&quot;</td>
<td>Calf (22)</td>
<td>0</td>
<td>1</td>
<td>&quot;</td>
</tr>
<tr>
<td>19 27/2/09</td>
<td>&quot;</td>
<td>Cow with calf (24)</td>
<td>0</td>
<td>2</td>
<td>&quot;</td>
</tr>
<tr>
<td>20 27/3/09</td>
<td>Native S. V.</td>
<td>Cow</td>
<td>0</td>
<td>3</td>
<td>Killed</td>
</tr>
<tr>
<td>21 5/4/09</td>
<td>Station</td>
<td>Cow with calf (22)</td>
<td>8</td>
<td>1</td>
<td>Died</td>
</tr>
<tr>
<td>22 13/4/09</td>
<td>&quot;</td>
<td>Bull (298)</td>
<td>0</td>
<td>1</td>
<td>&quot;</td>
</tr>
</tbody>
</table>
The experience of farmers that the sooner the paralysis sets in the sooner the animal dies, is confirmed by a few cases mentioned in this table.

The mortality is rather high. Of thirty-six cases which I had the opportunity to observe, only 8, or 22 per cent., recovered. The mortality, therefore, is about 80 per cent.

From the already mentioned eight animals, two showed paralysis, and the remaining six were only stiff in the fore-legs.

We may, therefore, conclude that once an animal is paralysed, the chances of recovery are very small.

The prognosis in lamziekte is bad, as, according to farmers, cattle which have passed through an attack of lamziekte are more susceptible to the disease than those which have never suffered from it, and among such cattle the mortality is higher.

It is said that females, and especially cows in calf and milch cows suffer more frequently than bulls and oxen, and my own experience is in accordance with this view. Among the above-mentioned thirty-six cases there were twenty-four cows, i.e. 67 per cent.

2. The Anatomical-Pathological Lesions.

(1) General Appearance.—As all animals of which post-mortem examinations were made had been suffering from the disease for a longer or shorter period and were invariably more or less paralysed, and as the appetite in most cases of lamziekte is diminished, the carcases appear in various conditions. Of twenty-one animals on which I performed autopsies, about half were in poor condition, while the condition of the others was fairly good. Paralysis and a subsequent lying on the ground cause a more or less extensive oedematous, colourless, or yellowish infiltration of the subcutaneous connective tissue in the region of the shoulders, sternum, and hind-quarters. This phenomenon I observed in twelve instances, four of which were accompanied by haemorrhagic infiltration of the tissues. The distinct subcutaneous exudation is not always caused by the animal lying down for a long time, and two cases of such an oedema and haemorrhage have been recorded where the patients were only paralysed for one day.

On the other hand these abnormalities were absent in animals which were paralysed for a comparatively long period, and it must therefore be considered that these phenomena may have other causes than decubitis, such as affections of the heart, or the action of toxine.

Tympanitis has been observed, sometimes arising in a surprisingly short time after death, i.e. with a shorter interval than in healthy animals.

(2) The external lymphatic glands.—These are usually normal, but in a few cases some of them were found to be oedematous (prescapular and inguinal glands).

(3) The circulatory system.—The blood shows no alterations either to the naked eye or microscopically (in one or two instances there was poikilocytosis). Coagulation is normal. In cases of long duration of the disease, where the animal falls off in condition on account of mal-nutrition, the blood appears thin and watery.

The pericardial membrane is usually unaltered, and only in three instances was an oedematous infiltration, and in one instance, ecchymosis noticed. Nevertheless, the amount of liquid in the pericardium has been found to be increased in about 50 per cent. of the cases examined, and in one-half of these cases the liquid was of a reddish colour, due to post-mortem decomposition and haemolysis.
In one instance the exudate coagulated, and in another case a flocculent coagulum was found floating in the liquid. The cause of the increase of the pericardial liquid appears in the epicardium, for except in one instance, epicarditis was present in all cases in which the quantity of liquid was greater than normal. This epicarditis was shown by the presence of echymoses and petechiae, and in four instances by sub-epicardial oedema along the coronary and longitudinal sulci.

The myocardium was in two cases of a flabby consistence, and showed in one instance haemorrhagic spots corresponding with similar alterations in the epicardium.

The endocardium was altered in one-third of the cases, showing echymoses and petechiae. It was especially the left endocardium which showed haemorrhagic infiltrations, and in every instance epicarditis was also observed. The right endocardium was very often of a diffuse dark red colour, caused by the absorption of haemoglobin (post-mortem, haemolysis and decomposition of haemoglobin); this discolouration might mask an inflammatory alteration preceding death.

The large vessels were never found to be affected.

4. Respiratory system.—In the description of the clinical appearances of lamziekte, a nasal discharge has been mentioned, and the cause of this phenomenon, excluding the abundance of frothy discharge sometimes seen, consists in an inflammation of the mucous membrane of the conchal cartilage, septum nasi, and, in some cases, of the cribriform bone. In five instances I found the mucosa concharum inflamed and inflammation was seen in one case on the cribriform bone, and also in one case on the septum nasi. In these cases the inflamed mucous membrane was of a deep red colour with strings and flakes of mucus on its surface.

