KRIMPSIEKTE.

By M. W. HENNING, M.R.C.V.S., Transvaal University College, Pretoria, late Research Officer, Onderstepoort.

THERE appears annually among animals in the most arid parts of the Cape Province a disease which is characterized by symptoms indicating an affection of the muscular and nervous systems. On account of the spasmodic muscular contractions, torticollis, and other nervo-muscular symptoms, this disease has appropriately received the popular name of " Krimpsiekte."

HISTORICAL.

Under different names, such as " nenta," " tnenta," " cnenta," " cerebro-spinal meningitis," etc., Krimpsiekte has been reported from various parts of the Cape Province for many years. The first official notice of the prevalence of the disease is to be found in the Report of the Cattle Diseases Commission (1877) (1), where it is stated that " there is a disease called rita (cnenta) which is very destructive to goats. It affects them in a way that seems to indicate paralysis of the central nervous system." Macowan (1) considered the condition to be a plant intoxication caused by the ingestion of a legume, and is quoted in the above report as follows: " A small leguminous plant, *Lessertia annularis* Burch, which in Karroo and ' gebroken-veld ' is sometimes very abundant, and acts as a poison upon sheep and goats, resembling the results reported of *Gastrolabium* R.Br. and some of its congeners in Western Australia. It seems to produce cerebro-spinal paralysis, by which the animal is unable to co-ordinate the movements of its several limbs. The head wags helplessly, and in severe cases death speedily ensues." A further reference to the disease is made by Macowan (2) in his article on the stock food-plants of the Cape. Dr. Brown (3) also mentions the *Lessertia annularis* as the probable cause of nenta.

The first reference to the disease by Hutcheon (4) appeared in 1882, when he reported on his investigations into the nature of nenta in the Jansenville District. In this report he states that farmers attribute the condition to plant-poisoning, but that there is great diversity of opinion as to the actual causal plant. At this time Hutcheon fed various suspected plants to goats, with negative results in every case; but he claimed to have successfully reproduced the disease in a dog by feeding the animal on livers of goats that had died from nenta.

In a later report Hutcheon (5) quotes the following from a letter written by Macowan in connexion with nenta: " The peculiar cerebral disorder referred to by the veterinary surgeon is due to quite another plant, viz., the *Lessertia annularis* Burch, a prostrate leguminous perennial. . . . This is the cnenta or tnenta, and

I have satisfied myself by examining the contents of the stomachs of animals dying from the peculiar diseases referred to that it is the direct cause of the mischief. The symptoms of Lessertia or tnenta poisoning are analogous to those observed in almost all cases of poisoning by other Leguminoseae." Hutcheon does not agree with this, and in the same report writes: "I have been unable to confirm that opinion, however. On the farm Bommidale, where I have principally studied the disease, I was unable to find this plant, and Mr. Berrington informed me that he found only three specimens of the plant on the whole farm. When we associate that with the fact that on that farm over a thousand goats were affected with the peculiar disease out of a flock of two thousand five hundred during this cnenta season, we are compelled to attribute the cause to something else."

Hutcheon (7) was the first investigator who succeeded in reproducing nenta experimentally. He fed the liver of a nenta goat to a dog, with the result that the animal developed acute symptoms of nenta after two days. He also drenched two healthy goats with the strained stomach-contents of diseased goats, with the result that both goats developed symptoms of nenta on the second day.

Soga (6) gives Mr. P. Weijer, of Darlington, Somerset East, the credit for first attracting attention to the true causal plant. In order to verify Weijer's observations, Soga personally commenced a series of experiments on the farm Darlington in 1890. He fed each of eight goats, obtained from nenta-free veld, on a daily ration of 2 oz. of the suspected plant for a period varying from three to five days. All the goats developed symptoms of nenta, and finally six died, the remaining two recovering from the disease. The plants were identified as *Cotyledon ventricosa* Burm. by Macowan, who expressed surprise that a crassulaceous plant should contain such active properties as to produce the nervous symptoms of nenta, and stated his doubt of the evidence upon which this plant was convicted. Veterinary Surgeon Tomlinson was, therefore, detailed to carry out feeding experiments with the Lessertia and other legumes found in veld where nenta was prevalent. The results were all negative (7), and Tomlinson reported as follows: "I have carefully examined the parts where the disease is most severe, but I have not been able to find any of the leguminous plants I have experimented with at Uitkomst; but with regard to the cotyledonous plant, it was very plentiful."

Soon after this Tomlinson obtained six goats from a farm in Sutherland (Cape Province), where no case of nenta had ever appeared, and fed them in the village on the plant *Cotyledon ventricosa*, with the result that all became affected and two died.

Veterinary Surgeon Dixon obtained similar results by feeding goats in the Victoria West District on a plant which he called *Cotyledon ventricosa*.

Species identification was, however, apparently not botanically controlled in either Tomlinson's experiments in Sutherland or Dixon's experiments in Victoria West.

At about the same time, Veterinary Surgeon Borthwick, conducting a series of experiments in the town of Somerset East, which is far removed from any locality where nenta was known to exist, was able to reproduce nenta in goats by feeding *C. ventricosa* obtained from Mr. Weijer at Darlington.

In concluding his report, Hutcheon (7) states: " The results of these experiments leave little doubt that the plant *C. ventricosa* is at least one of the plants which induce the disease known as nenta or krimpsiekte in goats and sheep. . . . But the best proof that this plant is the principal, if not the sole, cause of nenta is the fact that, on places where the disease was previously contracted by goats in a severe form, after this plant was removed, no further cases occurred."

In 1912 Kehoe (8) fed *Cotyledon orbiculata* to fowls and goats. Of the sixteen fowls fed on material from different parts of the country, five developed symptoms of poisoning, of which two died. Apparently the goats remained healthy. He concludes by stating that " the chief symptoms produced as a result of the toxic effects of the plant appear to be an affection of the central nervous system."

A disease that has erroneously been termed " *nenta in Australia* " (14) was investigated by Martin, and found to result from an intoxication following the ingestion of a leguminous shrublet, the Darling pea (*Swansonia galegifolia* R. Br.). Very strong views (14) were held in the Cape on the analogy between this disease and the condition nenta as it occurred in the Cape. If, however, the conditions under which the two diseases appear and the cause and symptoms be studied, there will be found no relationship whatsoever.

Another Australian disease, known as " staggers and shivers in live stock " (15), and investigated by Dodd and Henri, may be considered somewhat like krimpsiekte, but in symptomatology only. It has been reproduced in stock by feeding *Malva parviflora* and *Babium amplexicaula*.

The American disease, " trembles or milk-sickness," (16) brought about by feeding cattle on white snake-root (*Eupatorium urticaefolium*), resembles krimpsiekte or nenta in so far as the symptoms are of a nervous and muscular nature and are brought out by violent exercise.

A form of krimpsiekte (17) was produced by Otto Henning in 1893 by feeding bovines on a plant known as the " klimop " or " baviaanstouw " (*Cynanchum capense*). It resembles, however, the condition under review only in so far as it develops symptoms of a muscular and nervous nature, but differs so distinctly in all other respects that it cannot possibly give rise to confusion to the careful observer.

Many other nervous affections exist which resemble krimpsiekte in some form or other, but the peculiar conditions under which krimpsiekte occurs, and the specific symptoms which appear, render its diagnosis easy.

The Disease under Natural Conditions.

Distribution.—In 1921 the writer personally investigated several outbreaks and observed cases of krimpsiekte in the Ceres and Tangua Karroo, in the Districts of Sutherland, Calvinia, and Worcester (Matjesfontein). Outbreaks of the disease have been reported in Prince Albert, Beaufort West, Carnarvon, and western parts of the great Karroo. The condition known as nenta, and described by Hutcheon, appears more particularly on the lower and western slopes of the Zuurberg, near Lake Mentz, the western parts of Somerset East, and other parts of the eastern Karroo.

Conditions under which the Disease occurs.—Animals grazing on ridges, hill-tops, or slopes in affected areas are liable to contract the disease; a common feature on all the farms where the presence of krimpsiekte has definitely been established being the occurrence of a number of cotyledonous plants which generally grow on some hilly place or ridge in arid country.

The connexion between the disease and the presence of certain cotyledons has frequently been suspected, and many observant farmers have even pointed to a particular plant, *Cotyledon wallichii*, as the probable cause of the disease; others, again, have blamed either one or several of the following plants: *C. reticulata*, *C. paniculata*, *C. orbiculata*, *Asparagus stipulaceus*, *Euphorbia mauritanica*, and others. A few have even considered some unknown parasite to be the most probable causal agent. It is, however, generally agreed that, if the stock are removed from a badly affected area to some new locality, such as the "koue bokkeveld" or "roggeveld," where the *Cotyledon wallichii* does not grow or is only seldom found, no fresh cases of the disease appear.

Annual Incidence and Seasonal Variation.—Krimpsiekte occurs annually in the Karroo during spring and early summer, but disappears as soon as farmers move their stock to summer grazing, like the "koue bokkeveld" and "roggeveld," where the disease is not prevalent. The disease will, however, continually appear in stock kept in the Karroo during the summer. But if there have been good rains and the veld is consequently good, severe losses from krimpsiekte are sustained only in exceptional cases.

Climatic Conditions.—The disease appears only in the most arid regions of the Cape Province during spring and early summer. The smaller the rainfall is the more prevalent the disease becomes. In neighbouring areas where the altitude is higher and the climate cooler and less arid, conditions appear to be less favourable to krimpsiekte.

Animals Affected.—All the domesticated animals are susceptible to the disease, but, as the conditions in krimpsiekte veld are suitable practically only for goat-farming, this species is the most frequent sufferer, and the disease is consequently often erroneously regarded as specifically one of the goat. If, however, sheep are grazed on krimpsiekte veld, the mortality may even exceed that of goats. Cattle and horses are seldom exposed to "infection" and consequently seldom contract the disease, but krimpsiekte is by no means uncommon in these animals. Cases of krimpsiekte are often reported in dogs fed on the meat, blood, or entrails from diseased carcasses. Even man is considered liable to contract the disease if fed on raw or undercooked meat or blood of a krimpsiekte animal. It is, however, maintained that meat properly cooked is safe for human consumption.

Cases of krimpsiekte are also frequently reported in domesticated birds.

Species Susceptibility.—All the domestic animals will contract the disease if kept under suitable conditions, but the dog is apparently the most highly susceptible and succumbs much more easily from its effects than other animals. Birds, too, are very sensitive to the effects of krimpsiekte.

Age and Sex.—There is no doubt that animals of both sexes are equally susceptible to the disease.

Cases of krimpsiekte have been reported in kids confined entirely to kraals and enclosures, before they were noticed to have commenced grazing. It has been suggested that the disease is contracted through the agency of the mother's milk, although the mother may be entirely free from clinical symptoms of krimpsiekte.

In the Karroo, it is customary to confine the kids to enclosures or restricted areas in the vicinity of kraals which are generally constructed on a kopje or on the slope of a hill, where suspicious vegetation is not infrequently found in abundance, while the mothers are herded all over the farm. Often these enclosures are temporary structures made of Karroo bush and shrubbery, amongst which various cotyledons are frequently included. Consequently kids kept in such enclosures must be considered to have access to suspicious vegetation. It would, therefore, appear that the evidence which has led some observers to conclude that krimpsiekte may be contracted by kids before grazing has commenced must not be accepted without reserve. There can, however, be little doubt that kids and young goats are much more subject to the malady than older animals raised on the affected area. This comparative frequency of cases in kids can be explained if their habits and the conditions under which they are kept be considered. It is the rule for a goat-farmer to separate the kids from the ewes during the day and to herd the latter some distance from the kraals, in the vicinity of which the kids are allowed to loiter. As kids have not yet learned to discriminate between different plants and as they are by instinct inclined to nibble at, bite at, or eat nearly any object they come across, they will be liable to take in poisonous as well as non-poisonous plants.

As an example, a severe outbreak of krimpsiekte was reported in kids confined to a shed constructed partly of a plant known as "kandelaar bos," on the removal of which no further cases appeared.

Another outbreak was reported in kids that were allowed to loiter in the kopjes near the homestead. On investigation it was noticed that a quantity of " kandelaar bos " had been partly browsed off by the kids, and a few kids were actually seen nibbling at the plants.

On the other hand, kids allowed to run with the ewes have less leisure to loiter about and contract the disease less frequently.

Comparative Susceptibility of Indigenous and Imported Stock.—Newly introduced stock are apparently much more susceptible to the disease than animals reared on the affected areas, and generally contract a very severe form of the malady. As an example, an outbreak can be mentioned where twenty-nine out of forty-four newly introduced goats contracted a very acute form of krimpsiekte and succumbed within three days after exposure to the dangerous veld, whereas not a single one of a large flock of goats which had been reared on the farm, and with which the imported stock were herded, became affected. Frequently, also, severe outbreaks of krimpsiekte have occurred in small stock moved from clean veld to or over affected areas where they were allowed to graze.

Breed Susceptibility.—In goats, the Angora appears to be the more susceptible breed, and succumbs more readily to the effects of krimpsiekte than the hardy boer-goat. Consequently Angora-goat farming has not become popular in parts of the country where krimpsiekte is very severe.

SYMPTOMATOLOGY.

(a) *Acute or "Opblaas" Krimpsiekte* generally follows when hungry, newly introduced, or strange stock are herded in badly affected veld and frequently during trekking. In the case of goats and sheep, after from one to three or more days' exposure, some of the animals may be found dead without having shown any obvious symptoms of the disease, while others may be visibly sick, dull, and present a miserable appearance. An affected animal either lies down, obviously in severe pain, or may remain standing, and usually shows a variable degree of tympany. The lower jaw frequently droops partly and is moist with saliva. The tongue may protrude partly, and feeding and rumination are suspended. Balls of unchewed or only half-chewed hay are periodically found in the back of the mouth partly occluding the oral passage and probably accounting for the excessive salivation.

At first the animal may be fairly active and run off when approached, but soon it becomes exhausted if chased, goes down, and refuses to stir even if urged with a stick. If tympany is a symptom, regurgitation of the ingesta frequently takes place.

If the animal is left undisturbed for a while, it becomes temporarily refreshed, and will jump up and run off when again approached, but very soon tires. If it is harassed now by being compelled to move, the animal becomes very much distressed, presents a pitiful appearance, and may collapse. Meanwhile respirations have become very much accelerated, irregular, and spasmodic; and the pulse is now fast, weak, and almost imperceptible. Salivation has become even more profuse.

If the animal survives for a day or longer, it may attempt to eat, but on every occasion the first mouthful of hay will collect in the form of a ball at the back of the mouth as before, owing to its loss of control over the muscles of mastication and deglutition, which are in a state of clonic spasm.

The animal, especially if excited, frequently trembles and shivers in parts or all over the body, involving the superficial muscles in particular. Sometimes the patient exhibits a marked degree of hyperaesthesia, and the nervous symptoms become considerably aggravated if the animal is exposed to the sun.

The animal may die soon after the appearance of symptoms or may survive for some days, and in milder cases the condition may even pass over to the chronic form. If it survives for a few days, tympany, when present at the commencement, passes off, and the animal, being unable to eat, becomes empty-bellied, thin, and tucked up, and presents a miserable appearance with deeply sunken eyes. It may stand with an arched back and a drooping and moist lower jaw for many hours on end in a corner or in a shaded spot, avoiding the sun as far as possible. In other cases it may lie down in one spot until it dies. Sometimes the animal may exhibit spasms of acute abdominal pain followed by long-continued dull pain. Whether the course is long or short, death practically invariably supervenes in this form.

In the horse, symptoms generally resemble those given above, but, in addition, there are profuse sweating, more intense abdominal pain, and restlessness. This animal appears to be even more sensitive to the sun and more distinctly hyperaesthetic.

In the dog, no acute form of krimpsiekte has presented itself for observation.

The fowl may be found dead without having shown any symptoms of krimpsiekte, or, if disturbed or driven a short distance, an apparently healthy bird will topple over unable to move farther, struggle for a few yards, and die.

When tympany is associated with this form of the disease, farmers frequently call it " opblaas krimpsiekte."

(b) *Chronic or "Dun" Krimpsiekte* either follows after a protracted acute attack or may be brought about when an animal is exposed to favourable conditions for a fairly long period without developing the acute form. The disease may not appear until months or even years after exposure, or may never distinctly appear in some animals. Frequently, however, animals which are apparently quite healthy when casually observed develop the most typical symptoms if disturbed or exercised. Even when the disease has definitely been established, the most characteristic symptoms are manifested only when the animal is excited and disturbed. Hence it is deemed advisable always to exercise the animal before symptoms are studied.

The symptoms which are at first noticeable vary considerably in different outbreaks and in individual cases. This is due in part to the different clinical types of the disease and partly to the conditions under which the animal has been kept. It is quite clear that farmers who have had considerable experience are always able to detect cases at a remarkably early stage merely by watching the habits of the animal in the veld.

As in many other diseases, isolation from the flock may be an early symptom, and attention may first be attracted by the fact that the animal lags behind the flock, easily tires when driven, and drops down when left, apparently exhausted from very little exertion. If it is now approached or threatened with a stick, it will jump up and run a short distance, only to go down again still more exhausted. If it is again disturbed, it will repeat this performance until it becomes completely exhausted, when it will refuse to move, even when urged.

There may now be noticed frequent mouthing movements with periodical protrusions of the tongue, accompanied by a dribling of a somewhat frothy saliva. At this stage or later, half-chewed balls of hay will be found partly to occlude the oral passage, as is sometimes the case in the acute form. If the animal has been much disturbed and excited, some of the superficial muscles may quiver or tremble, and some groups of muscles may be in a state of clonic spasm, with the result that the animal assumes an unnatural attitude.

Even before the characteristic symptoms have appeared, the animal may adopt some peculiar attitude of holding its head, and on casual observation the observer might get the impression that the mouth is swollen. On closer examination, however, it will be noticed that the lower jaw is drooping; the false impression was conveyed by the fact that the mouth was plugged with half-chewed hay. It will also be noticed now that, owing to clonic spasms, the animal has partially lost voluntary control over the muscles of mastication and deglutition, which accounts for the drooping lower jaw.

The excessive salivation apparently results from the accumulation of ingesta in the pharynx.

The appetite generally remains good, but the ability of the animal to eat depends largely on the degree of spasm in the muscles of mastication. In fact, in the early stages the affected animals may eat as diligently as the others.

The appetite remains so capricious that the animal, although unable to swallow, will eat practically anything within its reach and fill its mouth with balls of food as often as these are removed. Even animals too badly affected to move about actively may continue taking in food, only to accumulate it at the back of the mouth.

When the animal is exercised, at first it may be active like a healthy animal, but it tires out much sooner if chased continuously, and drops down exhausted, to lie in a normal position, panting and apparently very much excited. Clonic spasms now appear in a much more marked degree and may spread to involve individual muscles, groups of muscles, or even, in advanced cases, the whole musculature. The contractions include mere fibrillations, tremors, or even tetaniform convulsions. In some case twitchings appear to be confined to the superficial muscles of the head and neck, but more often spread over the shoulder and the rest of the body. The clonic spasms involve in particular the muscles of the head, neck, and back, giving rise, in addition to the masticatory troubles, to a well-marked spasmodic torticollis, associated in many cases with an arched back.

As a result of the torticollis, the animal may have great difficulty in holding up its head, which therefore frequently nods and dangles loosely during progression. In severe cases the head may be held very low and often pressed down on the sternum. Any form of excitement will now materially aggravate the condition and provoke the most alarming symptoms. The animal may become hyperaesthetic to a degree depending more or less on the amount of excitement to which it is subjected.

Another factor which considerably intensifies the condition is exposure of the animal to the direct rays of the sun.

If the patient is allowed to rest in a cool shaded spot, the symptoms abate temporarily, and it becomes somewhat refreshed and will walk for a short distance again; but even the slightest exertion now may induce the most violent symptoms. It moves with great reluctance, and, if it is compelled to move, the most distressing symptoms may be manifested. It may even refuse to stir and, if further harassed, may collapse and die from over-exertion and syncope. The animal presents a pitiful appearance, and not only have the clonic spasms of the muscles increased to an alarming extent, but respirations have become spasmodic and increased in frequency; the pulse-rate is very much accelerated, and the pulse thin, wiry, and sometimes imperceptible.

The animal remains completely conscious, but takes no interest in its surroundings, and may either lie down or stand with legs wide apart, obviously in great pain. When an affected animal is left undisturbed, it will, whenever possible, make for a shaded spot, where it will lie down for hours or even days on end if it survives.

In the later stages inco-ordination of movements is often shown by irregularities and stiffness of gait, but in a number of cases the gait alters very little up to the time when the animal goes down in an exhausted condition. If a flock of goats, amongst which there are a number affected, be watched coming to the kraal at night, a string

of affected animals will be noticed lagging behind, laboriously struggling along, the worst cases being farthest behind and often failing to reach the kraal before nightfall.

On account probably of the peculiar position it assumes when drinking, the suckling kid manifests symptoms somewhat more characteristic than those of the adult, spasmodic torticollis in particular being more marked.

The head is twisted sideways and is maintained more or less in this position during the periods of rest; but when the kid is disturbed or excited, the wry-necked appearance becomes extraordinarily exaggerated, especially if it be induced to suck. In attempting to manipulate the teat with its mouth the kid is unable to raise its head sufficiently to attain its objective, or attains it only with obvious difficulty, in which case it is unable to maintain the sucking position, and the head drops and hangs helplessly. The kid has practically completely lost voluntary control over the muscles which elevate the head. It realizes its helplessness and incompetence, and is obviously very hungry, but has become entirely exhausted in its attempts to drink, and bleats pitifully. Even if it is assisted by having its head held up and a teat placed in its mouth, the kid may still have great difficulty in sucking, owing to spasm of muscles of the tongue and lower jaw. Generally, however, it is able to partake of some milk, in which case it often survives for some days and may even improve and recover; but there is always a very rapid loss of condition. If the symptoms have once appeared and the kid is only slightly disturbed afterwards, the characteristic wry-necked appearance, which is assumed in the attempts to drink, presents itself in a greater or less degree.

In the horse, symptoms more or less resemble those enumerated above, but this animal lies down only during the later stages of a severe attack, and when there are obvious signs of abdominal pain. Spasmodic torticollis generally appears more distinctly and more regularly than in the adult goat. The horse is more hyperaesthetic and is more sensitive to exciting influences than the goat. It easily becomes irritated and annoyed by the presence of flies and sounds like that made by a loose halter chain. It generally sweats profusely, is often very restless, kicks, tramps, and strikes and bites towards its flanks. A symptom which may be considered premonitory and which is frequently brought out by exercise is paralysis of the lower lip, which consequently hangs loosely.

As in the case of other animals, symptoms are brought out in an aggravated form after exercise.

In the dog, symptoms appear as in other animals; but, as a result of torticollis, the animal trots with a high stepping gait, with the neck so distinctly arched that the lower jaw is pressed up against the sternum and the mouth kept unnaturally wide open. If the animal is exposed to the sun, the eyes are staring and the pupils somewhat dilated. Vision is never distinctly impaired and lacrimation remains normal.

In the fowl, there may be complete inco-ordination of movements. The neck may be somewhat spirally twisted, with the result that the beak points backwards, the left eye facing the right and forwards.

In all species, excessive salivation is not only an early symptom, but, like clonic spasms of the muscles, continues throughout the course of the disease.

The urine is always clear, and during an attack the patient generally micturates small quantities at short intervals, with the result that the bladder is often found contracted and empty at post-mortem. No excess of sugar has been detected in the urine, but tests on the urine of the same animal on different occasions have shown the presence of albumen in some cases (horse).

The temperature has never been found to be affected to any significant degree, and abnormalities in temperature were always found to be associated with factors which had no bearing on the disease.

During the periods of rest the pulse-rate is somewhat decreased, and the pulse, although still fairly weak, can be made out distinctly and is regular. The respirations also become increased in frequency, but remain spasmodic.

Course.

As has already been stated, the appetite is apparently not very much affected during the early stages of the disease, but in the majority of cases, soon after the appearance of symptoms, the animal becomes unable to eat, and quits its food owing to clonic spasms and loss of voluntary control of certain muscles. A fairly rapid loss of condition is generally observed, and sometimes this may be noticed even before the incidence of the disease. On the other hand, in acute cases the patient may be in the most excellent condition when symptoms appear, but soon loses condition rapidly if it survives for any length of time. Most animals, however, that contract the disease after prolonged exposure are more or less emaciated.

If the animal survives an acute attack, it progressively becomes weaker and weaker, lies down most of the time (excepting horse), and will move only when urged, in which case the most distressing symptoms may be manifested. Any form of exertion, excitement, exercise, or exposure to the sun has a deleterious effect on the animal, and considerably aggravates the disease.

Cool cloudy weather or confinement to shade has a most favourable influence on the course of the disease, whereas clearing of the sky and renewed exposure to strong direct sunlight may precipitate the reappearance of nervous symptoms, in many cases within a few minutes.

Animals may recover after displaying a number of symptoms of the chronic form provided they are left undisturbed and properly sheltered, and are able to take food, or are judiciously fed. A number of deaths result from starvation, exposure, and exhaustion rather than from the direct effects of the disease. Hence by eliminating these contributory factors the course of chronic krimpsiekte often becomes much more benign, and sometimes ends in recovery.

The mortality, therefore, varies according to the form of the disease (chronic or acute), and the conditions under which the animals are kept. It may be over ninety per cent. in some cases and lower than twenty-five per cent. in others.

Immunity.

Animals known to have suffered from the disease once, seldom or never contract krimpsiekte again. It is only too well known that old animals annually exposed to affected areas seldom contract the disease, whereas equally old animals, newly introduced, or young animals reared in the locality, contract the disease in a most alarming

proportion. It would therefore appear that, by long-continued exposure, in the former case the animals have either acquired an immunity to the disease, if it happens to be of an infectious nature, or have learned to avoid eating the plant, if the disease is of the nature of a plant intoxication.

Pathological Anatomy.

The main lesions noted in Appendix B can be considered as follows:—

The Mouth and Lips.—These are soiled with dirty saliva and particles of ingesta. The mouth is partly open and the tongue frequently hangs out. The coat is ruffled.

The Spleen, Lymphatic Glands, Lungs, Pancreas, Sexual Organs, and Nervous System.—Not obviously affected.

The Heart.—Haemorrhages are observed underneath the lining membranes. These vary from mere petechiae and ecchymoses to large haemorrhagic extravasations, which are particularly well-marked underneath the endocardium.

The Myocardium.—When cut, this often shows distinct light striations, and the general appearance of the cut surface is somewhat pale. The muscle appears opaque and frequently feels flabby. The cavities are partly or wholly distended with coagulated blood. The pericardial sac sometimes contains an abnormal amount of fluid. The epicardial fat may be soft, translucent, and gelatinous in appearance with a yellowish pink tinge and a number of small red spots.

The Liver.—It remains more or less normal in size and the colour varies, on section, from light yellowish brown to reddish brown. In many cases the lobulation can be made out, blood escapes from the cut surface, the central vein is dilated, and the periphery of the lobule is paler than the central part, or vice versa. In other cases, again, the lobule is uniformly pale.

Sometimes the consistency is quite firm, but at other times the liver may be somewhat friable. Where a gall-bladder is present, it is distended with yellowish green viscid bile.

Kidney.—The capsule is easily detached and the renal corpuscles are fairly prominent. The kidney is somewhat swollen and the boundary zone is distinct. The cortex is light yellowish brown or brownish red. The consistency is firm. The perirenal fat varies according to the condition of the animal.

The Urinary Bladder is generally empty and the mucous membranes of the urinary tract are normal.

The Thyroid and Adrenal Glands are normal in appearance.

The Digestive Tract.—The mucous membrane of the glandular part of the stomach is often dirty greyish pink and swollen. There may be small hyperaemic areas on the mucous membrane of the stomach and small intestine. These may be of the nature of petechiae, ecchymoses, or large streaks of extravasations. The mucosa is swollen in parts of intestine, and the ingesta, small in amount, is pulpy, pasty, or mucoid. Regurgitated or only partly digested food may appear in the mouth, pharynx, or oesophagus. Often the large intestine may present lesions similar to those of the small intestine.

The Musculature.—This may appear somewhat dull opaque and brownish red, but no obvious changes can be made out with the naked eye.

There is evidence of inanition and cachexia in those animals where there has been starvation for several days as a result of the disease.

PATHOLOGICAL HISTOLOGY.

Heart.—The muscle fibres stain well with Haemalaumeosin. The chromatin of the nucleus and the nucleolus are clear and distinct. The striation is not obviously affected, but in sections stained with Scharlach numerous small globules of fat, arranged longitudinally, become evident. Apparently the globules are arranged between the fibrillae. Some fibres in the same field may contain considerably more fat droplets than others. Amorphous aggregations of brown pigment are found in many cases at the poles of the nuclei and are suggestive of lipo-fuscin associated with brown atrophy.

Salivary Glands.—In most cases there were no obvious changes, but in Horse No. 15134 some droplets of fat in the epithelial cells of the alveoli and tubules were seen.

Adrenal Glands.—The cellular elements of both cortex and medulla show nothing abnormal, but in the case of Horse No. 15134 an excessive amount of fat was observed in the cortical cells.

Kidneys.—Some of the epithelial cells of the tubuli contorti contain droplets of fat in fair amount. In some cases all the cells in a particular field may be involved. The nuclei of the epithelial cells generally stain well, but occasionally some nuclei stain uniformly dark and are smaller than others (Pyknosis). In other cases, again, it is not possible to make out nuclei definitely (Karyolysis). The capillaries are generally distended with blood.

Liver.—The intralobular blood sinuses are distended with blood and distort the columns of hepatic cells. Generally the nuclei stain well, but in some cases definite changes are observed. Some nuclei are contracted and small, and stain uniformly darker (Pyknosis). In other cases, again, no distinct nuclei can be made out (Karyolysis). The cytoplasm of the hepatic cells contains droplets of fat of varying sizes. The globules may either be scattered irregularly throughout the cytoplasm of the cells or coalesce to form one large droplet, which may be larger than the cell proper and force the nucleus eccentrically. The fat may affect the cells of a lobule uniformly or may involve the peripheral cells more than the central ones, or vice versa.

Striped Muscle.—Apparently definite lesions associated with this disease occur in certain muscles. The nuclei of the sarcolemma, arranged longitudinally in bead-like fashion, are apparently increased in number. The capillaries are distended with blood. The muscle fibres, stained with Scharlach, exhibit the presence of fat, which is arranged in the form of small droplets between the fibrillae. These may be so numerous and closely packed as to distend and discolour the muscle fibre. The droplets may even partly obscure the striation and are usually arranged in longitudinal rows. In some muscles the fat globules are very small and scattered irregularly throughout the fibre, whereas in others, again, no fat can be made out.

The affected muscles may be divided into three groups:—

(1) Those muscles which exhibit spasm during life and which are extensively infiltrated with fat. Under this category may be included *M. masseter*, *M. pterygoideus*, crura

of the diaphragm, and in a number of cases also *M. splenius, M. trapezius, M. serratus ventralis, M. complexus, M. longus colli,* and *M. rectus capitis.*

(2) Those muscles which are moderately infiltrated with fat and which do not exhibit symptoms of spasm during life. These include *M. brachiocephalicus, M. omo-hyoideus, M. longissimus capitis et atlantis, M. intertranversales colli,* and *M. triceps* (slightly).