The mucous membrane of the trachea and bronchi was normal, but in most cases these channels contained white froth due to pulmonary oedema. Pulmonary oedema was present in more than two-thirds of the cases examined post-mortem, but in most cases it occurred only during the last stages preceding death and was not specific.

In about 50 per cent. of cases it was accompanied by a very pronounced hyperaemia of the pulmonary lobules, and I am inclined to think that this also usually develops when the animal is in extremis.

I met with one case of a diffuse necrotic pneumonia due to a foreign body; in this case medicine had been administered after paralysis of the tongue and pharynx had set in.

Fibrous thickenings of both the costal and pulmonary pleura were not rare, but were merely accidental and had no connection with lamziekte.

Oedematous infiltration of the mediastinum was rare. The mediastinal glands were often affected, being either oedematous or congested; in some cases they were both oedematous and congested.

5. Digestive Tract.—The digestive organs were always affected. As a consequence of the paralysis of the muscles of mastication and deglutition, portions of badly masticated forage could be found in the mouth, pharynx, and in one case also in the oesophagus.

In about 25 per cent. of the cases lesions of an angina were present, and in such cases the tissue surrounding the tonsils was swollen, oedematous, and even of a gelatinous consistency. The tonsils were usually congested, enlarged, and infiltrated. In two cases hyperaemia of the soft palate was noticed, and in one case lymphatic infiltration of the interstitial tissue of the parotid gland.
The first three compartments of the stomach are usually normal. The rumen was more or less full, according to the appetite and ability to swallow, and the contents were generally fairly moist. In three instances reddish patches were found in the sub-mucous tissue. Amphistoma were frequently present, in perhaps 50 per cent. of cases.

The mucosa of the reticulum is usually normal. The contents were dry, and in rare cases a patchy hyperaemia of the sub-mucous tissue was noticeable.

The mucous membrane of the omasum showed no pathological changes, but in a few cases the sub-mucous tissue was injected.

The abomasum showed constantly the typical lesions of lamziekte in the form of a serous or haemorrhagic gastritis, and this gastritis was absent only in two cases. In about 33 per cent. of cases there was a more or less distinct congestion of the mucosa; this congestion was at first diffuse and as a rule more pronounced on the folds of the mucosa; in some instances it was punctate, and in other cases appeared in the form of large patches, or of strips following the folds of the mucosa. The diffusely reddened mucosa was sometimes spotted with ecchymoses.

More typical of lamziekte were the erosions seen in 70 per cent. of cases and almost constantly accompanied by congestion (in some cases by haemorrhagic spots and streaks). Erosions without accompanying inflammatory lesions were rarely met with.

These erosions varied in diameter from 4 to 25 m.m.; when small they were more or less circular in shape, but the larger erosions were usually elongated and irregular. In most instances they were a dark red haemorrhagic colour. The margins of the erosions were sometimes slightly elevated, but in most cases this was not seen. Except in a very few cases they penetrated deeper than the mucosa; they were generally more frequent in the region of the pylorus, and were usually situated on the folds.

Swelling and oedema of the mucosa was observed in three cases.

The contents of abomasum were usually of normal consistence, but in a few instances with thick, dry, and of a brown or greenish colour.

The small intestine was in almost all cases inflamed, the lesion being of the nature of a simple catarrhal or haemorrhagic enteritis with nodules and erosions.

The mucosa of the duodenum showed various degrees of congestion in the form of petechiae, ecchymoses, large patches, transverse or "zebra" streaks, or longitudinal streaks; in some cases there was diffuse congestion. In about 30 per cent. of cases the mucosa was swollen and oedematous.

In rare cases yellow or greenish discolouration of the mucosa, erosions similar to those occurring in the abomasum, nodules, and a mucous exudate were found.

The mucous membrane of the jejunum showed similar but more advanced and extensive lesions. Sometimes there are small nodules the size of peas, with a dry white centre and a red or bluish periphery; these nodules occur more frequently than in the duodenum, but even here are comparatively rare.

The lesions in the ileum resemble those of the duodenum, but swelling of the mucosa is more frequent and the nodules are more frequently found and occur in greater numbers.

The contents of the small intestine are in about 25 per cent. of cases greatly altered, being of mucous consistence and of a whitish grey or reddish colour; in later stages they may be slate coloured.

The large intestines show lesions similar to those of the small intestines; the caecum and colon were found to be affected in about 50 per cent. of the
cases examined. They showed diffuse or patchy congestion, haemorrhagic streaks and ecchymoses. Swelling of the mucosa and the formation of nodules are seldom noticeable.

Sometimes only the first portion of the colon shows lesions. In one case only were ulcers noted in the anterior part of the colon, and in another case white strings of mucous.

No pathological alterations were noted in the rectum.

The mesenteric glands were affected in the majority of cases, being either congested or oedematous. In a few instances they were highly pigmented, being black or slate coloured.