(3) Those muscles which are more or less unaffected; of these the following are examples: The extensor and flexor muscles of the carpus, tarsus, and digit, *M. gastrocnemius, M. psoas major, M. gluteus.*

Nervous System.—Although various preparations were made from the central nervous system and the peripheral nerves, no obvious pathological alterations were detected.

Summary.

The following lesions were observed in cases of krimpsiekte:—

(1) *Liver.*—Fatty infiltration, venous hyperaemia, necrobiosis.
(2) *Heart.*—Brown atrophy, fatty infiltration.
(3) *Kidney.*—Fatty infiltration, venous hyperaemia, necrobiosis.
(4) *Serous Membranes.*—Sub-epicardial and sub-endocardial haemorrhages.
(5) *Striped Muscle.*—Fatty infiltration, slight atrophy.
(6) *Intestines.*—Hyperaemia of mucosa, enteritis.

Discussion.

Probably in a few cases some of the changes observed in the liver and kidney may be of the nature of an autolysis (post-mortem), but in those cases where a post-mortem was made immediately after death the alterations can be explained as due to regressive changes.

In the musculature lesions are well-marked in those muscles or groups of muscles which are affected with spasm in the living animal, e.g. the muscles of mastication, the extensors of the head on neck, and the crura of the diaphragm. On the other hand, muscles, like those of the limbs, which do not manifest a cramped condition during life, do not develop definite changes. It would, therefore, appear that the spasm in certain muscles will probably account for the fatty infiltration observed in those muscles; but, as spasm is partly a nervous affection, it must be admitted that, although no obvious lesions were noticed in the nervous system, it is by no means clear that the nervous system is not affected histologically in some way or other.

Experimental Determination of the Cause of the Disease.

(For details see Appendix A.)

When these investigations were instituted, some definite information was available to the effect that Hutcheon (7) had reproduced a disease nenta in the eastern Cape by feeding goats on *Cotyledon ventricosa* Burm. and krimpsiekte (12) in bovines by feeding "klimop" (*Cynoctomum capense*). But, on account of the seriousness of a condition known also as krimpsiekte in the western Karroo and the scepticism with which the connexion between this disease and certain cotyledonous plants was regarded, it was decided

by the Director of Veterinary Education and Research to institute investigations in order to elucidate the cause of krimpsiekte as it occurs in the Districts of Ceres, Matjesfontein, Sutherland, Calvinia, Prince Albert, etc.

Acting, therefore, on instructions from the Director of Veterinary Education and Research, the writer arranged to make a tour through the most seriously afflicted areas in company with Dr. Marloth, who, along with other observers doubting the relationship between cotyledonous plants and krimpsietke, urged the necessity of an exhaustive investigation into the causes of the disease. Prior to this, however, Curson (9), of this division, had fed goats on *Cotyledon wallichii* obtained from Dr. Luttig at Prince Albert, with the result that some died from an intoxication which he called "opblaas krimpsiekte."

The conclusions of Curson were, however, not acceptable to Marloth, who writes: "To me, who am merely a layman in these matters, it appears that the disease has some resemblance to trichinosis and may consequently be due to an internal parasite, either of animal or microbic nature."

In another letter Marloth (10) concludes:—

"1. I consider krimpsiekte and opblaas krimpsiekte as two entirely independent diseases.

"2. I consider the true krimpsiekte (nenta) to be connected with or caused by some kind of parasite of animal or bacterial nature.

"3. The nature of the parasite could probably be ascertained by an investigation of the flesh and blood of krimpsiekte animals."

On account of the strong views held by a botanist of such repute as Dr. Marloth, and the latter's acquaintance with krimpsiekte areas, the writer was greatly pleased when Dr. Marloth consented to the proposed tour through the Ceres and Tanqua Karroo via Sutherland to Calvinia. From 1st to 10th September, 1921, a number of localities were visited where krimpsiekte was known to occur annually from about July to November, or even later, if the animals were not previously removed to non-affected veld. It was observed that farmers were by no means unanimous as to the possible causal agent of the disease. Although the majority contended that the disease followed some plant intoxication, the probability of a parasitic causal agent was strongly supported by a number of careful observers. It was even suggested by some and strongly believed by others that krimpsiekte had some relation to Malta fever.

The symptoms appeared obviously to result from the action on the nervous and muscular systems of a toxin or poison, and the object of this inquiry was to indicate, if possible, whether the hypothetical toxin substance was elaborated by some living organism which had gained access to the animal body, or whether it was normally present in some species of plant eaten by susceptible animals, or was produced in such a plant as a result of the invasion of its tissues by some organism or other.

Whether or not the cause of the malady is of a vegetable nature, it was observed during the course of the expedition through the Karroo that cotyledonous plants grew in great abundance on all affected areas. Further, no evidence came to light to disprove the contention that the disease was due to an intoxication following the ingestion of a plant.

Although an occasional case of krimpsiekte was reported from localities where the suspected plants, especially cotyledons, were considered not to occur, it was revealed on closer investigations that these plants did grow in scattered areas in such localities.

On the other hand, there is little doubt that suspicious cotyledons occur, sometimes abundantly, where the disease has never been noticed to appear. It is suggested as a tentative explanation that this might be due to differences in composition existing between the specimens of plants found in the various situations, such differences arising directly from changes of environment or from the occurrence of two or more species or varieties, or from the presence, in some instances, of parasitic organisms either in or on the plant.

The bulk of the evidence collected, however, suggested very conclusively that the conditions under which the animals were kept were chiefly responsible, and that, whereas in some cases the animals on affected farms had very little opportunity of eating suspected plants, the most important point was that there was no inducement or necessity for doing so, and therefore the animals avoided the plant.

Although the mass of evidence collected during the expedition pointed distinctly towards the probability of a plant intoxication, and in particular towards a cotyledonous poison, it was attempted during the course of this inquiry, as far as practicable, to explore simultaneously all the more probable hypotheses.

Throughout the whole course of these investigations all experimental animals utilized were obtained from localities where the disease had never been known to occur. The food given to all animals was in all cases known to be above suspicion. The same foodstuffs were given to a number of other animals either available for experiment or utilized for experiments of an entirely different nature.

Preliminary Investigations.

1. *Transmission by Contact and Inoculation.*

Attempts were made to transmit krimpsiekte to susceptible animals, both by contact and by inoculation. A typical natural case of krimpsiekte in the goat was brought to the laboratory and left in contact with healthy susceptible goats for thirty-six hours. Not a single one of these showed the slightest tendency to develop the disease.

Between 20 to 50 c.c. of blood was transfused from a natural case of krimpsiekte to a susceptible kid, with negative results.

2. *Bacteriological Examination.*

Attempts were made to cultivate micro-organisms from various tissues and organs of an affected animal killed for the special purpose, common laboratory media being used. All the tubes remained sterile after inoculation with cerebro-spinal fluid, blood, aqueous humour, brain tissue, and spleen.

A number of fresh preparations were made from the blood, tissues, and body fluids of krimpsiekte animals. After fixing and staining, these were examined microscopically, with negative results.

The only parasite revealed in sections of the various organs and tissues was an occasional sarcosporidium, which, according to Viljoen, (13) will not cause obvious symptoms.

3. *Macroscopical Parasites.*

On post-mortem examination *Haemonchus contortus*, *Oesophagostomum columbianum*, etc., were found in the digestive tract, but as these could not be considered to have any bearing on the disease, not much significance was attached to their presence.

4. *Transmission by feeding Affected Carcasses.*

In order to determine whether the disease could be reproduced by feeding carnivora on krimpsiekte carcasses, the meat of a natural case of krimpsiekte was fed to one dog, the brain and spinal cord to a second, and the stomach and intestines to a third. The first two dogs remained healthy and were discharged after twenty-five days, but the third was found dead in its kennel after ten days without manifesting any obvious symptoms.

Experimental Determination of the Disease.

On account of the analogy which krimpsiekte bears to the condition nenta (7) described by Hutcheon, the negative results obtained by attempting to transmit the disease by contact or inoculation, the failure to discover any probable parasitic causal agent, and the strong popular opinion in favour of a probable vegetable intoxication, strengthened by the seasonal occurrence and distribution of the disease, it was decided to commence extensive plant-feeding tests in order to determine whether any of the suspected plants could be incriminated.

As a result of field observations, it was noticed that the distribution of krimpsiekte varied directly with the distribution of certain cotyledons.

In fact, in no instance was a case of krimpsiekte reported on a farm where these plants did not occur in abundance. On numerous occasions farmers have actually pointed out the kandelaar-bos or bandjes-bos (*Cotyledon wallichii*) as the suspected cause of the disease. Other plants incriminated were *C. paniculata*, *C. orbiculata*, *C. reticulata*, *Asparagus stipulaceus*, and *Euphorbia mauritanica*.

Large quantities of each of these plants, excepting the euphorbia, were fed to goats, sheep, and fowls, with the result that every animal fed on the kandelaar bos (*C. wallichii*) died within a week. One of the goats fed on *C. paniculata* showed symptoms of laminitis, which disappeared on discontinuing the plant-feeding. Both *C. orbiculata* and *C. reticulata* had no effect on goats and sheep, but gave rise to an intoxication in fowls, characterized by a marked inco-ordination of movements. The asparagus was eaten in large quantities by the different animals with impunity.

On account of the remarkable toxicity of *C. wallichii*, extensive feeding-tests were commenced with this plant, goats, horses, fowls, and sheep being used. Later on dogs were fed on the carcasses of the dead animals.

Cotyledon wallichii Harv. belongs to the natural order Crassulaceae, and is popularly known as kandelaar bos or bandjes bos. Mr. A. O. D. Mogg describes the plant as follows:—

Stems short, thick, 6 to 18 inches, branched at base, forming a coarse suffruticose rosette; *epidermis* cerosotranslucent, leathery, grey, densely tuberculate with spirally arranged echinulate,

hardened leaf scars; *sap* yellowish, densely viscous, highly hygroscopic; *leaves* terminal, rosulate, scattered, fleshy, green, glabrous, terete, fusiform, 1 to 3 inches long, $\frac{1}{8}$ to $\frac{1}{4}$ inch in diameter, deciduous; *peduncles* elongate, 1 to 2 feet long, panicled, the branches of the panicle alternate; patent, simple, or forked, scorpioid; *flowers* shortly pedicillate, sub-secund, nodding, yellow-green, $\frac{1}{2}$ inch long; *tube of corolla* $1\frac{1}{2}$ to twice as long as the calyx, rather longer than the oblong, acute limbs of the petals. The peduncles, panicle, calyx, and corolla are densely covered with a viscous brown pubescence.

Habitat.—Elandsberg, *Dr. Wallich*, north sides of snowy mountains; *Burka*, Cape; *Villette*, in Herb, Hook. An extreme xerophyte of the western Karroo about Prince Albert, Hex River, Matjesfontein to Williston and Calvinia, and farther west.

Closely allied to *C. fasicularis*, but differs in pubescence, smaller flowers and larger calyx, in proportion to corolla ("Flora Capensis").

At the commencement of the experiments the procedure was to starve the animals for about twenty-four hours and then to give a weighed ration of the food either alone or mixed with other food. In all cases the animals displayed such reluctance to eat the plant, in spite of hunger, that it was decided to resort to drenching, balling, or forcible feeding. During the experimental feeding the animals were exercised periodically in order to bring about conditions somewhat resembling those found in the field. In the first series of feeding-tests such heroic doses were given that in nearly every case the animal succumbed before characteristic symptoms were manifested. The main fact disclosed, therefore, was the remarkable toxicity of the plant.

The Source and Condition of Cotyledon used.—The plant was obtained from Prince Albert and Matjesfontein, Cape Province. It was identified as *C. wallichii* Harv. by Dr. Schönland in Grahamstown, and later by Dr. E. P. Philips when the experiments were transferred to the Onderstepoort Laboratory. In cases where the plant was not fed immediately, it was spread out on a cement or wooden floor to prevent fermentation and growth of moulds. Both leaves and the flowering shoots were fed, either in the fresh state or after drying in the sun. Apparently the flowering shoots were more toxic than the leaves, and drying did not appreciably affect the toxicity of either leaves or flowers. In all cases the plant was either minced or ground into a powder before dosing.

The Period necessary for the Appearance of Symptoms.—In cases where small doses were given periodically, symptoms did not appear until a toxic amount had been received and assimilated; if, however, one or more lethal doses were given, symptoms were manifested in two, three, or four days' time, or the animal died without showing characteristic symptoms. Consequently, depending upon the amounts fed, the period between the commencement of the experiment and the first appearance of symptoms varied from a few days to many weeks.

It was consistently found that by dosing two animals of the same species and of about the same weight concurrently on equal doses of leaves and flowering shoots, those dosed on the latter invariably developed symptoms sooner and succumbed earlier to the toxic effects of the plant than the former. In fact, in all cases much smaller doses of flowers than of leaves were required to produce symptoms.

Symptoms Produced by C. wallichii *Intoxication.*—By feeding different animals varying doses of the plant at different periods various phases of the intoxication could be observed.

As already stated, an initial large dose frequently killed the animal before symptoms were manifested. Consequently a gradual reduction of the dose tended to bring out symptoms which in many cases were characteristic of krimpsiekte as observed in the field. It was further found that by giving small doses at short intervals more characteristic symptoms were brought out than when a single lethal dose was given at once. In all cases the incidence of symptoms was considerably hastened by exercising the animal. In fact, many animals which were apparently healthy when left undisturbed evinced the most alarming symptoms when excited or exercised. Further, exposure of the patient to the sun always accelerated to a remarkable extent acme, and the appearance of the most distressing symptoms.

The incidence of symptoms, therefore, depends on four factors, viz., the amount of toxin ingested, the period over which dosing extended, the amount of exercise given, and the amount of exposure to the direct rays of the sun.

The course of the disease also depended on these four factors, but indirectly it was materially influenced, especially in milder or chronic cases, by the amount of nourishment which the animal was able to take. Thus, when comparatively mild cases end fatally, the cause can generally be attributed to starvation, over-exertion, or exposure.

It is evident then that when an overdose of the plant is given at once, or during a comparatively short period, the animal may be dead before the manifestation of symptoms, or symptoms may be of such an acute nature that the animal seldom survives for longer than twenty-four to thirty-six hours. The symptoms evinced under such conditions tally more or less with those described as *opblaas krimpsiekte* in the field, and any deviation from the natural state could be explained by the artificial conditions under which the experimental animals were kept.

In those cases where small repeated doses were given over a long period there was generally noticed a progressive loss of condition by the time that symptoms became manifest. It is difficult in such cases to estimate the relative proportion of loss in condition which can be attributed, firstly, to the direct loss resulting from the interference with metabolism and the atrophy and degeneration in certain tissues brought about by the toxic properties of the plant, and, secondly, to starvation resulting from inhibition of certain groups of muscle, and consequent interference with mastication and inability to eat.

Such animals developed a chronic or insidious form of the intoxication, which coincides with the condition already described as *chronic krimpsiekte*, and, with only slight modifications, all the manifestations of the natural diseases were reproduced. Hence the description already given for the symptomatology of krimpsiekte can be adopted also for that of *C. wallichii* intoxication.

By feeding this plant, symptoms following an intoxication were produced in horses, goats, sheep, and fowls, and, subsequently, an intoxication giving rise to similar symptoms was produced in dogs by feeding the liver and meat of krimpsiekte carcasses.

Some of the Properties of the Toxin.—The toxin of *C. wallichii* was found to be thermostabile, as it was not destroyed by autoclaving at 120° C. for 15 minutes, nor by boiling in water for 30 minutes.

Attempts were made to extract a toxin from the plant, with the result that only a very small amount was extracted in water by boiling for 30 minutes, but the bulk of the toxin appeared in the filtrate after extracting the plant with 60 per cent. alcohol acidified with hydrochloric acid (1 per cent.).

Immunity.—An animal that had recovered from krimpsiekte remains susceptible to the disease, but acquires such a dislike for the plant that it is not likely to eat it even under coercion.

Conclusions.

On reviewing the results of these experiments, there can be little doubt that the plant *Cotyledon wallichii* Harv. (kandelaar bos or bandjes bos) is at least one of the causes of the disease known as krimpsiekte in the western Karroo. The possibility of the parasitic nature of the disease can be completely ruled out for the following reasons:—

(1) Neither by inoculation nor by contact was it possible to transmit the disease.

(2) In no instance on post-mortem was a parasitic organism found which might suggest a parasitic nature of the condition.

(3) By sterilizing the plant, both by boiling and by autoclaving, the virulence of the plant was not appreciably reduced, as would be expected in the case of a parasitic organism.

(4) Very large quantities of the meat of krimpsiekte animals were required to be fed to dogs in order to produce symptoms, since the toxin had apparently been considerably diluted as a result of its distribution through the tissues. If the disease were of a parasitic nature, considerably smaller doses would be expected to reproduce the malady.

(5) Furthermore, since the toxin of this plant is apparently thermostabile, the meat and organs of a krimpsiekte animal cannot be considered safe for human consumption even after boiling or cooking.

This conclusion, it will be observed, is contrary to popular belief.

Treatment.

In most cases, by the time symptoms are observed, the condition has developed so far that treatment seldom can be of any avail. In milder cases, however, if the patient is judiciously fed, not disturbed, excited, or unnecessarily exposed to the rays of the sun, recovery may occasionally supervene.

Any form of treatment should, therefore, be of a prophylactic nature. The animals should be kept away from affected areas, and, more particularly, animals not used to the vegetation should not be allowed to graze in veld where *C. wallichii* grows, especially after they have been driven for some distance without grazing.

The safest and most rational method would, however, be to eradicate the plant; but for economic reasons the difficulties are practically unsurmountable, as the plant grows over vast areas in the driest parts of the country where the ground is in most cases of comparatively little value. Where the value of the ground would justify the expense, eradication of the plant appears to be the only sanguine method of preventing krimpsiekte.

ACKNOWLEDGMENT.

I wish to express my appreciation of the assistance given me by the following gentlemen:—Dr. G. de Kock, Dr. E. P. Phillips, Dr. R. Marloth, and Dr. P. C. Luttig.

APPENDIX "A."

The following records were made as a result of feeding various animals on *Cotyledon wallichii*:—

GOAT No. 8.—Admitted to experiment on 15*th August*, 1921, is fed on 1,100 grammes of the chopped leaves over a period of five days.

20*th August*.—Although animal is duli, it is dosed.

21*st August*.—Animal is distinctly sick. It is in great pain and is tympanitic; it grunts, looks distressed, and saliva dribbles from its mouth. When it is driven a few paces, animal becomes more distressed, seeks shade, stands with its mouth wide open, and bleats moanfully. It lies down later and groans *continuously*.

22*nd August*.—Animal lies down in corner apparently in great pain and shows evidence of purgation. When disturbed, it runs for a short distance, lies down again, and rises to move only reluctantly. Symptoms remain the same as on previous day, but some of the muscles tremble slightly. Breathing is accelerated and pulse fast and imperceptible. When animal is urged to move, it staggers, sways, falls, and dies.

GOAT No. 1.—Admitted to experiment on 23*rd August*, 1921, and is fed on 75 grammes of the chopped leaves over a period of six days.

28*th August*.—Animal refuses to eat, looks dull, and salivates profusely. Respirations are accelerated and pulse quick.

29*th*-31*st August*,—Symptoms remain the same.

1*st September*.—Symptoms as before, but lower jaw is hanging and mouth is open.

2*nd September*.—On exercise animal goes with a weak and staggering gait and is in great pain. It lies down when left undisturbed, refuses to eat, and looks very thin and empty. It dies during the night.

GOAT No. 34 X.—Admitted to experiment on 9*th November*, 1921, and is dosed with 92 grammes of the minced plant over a period of eight days.

17*th November*.—Animal lies down, showing evidence of dull pain. Lower jaw is drooping slightly and moist with saliva. A large unchewed ball of grass is found in the mouth. When driven, animal soon lies down exhausted. Respiration and pulse-rate increased in frequency. Animal is somewhat tympanitic.

18*th*-21*st November*.—Symptoms remain the same, and animal lies *continuously*.

22*nd November*.—Saliva is dribbling over lower jaw. Mouth is partly open, and muscles of mastication appear paralysed, as the animal cannot even bite a finger placed between its molars. It is found dead at noon.

GOAT (KID, THREE MONTHS OLD) No. 46.—Admitted 9*th November*, 1921, and is dosed on 24 grammes of the minced flowers over a period of seven days.

16*th November*.—Kid looks dull and dejected. When exercised, it rushes to shade, where it lies down exhausted from the slightest exertion. It becomes distressed when exposed to sun. Saliva is dribbling and mouth is partly open, but kid is able to eat apparently with comfort. Neck appears stiff, and head is held slightly to one side. Muscles of neck feel hard and are apparently in a state of clonic spasm.

17*th November*.—Animal lies down and shakes head up and down on exercise, as if head is loosely attached to neck. When left, kid flops down exhausted on ground, always making for the shade. When rested for a while, it becomes active again and trots with its neck somewhat arched.

18*th November*.—Kid lies down, looking sick and miserable. Symptoms remain as on previous day. It moves now only when urged. Salivation is greatly increased, and animal is unable to eat and is fed on milk. Respirations and pulse-rate are increased in frequency.

19*th*-21*st November*.—Symptoms remain the same.

22*nd November*.—Kid still lies down and looks miserable. Mouth is partly open and is filled with a half-chewed ball of hay. On exercise, the muscles of neck feel stiff, and head shakes much more loosely up and down. It dies the same day.

GOAT (KID) No. 47.—Admitted to experiment on 9*th November*, 1921, and is fed on 36 grammes of the minced flowers over a period of six days.

15*th November*.—Kid lies down, but jumps up on being approached. Mouth is partly open and saliva dribbles over lower jaw. A large ball of unchewed hay is found at the back of its mouth. On exercise, the head shakes loosely up and down. If exercised in sun, kid becomes very much distressed, trembles and shivers all over body. It has lost control over muscles of head and neck, and is unable to fix its head in a position to take hold of its mother's teat, although it appears very hungry. When it is assisted to get hold of the teat, it is unable to maintain its head in a position to suck.

16*th November*.—Kid lies down, with mouth partly open, tongue hanging out, and lower lip moist with dribbling saliva. Another ball of hay is found in mouth. The animal has lost control over its muscles of mastication. The muscles of head and neck are hard and stiff and in a state of clonic spasm. The head is twisted to one side. Kid is very hungry, but is unable to suck or keep its head up.

17*th November*.—Symptoms as before. Kid shivers and trembles all over if exercised in sun. Pulse-rate and respirations are markedly accelerated.

18*th November*.—Symptoms as before, but animal gets very thin; appetite remains good, but animal is unable to eat or suck.

19*th*-22*nd*.—Kid becomes weaker and dies on 23rd November.

Goat (Kid) No. 17.—Admitted 19*th October* and fed on 50 grammes of minced leaves over a period of six days.

25*th October*.—Kid is apparently normal, but mouth is slightly open and a ball of grass is found at the back of its mouth. On exercise, it soon tires out and lies down exhausted and salivates excessively.

26*th October*.—Kid looks dull, and when exercised shakes head up and down; muscles of neck are stiff and hard. Head is twisted towards one side, and the jugular furrow is deep and wide. The animal has great difficulty in lifting its head. Mouth is kept partly open and salivation is profuse.

27*th*-31*st October*.—Symptoms remain the same.

1*st November*.—There are signs of improvement.

11*th November*.—Kid is apparently quite well and is discharged.

22*nd November*.—Animal is again returned to experiment and fed on 7 grammes of the flowers.

24*th*-25*th November*.—Salivation is excessive, lower jaw is moist. Muscles of the neck are hard and stiff. When exercised, head shakes loosely up and down, neck is stiff, and head is held towards one side. When exposed to or exercised in the sun, muscles all over body tremble, and kid is hyperaesthetic.

26*th November*.—Symptoms are much more marked. Muscles all over body show twitchings and tremblings. The animal becomes hyperaesthetic. Head is hanging and animal has no power to hold it up. It tries to suck, but cannot lift its head to a position suitable for sucking. When assisted to get hold of teat, its head falls back to its previous position when left to itself. It is very hungry and realizes its inability to drink, and bleats pitifully. Pulse-rate and respirations are accelerated.

27*th November*.—Symptoms are much more aggravated. Muscles of the neck become stiff, forming a large swelling on the one side and a deep hollow on the other, and the head is twisted on a horizontal axis. Kid is exposed to the sun, trembles and shivers all over body; hyperaesthesia is marked. Animal becomes very much distressed, stands with legs wide apart, and does not even make an attempt to suck. It dies soon afterwards.

Goat No. 1235 (Weight, 38 Kilos; Eight-toothed).—Admitted 16*th September*, 1922, and is fed on 116 grammes of the minced leaves over a period of twenty-five days.

14*th October*.—Goat looks dull, protrudes tongue periodically, and salivates excessively. On exercise, it becomes distressed, but it remains active.

15*th*-17*th October*.—Symptoms remain the same, and lower jaw remains moist with saliva.

18*th October*.—Mouth is partly open, and saliva is dribbling over lower jaw. Goat cannot bite a finger placed between its molars. It is very hungry, but is unable to masticate or swallow hay, which accumulates in the form of a ball at the back of the mouth. This is removed.

19*th October*.—Condition of animal gets worse; it looks miserable, thin, and empty. A large ball of grass is again removed from the mouth. Animal remains hungry. When exercised, it runs in a listless manner, tires easily, and makes for the shade when left.

Symptoms remain the same; animal becomes more and more emaciated, until it dies on 23rd October.

Goat No. 3223 (Weight, 38 Kilos, Six-toothed).—Admitted 16*th September*, 1922, and is fed on 17 grammes of minced green leaves over a period of twenty-five days.

14*th October*.—Goat is dull and sick and lies down in pen. When driven, it gets tired soon, pants, and drops down on ground to rest. Animal is hungry and picks up bits of grass to eat.

18*th October*.—Animal is exercised in the sun, is active at first, but soon tires out and lies down. During progression, head dangles loosely, neck is arched, and head is pressed up against the chest. Mouth is partly open and salivation excessive.

21*st October*.—Symptoms remain the same, but head is twisted towards one side, and on exercise symptoms become aggravated and animal moves only when urged. The muscles of neck feel hard and are in a state of clonic spasm. Goat is very hungry, but it has lost control over the masticatory muscles and cannot eat, consequently unchewed grass accumulates in the mouth.

The symptoms remain the same until 27*th October*.

28*th October*.—Animal is very sick and listless, lies down in the pen with a dejected look and eyes sunken; it moves only when coerced. Salivation is more marked and mouth is open. Animal is still hungry, but is unable to eat, and a large ball of grass is again found in its mouth. Animal is fed on mealies, and symptoms remain the same for another week, when the goat shows signs of improvement.

24*th November*.—Goat is apparently well, but when exercised it soon tires and pants excessively.

31*st December*.—Goat has apparently recovered.

4*th-5th January*.—Animal is fed on 14 grammes of dried leaves of *C. wallichii*.

6*th-7th January*.—Goat appears sick and is not eating well.

8*th January*.—Animal lies down, inactive, and appears very sick and miserable. Mouth is partly open, salivation is excessive.

9*th January*.—Its condition is worse, and animal lies down and gets up alternately if disturbed. The jugular furrow is wide and deep. The muscles of the neck are stiff and hard, and some stand out prominently.

10*th January*.—Symptoms as before, but on exercise the animal soon becomes tired out and lies down, refusing to stir. The mouth is wide open, tongue is hanging out, and frothy saliva dribbles over lower lip. The muscles elevating the lower jaw appear to be paralysed; a large ball of unchewed hay is found in the mouth.

During progression and immediately after exercise the neck is arched, head is overflexed and twisted to one side, the muscles of the neck feel hard, and bulge out in the form of a large swelling on the one side, resulting in a deep hollow on the other. The animal is unable to lift its head and holds it somewhat skew or slanting.

When animal is exposed to the sun, the symptoms become very much intensified and muscles all over body tremble and twitch. Goat has now become very much distressed and presents a pitiful condition. Pulse-rate and respirations are increased in frequency. The goat dies the same day.

Goat No. 5666 (Weight, 36 Kilos).—Admitted on 5*th January* and dosed with 7 grammes of the dried leaves. This dose is repeated after seven days.

16*th January*.—Goat is lying down in pen. When made to rise, it stretches itself out and manifests abdominal pains. The mouth is kept partly open, and lower lip is moist. Animal holds neck stiff and distended. After a little exercise, goat lies down and shivers all over. During progression head is overflexed on neck and is twisted partly to one side. The muscles of neck become hard and stand out prominently on one side and appear to be in a state of clonic spasm. Exposure to the sun intensifies the symptoms.

17*th January*.—Symptoms remain the same, but animal moves only when coerced. Muscles bulge to form a large prominence on the one side and a corresponding depression on the other side, resulting in twisting the head towards one side.

18*th January*.—Animal lies down with head twisted towards one side. There are clonic spasms of the muscles of neck and back. Lower jaw is markedly depressed and moist with frothy saliva. The animal periodically bleats in a hollow soft tone as if in great pain. It refuses to stir, but when lifted stands with a stiff neck and head twisted so as to be held more or less horizontally. It soon drops down exhausted, lies prostrate, and dies shortly afterwards.

Other goats were fed on varying doses of the plant, resulting in symptoms resembling those in the cases above.

HORSE No. 1.—Admitted on 1st September, 1921, and is fed on about 2,500 grammes of the minced leaves in a bran-mash over a period of forty-nine days. Apparently some of the material is wasted.

9th October.—Horse salivates excessively, but its appetite is good.

10th-25th October.—Salivation continues and lower lip hangs.

27th October.—Horse looks dull and does not eat its bran-mash. Lower lip is hanging and saliva is dribbling all over lower lip and manger. On exercise, salivation becomes more profuse, muscles of neck become hard and stiff, forming a large prominence on the one side of neck and a deep depression on corresponding part of other side. There are clonic spasms of the neck muscles. The neck becomes arched and head twisted to one side and held at a slant (torticollis), the jugular furrow is deep and wide, and some of the muscles of the neck stand out prominently. During progression the animal also shakes its head up and down involuntarily. The horse sweats profusely and soon becomes tired. Muscles all over body now twitch and tremble, the animal is hyperaesthetic, and is irritated by flies. If urged to move, it moves only with reluctance and a staggering gait and becomes very much distressed. Respirations are 36 and pulse 90. When the horse is left undisturbed in the stable, the symptoms subside and the animal becomes relieved. If it is again disturbed, the symptoms reappear in an exaggerated form. Mere exposure to the sun brings out and intensifies the symptoms.

Symptoms remain the same until *30th November*, but the animal now refuses to eat a bran-mash containing the plant.

There is now distinct evidence that the horse is losing condition rapidly.

1st-10th November.—Symptoms gradually disappear and are not brought out even on excessive exercise.

12th November.—Animal is discharged as healthy.

17th November.—Horse is returned to experiment by balling it with 112 grammes of the minced flowers.

19th November, 7 *a.m.*—Horse is distressed, looks dull, lower lip hangs loosely, and saliva dribbles over lower lip. Pulse is 80 and hardly perceptible, respirations 30.

Later the animal shivers, trembles, and is hyperaesthetic. It is very hungry, but cannot eat; a large ball of unchewed grass is found in its mouth and removed. The horse again takes up some hay, which it is unable to chew or swallow and which again collects to form a ball in its mouth.