6. Liver.—The liver as a rule showed no lesions, congestion of this organ being rare. In the opinion of farmers distention of the gall-bladder is typical of lamziekte, but this is only seen in a little more than 50 per cent. of cases. The mucosa is rarely affected, but in one instance I found it to show oedematous infiltration, and in two other cases ecchymoses were noted. In some cases the bile is dark green in colour, turbid, and thick, but in the great majority of cases it was clear, slightly viscid, and of a greenish or yellowish green colour.

7. Spleen.—The spleen was generally normal, but very exceptionally, congestion was seen.

8. Urinary Organs.—Lesions of the kidneys were exceptional, but in a few cases hyperaemia was seen. In a small number of cases lesions were noted in the mucous membrane of the bladder; these were diffuse congestion, injection of the vessels, and petechiae.

9. Nervous system.—In about 70 per cent. of cases, the brain showed lesions of leptomeningitis. This inflammation was usually localized to the pia mater of the nasal portions of both hemispheres, being sometimes more pronounced on one side. It was also seen in the fossa of sylvius and the temporal lobe of one hemisphere, and in some instances was generalized, extending over the entire pia mater. The lesions consisted of an intense injection of the vessels with more or less intense grey discolouration and oedematous infiltration, and subsequently the pia showed a muddy or turbid appearance, this being especially distinct in young animals. In some cases a whitish thickening of the membrane, occurring especially along the course of the vessels, was seen together with the above lesions. The grey and white substance of the brain showed no abnormalities.

In many cases a reddish turbid liquid, varying in quantity up to 20 c.c., was found in the subdural space.

The spinal cord showed no pathological changes. These were especially looked for in the lumbar portion of the cord, but without success.

10. Muscles and connective tissue.—To the naked eye the muscles appear normal, but the intermuscular tissue was altered in about 33 per cent. of cases, showing oedematous infiltration and a jelly-like consistence. These changes were more frequent in the hind-quarters, but were also found in the intermuscular tissue of the sternal and subscapular regions.

11. Bones and articulations.—Nothing abnormal was ever observed in the periostium. The cancellated tissue of the epiphyses of the long bone showed in about 25 per cent. of cases red patches. In a certain proportion of cases the bone marrow had a watery appearance, but in these cases, with one exception, the animals were either old or in poor condition. In some cases reddish spots and patches were observed on the surface of the marrow, especially towards the end of the bone. The articulations were always normal, both in fore and hind legs.
In conclusion, we may say that the chief lesions of lamziekte are:

1. Haemorrhagic or ulcerative gastritis.
2. Catarrhal enteritis, with haemorrhage and ulcerations.
3. Exudative leptomeningitis.

These may be accompanied by oedema of the lungs, angina of the pharynx, epicarditis, and endocarditis, and osteomyelitis.

Immunity.

An attack of lamziekte seems not to confer any immunity on an animal, in fact the animal shows a greater liability to contract the disease the second time. Cases are known that cattle have acquired the disease even three of four times in successive years, and other cases have occurred where an animal was attacked twice in the same year.

Comparison with this Disease of the Lamziekte occurring in Cape Colony.

The descriptions of lamziekte given by D. Hutcheon (Cape Agricultural Journal, July, 1903, and April, 1904); W. Robertson (Cape Agricultural Journal, September, 1907); and J. Spreull (Cape Agricultural Journal, March and May, 1908), do not agree, nor do they correspond with my own experience.

I have never seen the “highly congested condition of the bones with softening of their cancellated tissue, the texture of the bone tissue being filled with a dark red gelatinous exudation” described by Hutcheon; nor have I seen “the muscles sodden as if soaked in some fluid” described by Robertson. As I have pointed out, it is especially the jejunum which, along with the abomasum, shows the most intense lesions. According to Robertson’s description, referring to the eastern provinces of Cape Colony, the caecum is the organ particularly affected.

Robertson describes a chronic or anaemic form, distinguished clinically by loss of appetite, intense chronic diarrhoea, pronounced emaciation, almost white flabby muscles impregnated with liquid, liquid in the pleural and peritoneal cavities, gastro-enteritis and abnormal quantity of synovia in the joints. This according to the reports of farmers and my own observations, is not present in the Lichtenburg District, and the oedematous or throat form and the thoracic form (Spreull) are not seen. The descriptions given of the paralytic form correspond fairly closely with my own observations, but there are a few discrepancies with regard to the post-mortem appearances. For instance, pulmonary hepatization, congestion of the liver, and haemorrhages in the renal cortex are not seen in the lamziekte of Lichtenburg.

Robertson and Spreull describe a bipolar staining organism as the cause of lamziekte. Amongst the bacteria which I was able to cultivate from cerebral exudates, and the mesenteric glands of several naturally contracted cases, I was never able to find a pasteurella. On the other hand, I was able to produce in cattle clinical and anatomical lesions similar to those caused by the above-mentioned bipolar organism, by intrajugular injection of several bacilli which I had isolated from cases.