2 *p.m.*—Some green lucerne is put in the manger; the animal takes it up eagerly, but is unable to hold it in its mouth or chew it, and the hay drops to the ground. Again and again the horse makes an attempt to eat, but it is unable either to apprehend, chew, or swallow the lucerne. Salivation has increased. The muscles over shoulder, neck, and back tremble and twitch. Animal appears nervous, shows colicky pains, lies down a good deal, and when led its gait is staggering.

20th November, 8 *a.m.*—Horse lies down on side in great pain and sweats all over body. There is evidence of struggling during the night. Animal periodically lifts its head and then lets it drop again. It refuses to rise when urged, but when assisted it rises and stands with legs wide apart and in great distress. The animal is nervous, hyperaesthetic, and its muscles all over the body tremble and shiver. Sweat drips from its body and saliva from its mouth. It looks miserable, takes no notice of its surroundings, threatens to go down, and has to be assisted to maintain its balance.

Reflexes remain distinct. Pulse is 130 and can hardly be made out. Respirations are 38.

The muscles of the neck are very hard and stiff and contract so as to form a large prominence on the one side and a deep depression on the other. The neck is arched, and head is twisted to one side and held at a slant: the muscles contract so that the transverse measurement increases at the expense of the dorso-ventral; the ventral curvature of the head and neck assumes the shape of an arc of a circle (torticollis). The jugular furrow is widened. The tongue hangs out, and the animal is unable either to eat or drink, although it makes frequent attempts. When it is left, the horse goes down and lies on its side, lifting its head at intervals and letting it drop again. It maintains this position and shows continuous dull pain, and later becomes semi-comatosed, in which condition it remains until it dies during the night.

HORSE No. 15079 (WEIGHT, 320 KILOS).—Admitted 16th September, 1922, fed on about 1,200 grammes of the minced leaves over a period of eighty days, is left undisturbed for twenty-eight days, and then dosed on about 500 grammes of the leaves dried.

The animal is exercised periodically.

4th November.—After exercise, lower lip is hanging loose and saliva is dribbling profusely over it. Symptoms remain the same for about three weeks; then horse is not fed for about one month.

1st January, 1923.—The horse is apparently normal and does not show symptoms on exercise. It has, however, lost condition meanwhile.

2nd-5th January.—Horse is dosed with about 500 grammes of the minced leaves, dried.

6th January.—Horse soon tires on exercise and shows evidence of dull abdominal pain. It takes up some green grass, but is unable to masticate it properly and collects it in its mouth. It swings its head towards flanks, scratches ground with its left foot, and bites towards abdomen. Muscles over shoulder and chest tremble and twitch. Animal becomes very much agitated by flies and is hyperaesthetic.

Horse is now exercised to excess in the sun. It shivers and trembles violently over whole body, lower lip hangs loosely and saliva runs over it. Respirations, 80; pulse, 150, and hardly perceptible.

When animal is returned to stable, it continually bites towards the flank, strikes with the forefeet, and appears very uneasy and in great pain.

7th January.—Animal lies down, but rises on being approached; it is tucked up and does not eat. Pulse, 130; respirations, 50.

Horse is taken outside; shivers and trembles all over body. It strikes the ground with its near forefoot and bites at its flanks. When taken to the water, it will not stop drinking. It will not stand stationary, but moves about and is irritated by the noise of the halter-chain and flies. It gets excited if pulse is taken, and if it is now exercised a little in the sun considerable intensified symptoms appear. The muscles of the neck become hard and stiff, contract to form a large swelling on the one side and a deep depression on the other; head is twisted towards one side and held at a slant; the neck is arched (torticollis). Symptoms become more marked during progression. The animal becomes very much distressed, trembles and shivers all over body, and threatens to lie down; it goes down, but rises again when approached. If urged to move, it becomes very much irritated and kicks at the driver. Salivation has very much increased, head shakes loosely up and down during progression, and the tongue protrudes partly. The animal soon becomes moist with perspiration without much exertion and bites at the observer. It is now taken into a loose box, where it soon goes down. The excitement and general clonic spasms subside, and the animal is temporarily relieved.

8th January.—Animal lies down with lower lip hanging. Respirations, 40; pulse, 120 and irregular. There is evidence of struggling during night, and a large ball of unchewed grass is removed from its mouth. The symptoms are the same as on the previous day, but become more intensified on exposure and exercise. The animal is weaker and not so active, and it moves only when coerced. Abdominal pain has somewhat subsided. It goes with an unsteady and staggering gait, falls to the ground, lies on its side, and makes no attempt to rise, even when urged and assisted. It trembles like an animal suffering from strychnine poisoning, and moves all four limbs backwards and forwards. It continues to struggle, but is unable to rise; if it is lifted, it falls back to ground powerless. It becomes comatosed, clonic spasms of the muscles disappear, muscular tremors are discontinued, respirations decrease, stop, and animal dies.

Horse No. 15134 (Weight, 345 Kilos).—Admitted on 7th November, 1922, and balled with 300 grammes of the minced flowers of *C. wallichii* over a period of eleven days.

14th November.—Animal is not feeding well.

16th-18th November.—Animal is not feeding well and is losing condition rapidly.

18th November.—There is evidence of stomatitis, and an evil-smelling odour comes from the mouth. The lips are slightly swollen, and there is a supra-orbital swelling, as in horse-sickness. There is evidence of abdominal pain; horse lies down a great deal and appears very uneasy. The superficial muscles tremble and twitch, especially over forequarters. There is marked torticollis, which is well brought out by exercise. Salivation is excessive, and lips hang loosely. Pulse is thready and fast, and respirations are jerky. Symptoms become more intensified on exposure to sun, and twitchings spread from omo-brachial region over neck, abdomen, and flanks. It dies the same night.

Horse No. 15165 also, on being fed on the plant, developed symptoms resembling the cases above.

Dog No. 120.—Admitted on 8th January, 1923, and is fed on the liver of Horse No. 15079; about 1 kilogram of liver is eaten.

11*th January*.—Dog is fed on liver of Goat No. 3223, and eats only about one-third of the liver.

12*th January*.—Fed on the liver of another goat (No. 4682) that died from *C. wallichii* poisoning, and about one-half was eaten.

18*th January*.—Fed on liver of another goat (No. 5666) that died from *C. wallichii* poisoning. Only a small part is eaten.

24*th January*.—Fed on liver of goat (No. 4580) that died from *C. wallichii* poisoning. The greater part of the liver is left and is removed later from kennel.

1*st February*.—Dog is noticed to be peculiar in its habits, but appears quite well otherwise.

2*nd February*.—Dog jumps up and barks when approached. It holds its mouth open unnaturally.

3*rd February*.—Dog stands about in kennel holding its mouth wide open. Tongue is coated with a frothy saliva which drips all over floor of kennel. Lower jaw is kept hanging unnaturally and dog looks in a peculiar squinting fashion. It realizes that something is amiss and barks a queer unnatural bark. It seems quite active, trots about in kennel with head flexed right up against chest and twisted slightly to one side, with the neck arched. Muscles all over the body twitch and tremble. Pulse is increased and respirations are irregular and jerky.

After a few hours' exposure to the sun, symptoms are much more marked; torticollis is distinct. When the animal is exercised, the head is pushed in between the front limbs; the mouth is kept wide open. The muscles of the neck feel hard and stiff. The dog is very nervous and hyperaesthetic, and snaps impatiently at flies.

5*th-6th February*.—Symptoms remain the same. Dog gets very nervous when handled or disturbed. It is exercised outside and trots like a hackney, lifting its feet very high, pressing its head against its breast, and arching its neck to a greater extent than in the kennel. It gets tired very soon and seeks shaded spots, where it lies down exhausted and panting. If it is allowed to run loose, it tries to run away, but trots for a short distance and has to lie down to rest. When resting, it may either lie down or sit on its haunches with its head flexed and hanging down vertically. It now gives an occasional spasmodic bark. The animal loses condition, but appetite is fair.

8*th February*.—Dog looks better in kennel; trembling and twitching of muscles seem to have disappeared, but when led for about 100 yards spasms of muscles, torticollis, and other symptoms reappear.

9*th-16th February*.—Symptoms remain the same.

16*th-28th February*.—Dog is improving and is discharged as healthy on 28th February.

Dog No. 139.—Admitted to experiment on 8*th January*, 1923, and fed *ad libitum* on the meat of Horse No. 15079 and Goats Nos. 3223, 5666, and 4580 from 8*th January*, 1924.

8*th February*.—Dog is discharged as healthy.

Dog No. 138.—Admitted on 8*th January*, 1923, and fed on brain and spinal cord of Horse No. 15079, Goats Nos. 3223, 5666, and 4580.

8*th February*.—Dog is discharged as healthy.

Dog No. 154.—Admitted on 19*th November*, 1923, and is fed on about 2 kilograms of meat from Horse No. 15134 between 19*th* and 24*th November*.

25*th November-2nd December*.—No meat available.

3*rd*-10*th December*.—About 1 kilogram of meat from Horse No. 15165 eaten.

28*th*-30*th November*.—Does not eat its food.

1*st-2nd December*.—It leaves practically all its food (pap) untouched.

3*rd December*.—Eats meat from Horse No. 15165 readily, but does not touch the mealie-pap.

5*th December*.—It eats only portion of the meat.

6*th December*.—Off its feed completely and meat is left untouched, and dog refuses to feed even when coaxed. It walks about with mouth partly open, holding head low and skew. When exercised, mouth is held wide open, neck bent, head flexed, and salivation is excessive. It is fairly active, but when returned to kennel it lies down in a shaded spot. When examined a little later it trembles, keeps mouth wide open, with saliva dribbling on the floor; it lies down or sits up on its haunches, with well-marked torticollis. When removed from shaded spot, it returns again and again, appearing uncomfortable, dull, and listless. It is irritable and nervous, and snaps at flies, somewhat excited. It appears to squint, and pupils are slightly dilated.

7th December.—Dog lies down in corner; when called it comes up, but soon trots back again to lie down in previous position. Symptoms remain as on previous day, but torticollis and clonic spasms of muscles of neck and head even more marked, especially during exercise.

8th December.—Dog appears very hungry, and sniffs at and even chews dry bits of skin when exercised outside kennel, but it refuses to touch the poisoned meat in the kennel. On exercise, it becomes exhausted and lies down, panting a good deal. Hyperaesthesia becomes more marked. Torticollis and clonic spasms and trembling of muscles appear as before. Respirations are jerky and spasmodic.

10th-12th December.—Animal gets weaker and loses condition rapidly. When led a few yards it becomes exhausted and lies down, refusing to move even when urged. It is listless, dull, and dejected, and lies *continuously* with its mouth soiled with saliva and resting it on the floor. It refuses to eat, but when coaxed to eat it picks up bits of bread, which it drops again in a disinterested fashion.

Condition remains the same until it dies on 17th December.

Dog No. 152.—Admitted *19th November*; is fed on about 3 kilograms of liver from Horse No. 15134 over a period of six days. It receives nothing for a week, and is then fed on about 3 kilograms of liver from Horse No. 15165 over a period of six days.

10th December.—Dog appears fairly active and hungry, but has lost condition meanwhile. Nothing is noticed amiss until the dog is exercised for about half an hour, when it appears exhausted and lies down a great deal in shaded corners. It keeps mouth wide open, salivates excessively, and pants a good deal. It is left in kennel, and when observed later it lies down listless, with mouth unnaturally wide open and saliva dribbling over lower jaw. Superficial muscles tremble slightly. Dog is fairly active again after a short rest.

11th December.—Dog does not take liver readily and leaves a good deal untouched. Symptoms remain as before, but dog now appears excited and nervous, with pupils dilated, especially after exposure to sun.

12th December.—Dog is fairly active but becomes tired if led for a few minutes and trots with mouth wide open and moist with frothy saliva. Symptoms remain otherwise as before.

14th-18th December.—Symptoms remain as before, but dog has completely lost its appetite and loses condition rapidly. It remains excitable and nervous.

19th December.—It is found dead.

Dog No. 74.—Was fed on meat from Horse No. 15165, with negative results.

Fowl No. 7.—Admitted *16th November*, 1921, and fed on 42 grammes of *C. wallichii* leaves during a period of seven days.

16th November.—Fowl is apparently well in cage, is active, and not easily caught, but when driven outside it runs for about 10 yards, falls forward on its sternum, flaps its wings, vomits a slimy material and dies.

Fowl No. 14.—Admitted on *16th November* and dosed with 7 grammes of leaves over a period of six days.

22nd November.—Fowl is apparently well and normal in cage, but, on being approached, it seems stupid. There is slight inco-ordination of movements, and when driven it runs for about 10 yards, settles down slowly on ground, bends neck, and pushes head down between the legs. It flaps its wings and dies.

Fowl No. 17.—Admitted *12th November* and dosed with 7 grammes over a period of seven days.

18th November.—Hen stands in corner of pen with neck bent and head twisted to one side. When driven, there is lack of co-ordination of movements, and, when stationary, fowl cannot maintain its balance and rests on its wings to support itself, but swings forwards and backwards. When chased, it runs in circles with neck arched and in the form of a sigmoid flexure and head twisted to one side. On exercise, twist and curve in neck is intensified, and muscles of neck feel hard and stiff. Hen is unable to run away owing to inco-ordination of movements.

19th November.—Neck is more bent, and clonic spasms of muscles more marked and head is held horizontally. Fowl moves in circles and topples over on its side. It pecks at grass.

20th November.—Head is completely twisted round, so that right eye faces backwards and to the left. The fowl has completely lost control over muscles of neck, and twist in neck remains.

21st November.—Symptoms remain the same.

22nd November.—Fowl lies down on its right side, with legs stretched out, wings spread out. Neck is twisted so much that left side of head comes in contact with ground. There is now complete lack of co-ordination, and animal can hardly sit up.

23rd November.—Found dead in pen.

APPENDIX "B."

Post-mortem reports and histological examinations of some of the animals that showed typical symptoms of krimpsiekte and died as the result of the disease.

GOAT No. 3223.—Full-mouth, condition good, abdomen not distended. Integument ruffled. The natural openings and visible mucous membranes show nothing unusual. The flesh is light brown in colour and somewhat opaque in appearance. The blood is dark red and semi-coagulated. The peritoneal cavity, pleural cavities, salivary glands, lymphatic glands, and thyroid glands show nothing unusual. The diaphragm is somewhat dull in appearance.

The mouth is open.

The mucous membrane of the oesophagus is pale and covered with some particles of ingesta. The lips are wet and soiled.

The pharynx is light red in colour. The mucosa of the larynx is also light red, but is covered with some mucus. The tracheal mucosa is light red, so is the mucosa of bronchi, and is covered with some mucus.

The right lung is smooth and glistening on the surface. Colour varies from light red to purplish red. On section, cut surface is light purplish red and cut edges are sharp. Margo acutus is sharp. The left lung resembles the right, but there is a dark red area on main lobe; this extends a small distance into pulmonary tissue. It appears solid.

The pericardium contains about 8 c.c. of liquid, which is tinged red.

The Circulatory Organs.—There is a fair amount of fat in the cardiac sulci. The epicardium varies in colour from light brownish slaty-blue to reddish brown. The right ventricle contains a large dark red blood-clot, which is continuous with a similar one in the atrium. Endocardium is of a light reddish brown colour. The myocardium is somewhat opaque and of a dull reddish brown colour. The valvulae are normal. The left ventricle is contracted and only the atrium shows a blood clot. On each bicuspid valve there is a dark red area 2 m.m. in diameter.

Liver.—Hepatic lymph glands are apparently normal. The liver is swollen; its colour varies from yellowish brown to light purplish red. The surface is smooth and glistening. On section, cut surface is moist with liquid blood; cut edges are rounded. Lobulation can be made out; areas at periphery of lobule are light brownish grey, and around central vein brownish red and dark in colour. Consistency is fairly firm. The gall-bladder is distended with a greenish viscid bile.

The pancreas is apparently normal.

The Spleen.—16.9.2 cm., soft, glistening on its surface, pulpa is of a dark red coloration.

The splenic corpuscles are small and not very prominent; trabeculae are not too distinct.

Adrenal Glands are apparently normal.

Kidneys.—There is a good deal of perirenal fat. The capsule comes off easily. The right kidney varies on its surface from yellowish brown to brownish red in colour. On section, the zones can be made out distinctly. The cortex is light yellowish brown. Medulla is darker in colour. The consistency is firm. Left like right.

Stomachs.—Rumen contains a good deal of semi-solid ingesta with some froth. The mucosa is normal. The reticulum is normal. The omasum contains some fairly hard pasty ingesta. The abomasum contains some greenish liquid ingesta. Mucosa is swollen and light pink in colour.

Small Intestine.—At beginning mucosa is swollen, red in colour, and contains a dirty blood-tinged mucoid ingesta. Jejunum and ileum are less tinged; contents are of a dirty reddish grey colour.

Colon and caecum mucosa are slightly red, and contents are of a dirty grey blood-stained material. Rectum mucosa is covered with mucus.

Mesentery is normal.

The bladder is contracted and empty. Sexual organs are normal.

Histology.

Liver.—The hepathic cells are infiltrated with droplets of fat, which vary in size in the different cells. All the liver cells contain a certain amount of fat. The fat may distend the cell and take up the greater part of the cytoplasm and so push the nucleus towards one side. The spaces between the columns of liver cells are distended with blood. The nuclei stain well, showing a fair amount of chromatin and a distinct nucleolus.

Kidney.—The renal cells stain well. Some nuclei are somewhat larger than others. The smaller ones stain deeper. There is an increase in round cells in the renal corpuscle. The capillaries are filled with blood.

Heart.—At the extremities of the nuclei are found clusters of brown pigment. The fibres stain well.

Striped Muscle.—The fibres of the masseter musle show an increase in their nuclei—these are arranged in long strings. There are also seen clusters of round cells at different spots.

Adrenal Glands show nothing unusual.

Spleen shows nothing abnormal.

Pathological Diagnosis.—Fatty infiltration of liver. Slight pyknosis of renal epithelial cells. Brown atrophy of heart. Stasis of liver and kidney.

Etiological Diagnosis.—Krimpsiekte.

Goat No. 5978.—Full-mouthed ewe; condition fair. Mouth is open and lips are wet. Rigor mortis not present. Natural openings, cyanotic; visible mucous membrane, cyanotic. Flesh and blood, dark red; subcutaneous tissues, congested and moist. Pleural and peritoneal cavities, normal. Salivary, thyroid, and lymphatic glands, normal. Pharynx, cyanotic. Trachea, dark red. Lungs, moist and dark red. Collapsed.

Heart.—Ventricles contain dark red blood-clots. Epicardium shows dark red spots and ecchymoses. Fat, discoloured red.

Liver.—Pale yellowish brown; somewhat friable; lobulation evident.

Spleen.—Normal in size; well-marked pulpa.

Kidneys.—Small amount of perirenal fat. Capsula comes off easily. Zones can be made out distinctly. Outer zone, palish red; inner, dark red. Consistency, fairly firm.

Stomach rumen distended with gas and ingesta. Reticulum omasum abomasum show nothing unusual.

Intestines.—Hyperaemic patches on mucous membrane.

Histology.

Striped Muscle.—Mm. brachiocephalicus, pterygoideus, longissimus capitis and Hyoglossus contain small droplets of fat between the fibrillae of about 10 per cent. of the fibres. The nuclei stain well. Striation remains distinct. There is a slight engorgement of the capillaries.

Crus of diaphragm and Mm. masseter and splenius contain fat in very fine droplets in about 20 per cent. of their fibres.

Mm. triceps, semimenbranosus, biceps femoris longissimus dorsi, rectus capitis ventralis major, psoas major do not show any presence of fat.

Myocardium.—A few of the fibres contain very small droplets of fat aggregated here and there. At the extremities of the nuclei are found clusters of brown pigment. A few sarcosporidia are present. The vessels are engorged, and there is a slight increase in round cells at various spots. The nuclei stain well and show up all the nuclear elements.

Kidney.—Autolysis has already commenced, so that the nuclei are in various stages of decomposition. The capsule shows an increase in round cells. The epithelial cells of many tubules show droplets of fat of varying sizes. There are found also bacteria around the cells; these are apparently putrefactive organisms. The capillaries are somewhat distended with blood.

Liver.—The intercellular spaces are distended with blood. The nuclei also here show evidence of autolysis, and the various stages in decomposition can be made out. There are also found putrefactive organisms between the hepatic cells. A fair number of the liver cells contain droplets of fat of varying size.

Pathological Anatomy.—Fatty infiltration of many striped muscle, and also to some extent the heart, liver, and kidney. Stasis of myocardium, liver, kidney. Autolysis of liver and kidney. Hyperaemia of small intestinal mucous membrane.

Etiological Diagnosis.—Krimpsiekte.

Dog No. 154.—Brown bitch, aged. Rigor mortis not present. Integument covered with ticks and fleas. Flesh dark reddish brown. Subcutaneous tissues show a fair amount of fat. Peritoneal and pleural cavities have a smooth and shining serosa. There is a fair amount of fluid present.

Diaphragm, salivary glands, lymphatic glands, tongue pharynx, oesophagus, and larynx appear normal. Tracheal vessels somewhat injected.

Lungs are collapsed, smooth, and glistening on the surface. Colour varies from light red to pinkish purple. On section, some blood escapes from the cut surface. Cut edges are sharp. Firm and elastic is the consistency of the lungs.

Pericardium contains a small amount of fluid. Epicardium varies in colour from light reddish brown to light red. Endocardium and valves, light reddish brown.

Myocardium.—Vessels are dilated and stand out prominently. At apex there are a few hyperaemic areas; for the rest myocardium is brownish red in colour, opaque, with greyish areas here and there.

Liver.—Light reddish brown, with numerous light grey areas here and there, especially in vicinity of hilus. The lobulation can be made out partly, the peripheral part being somewhat lighter than centre. Consistency somewhat friable.

Spleen.—Light red and somewhat atrophied. On section, pulpa is light red and trabeculae are well marked, but splenic corpuscles can be made out only with difficulty.

Stomach contains only a small amount of ingesta, which is mucoid.

Intestines.—There are a few red patches on the mucous membranes. Contents are mucoid and bile-stained.

Mesentery contains some fat.

Kidneys.—There is very little perirenal fat. Boundary zone is distinct; medulla is red, and cortex brownish grey and somewhat pale. Consistency firm. Bladder is distended with clear yellowish urine, and the sexual organs show nothing unusual.

Histology.

Myocardium.—At the extremities of some nuclei there are clusters of brown pigment with Scharlach the fibres stain of a light brown colour.

Spleen.—Apparently normal.

Kidney.—The epithelial cells, lining a large number of tubules, contain fairly large droplets of fat, which in some cases may displace the cytoplasm partly or entirely. Some of tubules lying adjacent to these are not affected in the least and appear quite normal.

In Haemalaum Eosin stained sections, the cytoplasm of the affected cells appears light and vacuolated, whereas the cells that are apparently not affected stain deeper. Some nuclei stain well, showing a distinct reticulum of chromatin and a definite nucleolus, whereas others stain darker (Pyknosis), are more or less homogeneous. These nuclei are also smaller that the reticular ones. In the vacuolated cells the nuclei are irregular in shape and the nuclear elements cannot be made out. In some cells no nucleus at all can be made out (Karyolysis).

Liver.—The trabeculae of hepatic cells are irregular and are separated by wide spaces filled with blood. The cells contain droplets of fat which in some cases may be very large and push the nucleus towards the periphery. The chromatin network and nucleolus can be made out distinctly in the nuclei of some cells, whereas in others the nucleus is one homogeneous dark staining mass (Pyknosis), and smaller than in the other cases. In other cells, again, there is no evidence of a nucleus, the cells being composed of fat and a small amount of cytoplasm only (Karyolysis). The nuclear changes are probably due to autolysis, as an occasional bacterium, which resembles a putrefactive organism, can be made out. Some cells contain brown pigment and others iron.

Striped Muscle.—M. masseter, m. lingualis, m. longus colli, m. brachio cephalicus, and diaphragm contain a variable amount of very fine small fat droplets between the fibrillae of the fibres. The striation remains distinct and the nuclei stain well, bringing out all the nuclear elements. The capillaries are engorged. M. splenius, m. biceps femoris are apparently normal.

Myocardium.—There are culsters of brown pigment at the extremities of the nuclei, which stain well. The capillaries are engorged.

Pathological Anatomy.—Fatty infiltration of striped muscle, liver, and kidney. Degeneration of liver and kidney. Brown atrophy of heart muscle. Hyperaemia of intestinal mucous membrane.

Etiological Diagnosis.—Krimpsiekte.

HORSE No. 15079.—Nine-year-old brown mare. Condition fair. Rigor mortis present. Visible mucous membranes yellowish brown in colour. Mouth is open and lower lip hanging. Sphincter ani is partially relaxed. Blood is dark red and semi-coagulated; flesh is of a dull yellowish and brownish red colour. Fat has a yellow tinge. Pleural and peritoneal cavities show nothing abnormal. Salivary glands are of a light pinkish colour. Lymphatic glands appear normal.

Respiratory Organs.—Mucosa of the pharynx is yellowish pink. Mucosa of larynx is also pinkish yellow and is covered with some dirty mucus. Mediastinal fat is yellow and gelatinous. The right lung is collapsed, smooth, and glistening on its surface. Its colour varies from light reddish purple to yellowish red. On section, cut surface is of a yellowish light red colour. Some blood escapes and cut edges are sharp. Mucosa of bronchi and trachea is yellowish pink and covered with yellow mucus. Pulmonary vessels show nothing abnormal. The left lung resembles the right.

The pericardium contains 90 c.c. of straw-coloured fluid. The epicardial fat is yellowish pink, and is studded with a number of red spots, varying in size from a pin-point to a pin's head. Towards apex and on left longitudinal sulcus there is a large diffuse red area. The epicardium varies in colour from light brown to a reddish slaty blue. Right ventricle is nearly empty and endocardium over musculi papillares is infiltrated with a yellowish gelatinous material. Right atrium is also nearly empty. The right heart contains a small dark red blood-clot. Valvulae are not affected. Myocardium is dull, opaque, reddish brown, with irregular streaks of red. Left endocardium has a colour varying from bluish purple to reddish brown. There are a number of reddish purple streaks extending from apex to base of heart. On cutting into endocardium, red areas extend underneath endocardium. There is present a small dark red blood-clot.

The hepatic lymph glands are normal.

The Liver.—Size normal; fibrous filiments appear on diaphragmatic surface. The colour varies from bluish brown to light brown reddish, with a distinct yellow tinge. On section, a good deal of liquid blood escapes; lobulation can be made out fairly distinctly; peripheral areas are lighter in colour than those around central vein. Consistency is fairly firm.

The Pancreas is light brownish yellow in colour.

The Spleen, 26.18.3 cm., is contracted and feels hard. It is smooth on its surface and on section trabeculae are prominent, but plenic corpuscles cannot be made out easily. Pulpa is dark reddish brown.

Adrenal Bodies are apparently normal.

Stomach.—Ingesta of a liquid nature.

Some gastrophilus larvae are present. Cuticular mucosa is light pink in colour. The glandular mucosa varies in colour from a dirty grey to a dirty yellowish pink colour and is covered with a yellowish mucus.

Intestines.—Mucous membranes show red patches and are slightly swollen and covered by a yellowish pink mucoid material. The mucosa of caecum is pinkish grey and is covered with some yellowish pink ingesta. The rectum contains some dirty mucus and a few hard balls of faeces.

Kidneys.—There is a good deal of perirenal fat. The capsule comes off easily. The cortex is yellowish brown with a number of red points. The medulla is reddish brown. Consistency firm.

Bladder.—Contracted and empty; mucosa, pale pink.

Sexual Organs.—The right ovary contains a few cysts varying in size from a pea to a large gooseberry. On section, a yellowish fluid oozes out. Left ovary is very fibrous on section and has a large dark red core. The genital tract shows nothing abnormal.

Nervous System shows no abnormalities.

Bone-marrow.—There is a good deal of yellow marrow surrounded by red marrow at the extremities.

Histology.

Liver.—In Scharlach-stained sections, groups of hepatic cells contain droplets of fat of varying size. The cells along periphery of lobule particularly are involved. The droplets in some cells are so large that the cytoplasm is considerably reduced in amount and the nucleus occupies an eccentric position. In some cells only very fine granular globules of fat can be made out. The spaces between the columns of cells are distended with blood. In some places along periphery of some lobules there are seen aggregations of round cells. The nuclei show up well and contain a good deal of chromatin. The nucleolus can easily be seen. Occasionally a nucleus is come across which is lobulated more or less and which stains darker than the rest.

Kidney.—The cells lining the tubules, especially the tubuli contorti, contain fat in varying amounts. The fat may be in the form of a number of small globules or one or more large droplets. The nuclei stain well, nucleoli are distinct, and chromatin is present in fair amounts. The intertubular capillaries are distended with blood. There is an increase of round cells at a few places. The amount of blood around renal capsule is also increased in amount.

Heart.—In Scharlach-stained sections there can be made out in some fibres a large number of brown sandy granules which give the fibre a brownish turbid appearance, and which are probably very small globules of fat. The fibres which contain the fat droplets stain darker than the rest. The nuclei stain well.

Striped Muscle (M. masster).—In Scharlach-stained sections some fibres stain distinctly brown, due to the presence of numerous very fine sandy droplets of fat. The fibres appear to be distinctly more brown in some patches than in others. The boundaries of the affected areas are well marked off. Some fibres are quite clear and are free from fat. In some cases, a particular fibre is distinctly brown in colour and filled, as it were, with fine droplets of fat, whereas an adjacent fibre is quite clear and apparently not affected in the least. The striation is quite distinct, both transversely and longitudinally. The nuclei stain well, but appear to be increased in numbers. The fibres appear to vary in thickness more than usual. The capillaries are distended with blood.

Pathological Anatomical Diagnosis.—Subendocardial haemorrhages; petechiae on the epicardium. Slight gastro-enteritis. Cyst formation and fibrosis of ovary. Fatty infiltration of liver, striped muscles, kidney, and, to some extent, the myocardium also. There is stasis in liver, kidney, and myocardium. Slight atrophy of striped muscle.

Etiological Diagnosis.—Krimpsiekte.

Horse No. 15134.—Six-year-old chestnut mare. Condition poor. Rigor mortis present, but not complete. Lips are swollen, dirty, and moist. Natural openings show nothing abnormal. Visible mucous membranes are normal. Blood is fluid and semi-coagulated. Flesh reddish brown, dull, and moist. Subcutaneous tissues pale. Peritoneal and pleural cavities have nothing abnormal. Diaphragm is apparently normal. Tongue, salivary glands, and lymphatic glands show nothing unusual. The mucosa of pharynx is pale, oesophagus has a pale mucous membrane, but thoracic part shows red longitudinal striation where epithelium is irregularly denuded. The mucous membrane of larynx and trachea is somewhat pale. The lungs are collapsed, smooth, and glistening on the surface. Colour varies from light greyish red to light purplish red. On section, cut surface is dry, cut edges sharp, and colour light brownish red. The bronchial mucosa is also pale. Pericardial sac contains 50 c.c. of clear serous fluid. The epicardium is distinctly pale in colour. Right ventricle contains some coagulated and some liquid dark red blood, continuous with similar blood in the atrium. Ventricle is partly contracted. Left ventricle is contracted and contains similar blood as right. The endocardium of left ventricle only contains small dark red areas. It is light pinkish brown in colour for the rest. Myocardium dull and opaque, but paler than usual. Consistency fairly firm.

Liver.—Apparently of normal size. Borders are sharp. Visceral surface is smooth; perietal surface shows some fibrous filiments. The colour is reddish brown. On section, a good deal of liquid blood escapes. Lobulation can be made out. The periphery of lobule is of a light greyish brown colour, whereas central part is reddish brown. Consistency not very firm.

Pancreas.—Light pinkish brown.

Kidneys.—Very small amount of perirenal fat. Capsule is easily removed. Blood escapes from surface on section. Cortical zone pale brown, with a number of red spots. Consistency firm. The medulla is reddish brown.

Stomach.—Cuticular part contains some gastrophilus larvae. Mucous membrane of glandular portion is slightly swollen and of a dirty greyish pink colour. A few small ulcers are present on the mucosa.

Small Intestines.—There are a number of small red patches on the mucous membrane at various intervals. The mucosa is somewhat swollen. Contents yellowish pink, soft, and semi-liquid.

Large Intestine.—Mucosa is greyish pink in colour and ingesta liquid.

Brain and Cord apparently normal.

Histology.

Heart.—In Scharlach-stained sections the fibres appear brown. The brown coloration is marked in patches. The brown colour is due to numerous very small brown intracellular globules of fat. These are arranged irregularly, but clumped at certain parts where the brown coloration is distinctly intensified. The striation remains distinct, nuclei stain well, and chromatin shows up well. The capillaries between the fibres are distended with blood.

Salivary Glands.—Some of the cells lining some of the alveoli and tubules contain droplets of fat. These are larger in some cases than in others. The nuclei stain well and show a good deal of chromatin and distinct nucleoli.

Kidney.—The epithelial cells of the renal tubules contain fat droplets, varying in size and amount in the different tubules. In some cases the nuclei stain darker and are smaller than in others (Pyknosis). Some cells even may be without nuclei (Karyolysis). The capillaries between the tubules are distended with blood.

Adrenal.—The cytoplasm of all the cortical cells contains droplets of fat in an abnormal amount.

Thyroid.—Some of the cubical epithelial cells of the thyroid vesicle contain small globules of fat. The nuclei stain well and show all the nuclear elements.

Liver.—The radiating columns of liver cells are separated by wide spaces, which are distended with blood. Large masses of hepatic cells, especially along periphery of lobule, contain droplets of fat of varying size. The deeper cells contain less fat and some may even be entirely devoid of fat. The nuclei stain well and show well-marked nucleoli and distinct networks of chromatin.

Striped Muscle.

M. Serratus Ventralis.—The fibres stain brown when stained with Scharlach. In patches the brown coloration is very distinct—these patches end abruptly. The brown coloration is due to the presence of numerous very small droplets of fat arranged longitudinally and apparently between the longitudinal fibrillae. Some fibres stain deeply brown on account of the large infiltration with fat, whereas other fibres adjacent to these may be quite clear and free from fat. Consequently, a Scharlach-stained section may appear to be made up of alternating brown and light blue fibres. In some cases the fibre may be more or less distended with droplets of fat. In all cases the striation, both longitudinal and transverse, remains distinct. There is an increase in nuclei, but each nucleus stains well, and all the nuclear elements are made out distinctly. The capillaries are engorged. Close on 70 per cent. of the fibres contain fat, some to a lesser degree than others. Some muscle fibres are very much smaller and thinner than others.

M. Rectus Capitus.—Like serratus.

M. Splenius.—About 50 per cent. of the fibres are affected. The affected fibres contain more fat and large droplets, so that the contrast between non-affected and affected fibres is more marked.

M. Triceps.—As above, but fibres are less involved.

Mm. Complexus and Gluteus.—Show only a small amount of fat in their fibres.

Pathological Anatomy.—Atrophy of spleen. A small amount of anaemia; degeneration of myocardium, liver, and kidney. Fatty infiltration of myocardium, striped muscle, liver, kidney, salivary glands, adrenals, and thyroid. Stasis of myocardium, liver, kidney, and striped muscle. Atrophy of striped muscle. Hyperaemia of intestinal mucous membrane, gastro-enteritis.

Etiological Diagnosis.—Krimpsiekte.

HORSE No. 15165.—Chestnut mare. Condition very poor. Abdomen sunken—interim immediately after death. Integument shows some bruises. Blood is liquid and dark red. Flesh of a light red colour. The subcutaneous tissues show a very small amount of fat. Peritoneal and pleural cavities contain the normal amount of fluids.

Salivary, thyroid, and lymphatic glands are normal. The pharynx is pale; oesophageal mucous membrane is pale. Trachea blanched. The lungs are collapsed; pleura smooth; lungs elastic on section and of a light purplish red colour. Bronchus-like trachea. Pulmonary vessels are normal.

Pericardium contains about 50 c.c. of yellowish fluid. The epicardium contains a very small amount of fat in the cardiac grooves.

Ventricles are both contracted; endocardium and the valves are normal. Myocardium soft, opaque, and greyish red.

Liver.—Small in size; some fibrous filiments adherent to capsule. On section, a good deal of blood escapes; lobulation is distinct. Colour light brownish red. The pancreas shows a few calcareous nodules and is of a light pinkish yellow colour.

Spleen.—Athophy, fibrous filiments adherent to capsule. Light red on section; corpuscles can be made out. Trabeculae distinct.

Suprarenal Glands.—Somewhat pale.

Kidney.—Capsule contains very little fat; on section, kidney is pale and even boundary zone is light brown; consistency somewhat firm. Capsule easily detached.

Stomach.—Mucosa, pale; glandular portion, grey.

Intestines.—Mucous membrane swollen and red in patches.

Nervous System.—Normal.

Histology.

Striped Muscle.

M. Serratus Ventralis.—The striation, both longitudinal and transverse, distinct. The number of nuclei of sarcolemma are increased. They may be arranged in clusters or in long rows like beads.

In Scharlach-stained sections some fibres show numerous droplets of fat, arranged longitudinally between the fibrillae. In some fibres the droplets are large and more numerous than in others. The part of the fibre involved sometimes ends abruptly, and there is a distinct line of demarcation between an affected part and a non-affected part. Some fibres again are not affected in the least, and they alternate with the affected ones, giving the section the appearance of alternating brown and light blue streaks. At some places the fatty fibres appear to bulge out. The capillaries between the fibres are distended with blood. About 60 per cent. of the fibres contain fat.

Diaphragm.—As above. The fat droplets can be seen arranged in bead-like fashion between the fibrillae.

Mm. Splenius.—Pterygoideus as before.

Mm. Rectus Capitus.—Ventralis. Trapezius as before, but the fibres contain less fat.

Mm. Omo-hyoideus.—Brachiocephalicus gastrocnemius, longissimus capitis, flexor digitalis superficialis, sterno-mandibularis, crico-pharangeus, triceps, extensor digitalis communis, intertransversalis cervicalis contain a variable amount of fat, but a good deal less than the diaphragm and m. serratus.

Mm. Biceps Femoris and Extensor digitalis Longus show no fat.

M. Masseter contains a very large amount of fat; about 90 per cent. of fibres are markedly involved, giving the whole section a distinct brown colour.

Adrenal and Thyroid Glands.—Normal.

Heart.—The capillaries are distended with blood. At the extremities of the nuclei are found patches of brown pigment. The nuclei stain well and show all the nuclear elements. The striation remains distinct.

Kidney.—The epithelial cells lining the tubules are somewhat denuded, and portions of cytoplasm appear in the lumen. Some nuclei are large and stain well, showing all the nuclear elements, others are smaller and stain dark, and the nuclear elements are not distinct (Pyknosis). Some cells again are devoid of nuclei (Karyolysis). Some of the affected tubules stand out well, and the epithelial cells stain deeper. There is distinct pyknosis and karyolysis, and the lumen of the tubule is partly filled with desquamated portions of the cytoplasm. The interstitial capillaries are distended with blood. The renal corpuscles show an increase of erythrocytes and round cells. Some of the epithelial cells contain small droplets of fat.

Spleen.—Normal.

N. Ischiaticus.—Normal.

N. Facialis.—Normal.

Sections of cervical spinal cord and medulla oblongata appear normal.

Liver.—The spaces between the trabeculae of liver cells are distended with blood. The liver cells contain small droplets of fat, but a large number of cells are free from fat. The nuclei stain well in some cases and show all the nuclear elements, but some stain darker and are smaller (Pyknosis). In some cases, too, the cytoplasm stains lighter.

Pathological Anatomy.—Degeneration of the kidneys, liver, and hyperaemia of the mucous membrane of the intestines. Fatty infiltration of the striped musculature; in particular certain muscles and group of muscles. Brown atrophy of heart. Stasis of liver, kidney, myocardium, and striped muscle. Atrophy of striped muscle. Fatty infiltration of liver, kidney, and muscle.

Etiological Diagnosis.—Krimpsiekte.

REFERENCES.

(1) Report of Cattle Diseases Commission, 1877, page 17.
(2) Agricultural Almanac (Cape), 1887, page 111.
(3) Cattle Commission Report, 1887.
(4) Report of the Colonial Veterinary Surgeon, 1882, page 6.
(5) Report of the Colonial Veterinary Surgeon, 1884, page 29.
(6) *Cape Agricultural Journal*, Vol. III, No. 15, page 140.
(7) *Cape Agricultural Journal*, Vol. XIV, No. 13, page 862.
(8) Report of the Director of Veterinary Research, Vol. III, page 387.
(9), (10), and (11) Private communication.

(12) Hutcheon: *Cape Agricultural Journal*, 1904, page 424.
(13) Viljoen: Seventh and Eighth Reports of the Director of Veterinary Research.
(14) *Cape Agricultural Journal*, Vol. XI, page 302.
(15) Sydney Dodd and Max Henri: *Journal of Comp. Path. and Therap.*, Vol. XXXV, page 41.
(16) Curtis and Kauff: *Journal of Comp. Path. and Therap.*, Vol. XXXI, page 51.
(17) Otto Henning: *Cape Agricultural Journal*, Vol. XXV, No. 4, page 398.

EXPLANATION OF FIGURES 1-33.

Fig. 1.—Sketch of *Cotyledon wallichii*.
Figs. 2-5.—Types of *C. wallichii*: veld, Prince Albert.
Figs. 6, 7, 12, 16.—Goats showing symptoms of krimpsiekte. Experimental cases.
Figs. 8, 9, 10, 11, 13.—Dogs showing symtoms of krimpsiekte. Experimental cases.
Figs. 14, 15, 18-24.—Horse showing symptoms of krimpsiekte. Experimental cases.
Fig. 17.—Fowl showing symptoms of krimpsiekte. Experimental case.
Fig. 25.—Microscopical section of diaphragm of a horse; a case of krimpsiekte; stained with Sudan III; fatty infiltration of many muscle fibres.
Figs. 26, 27, 32.—Microscopical section of skeletal muscles of a horse; a case of krimpsiekte; stained with Sudan III; fatty infiltration of many muscle fibres.
Figs. 28, 31.—Microscopical section of myocardium of a horse; a case of krimpsiekte; stained with Sudan III; fatty infiltration of many muscle fibres.
Figs. 29, 33.—Microscopical section of a liver, goat; a case of krimpsiekte; stained with Sudan III; advanced fatty infiltration of liver cells.
Fig. 30.—Microscopical section of a kidney, horse; a case of krimpsiekte; stained with Sudan III; fatty infiltration of *tubuli contorti*.

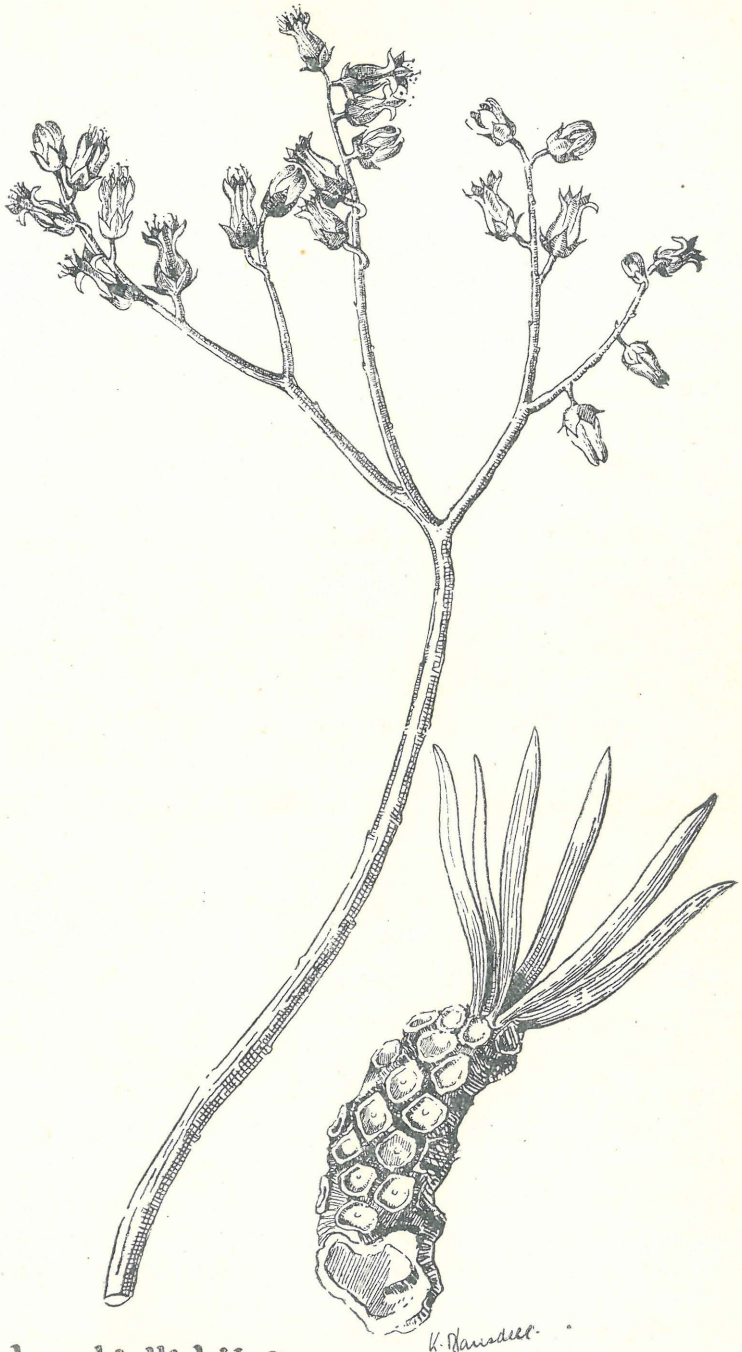


Fig. 1.

KRIMPSIEKTE. [*M. W. Henning.*

Fig. 2.

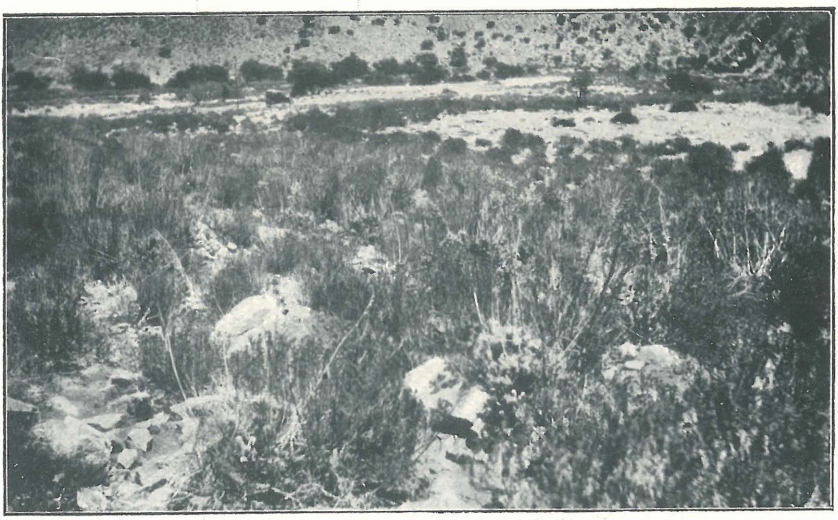


Fig. 3. Photo by M. B. Luttig, Prince Albert
C. Wallichii Veld.

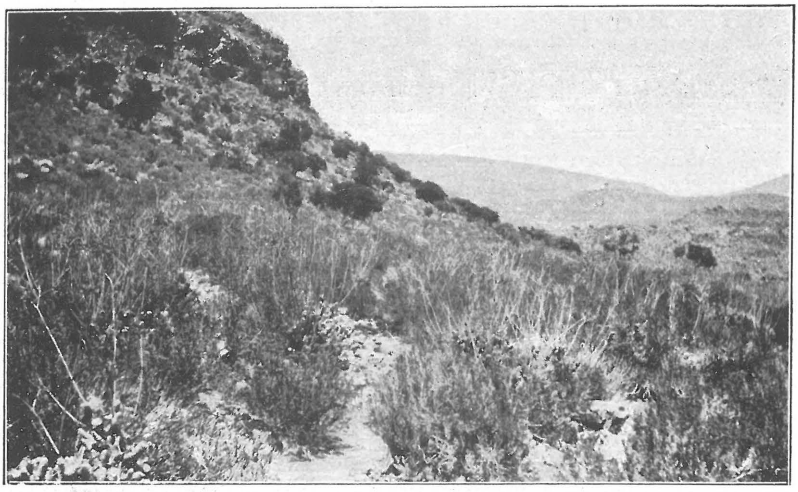


Fig. 4.

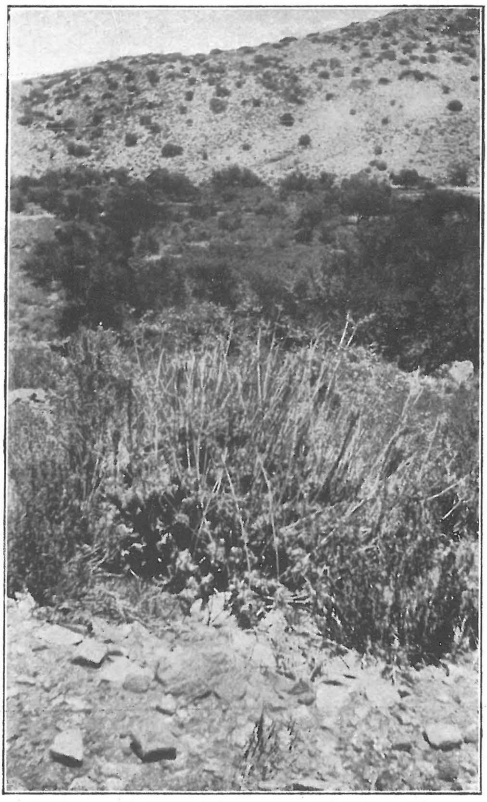


Fig. 5. Photo by M. B. LUTTIG, Prince Albert

C. Wallichii Veld.

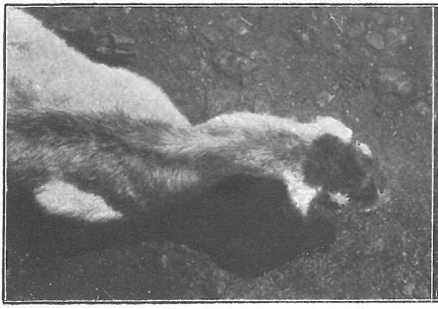


Fig. 6.
Goat 3223—10/1/23.

Fig. 7.
Goat 3223—9/1/23.

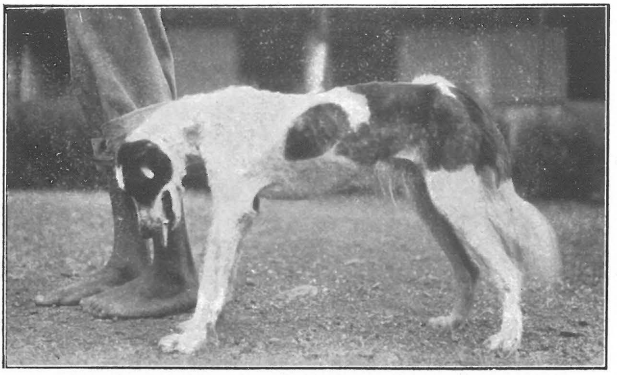


Fig. 8.
Dog 120—8/2/23.

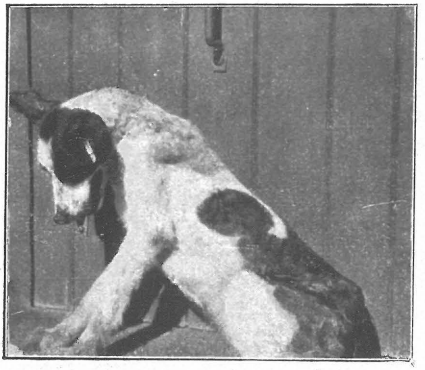


Fig. 9.
Dog 120—4/2/23.

Fig. 10.
Dog 154—10/12/23.

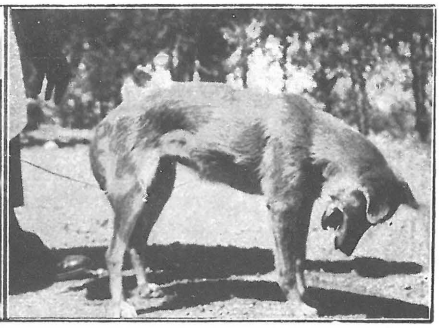


Fig. 11.
Dog 154—10/12/23.

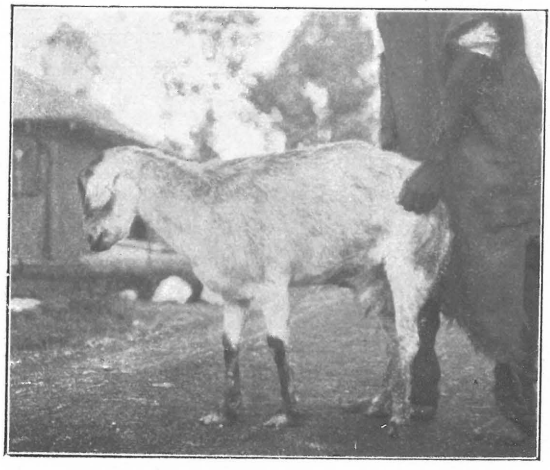


Fig. 12.
Goat 3223—25/10/22.

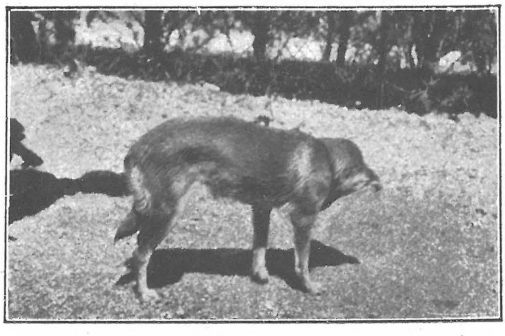


Fig 13.
Dog 154—10/12/23.

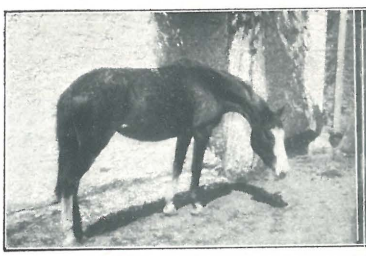


Fig 14.
Horse, Grahamstown.

Fig. 15.
Horse I, Grahamstown.

Fig. 16.
Goat 47, Grahamstown.

Fig. 17.
Fowl 17, Grahamstown.

Fig. 18.
Horse 15529, 7/1/23.

Fig. 19.
Horse I, Grahamstown.

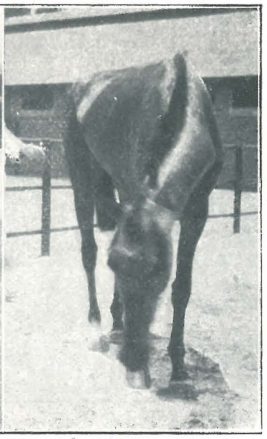


Fig. 20.
Horse 15529, 7/1/23.

Krimpsiekte.] [*M. W. Henning.*

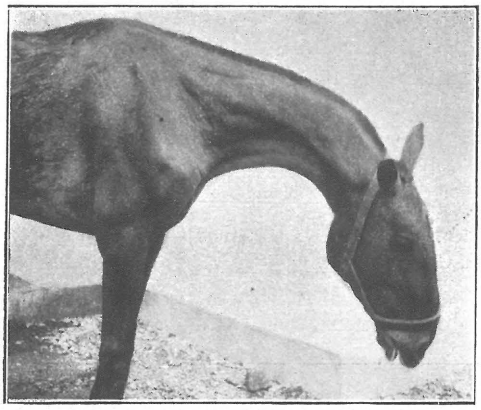


Fig. 21. Horse 15134—17/11/23.

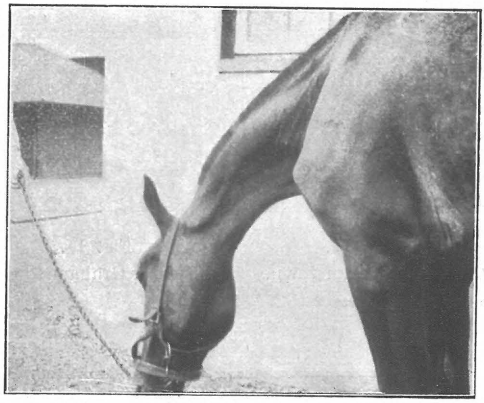


Fig. 22. Horse 15134—17/11/23.

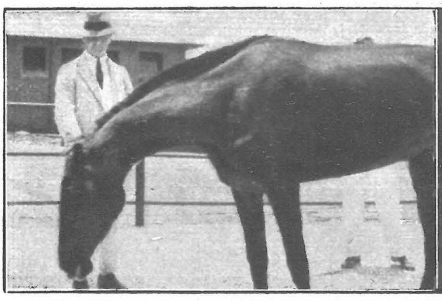


Fig. 23.
Horse 15529—7/1/23.

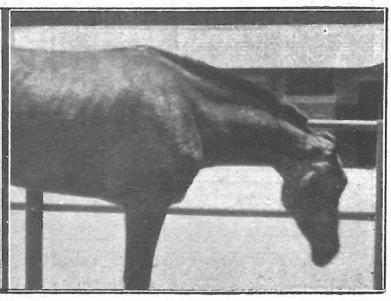


Fig. 24.
Horse 15529—7/1/23.

Krimpsiekte.] [*M. W. Henning.*

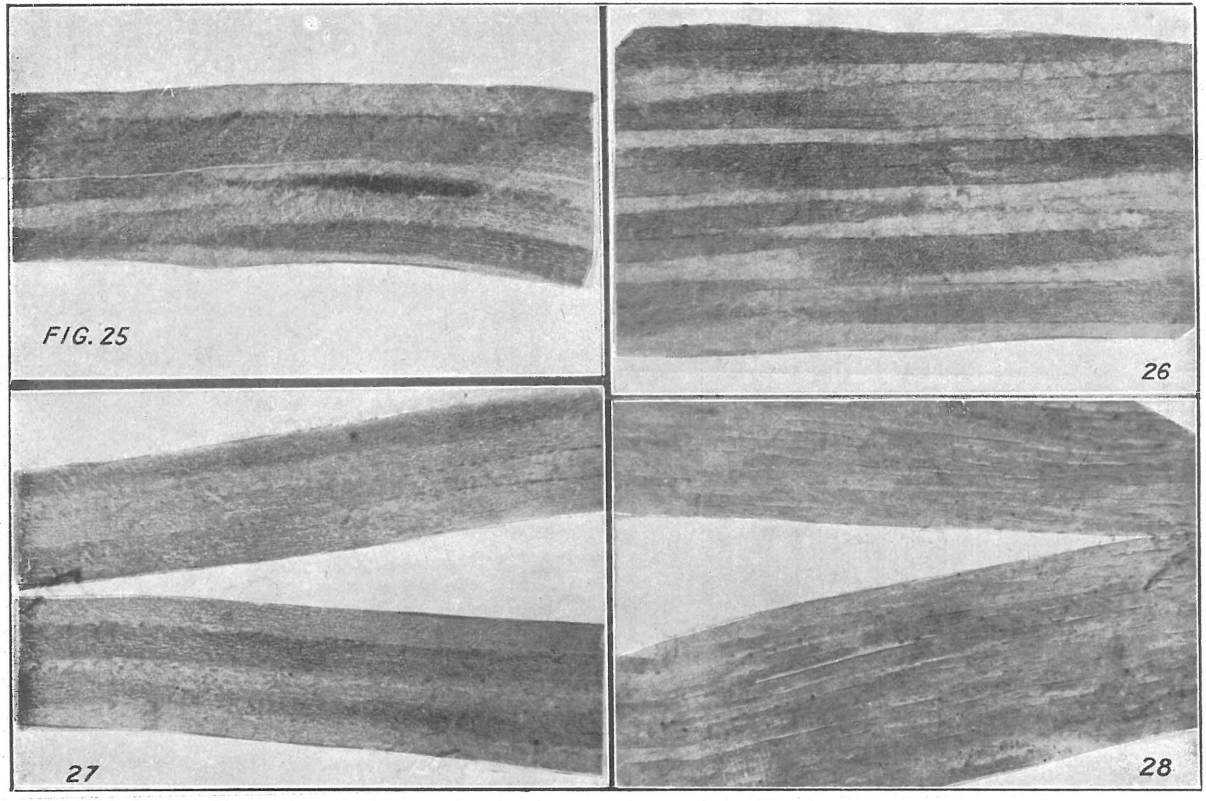


Fig. 25—Horse 15165, Diaphragm Magnif. 80 ×.
„ 26— „ 15165, M. Splenids „ 80 ×.
„ 27— „ 15165, M. Pterijgoidens „ 80 ×.
„ 28— „ 15134, Myscardium „ 80 ×.

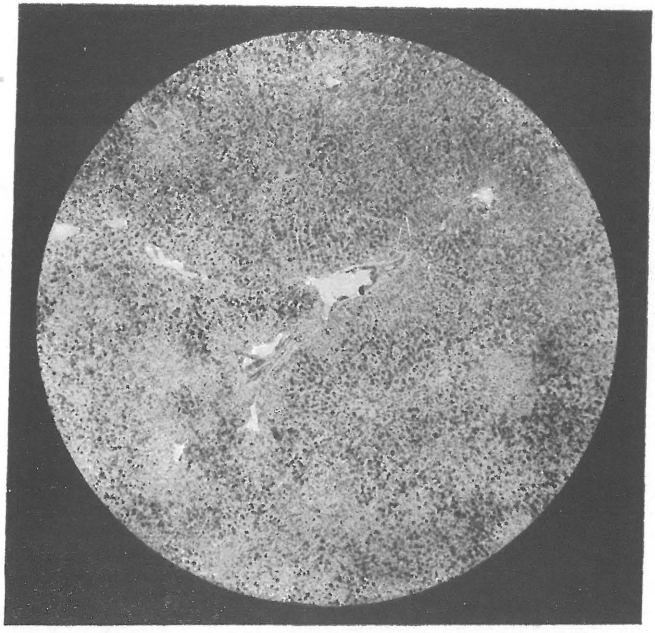


Fig. 29.
Goat 3223 liver. Magnif. 25 ×.

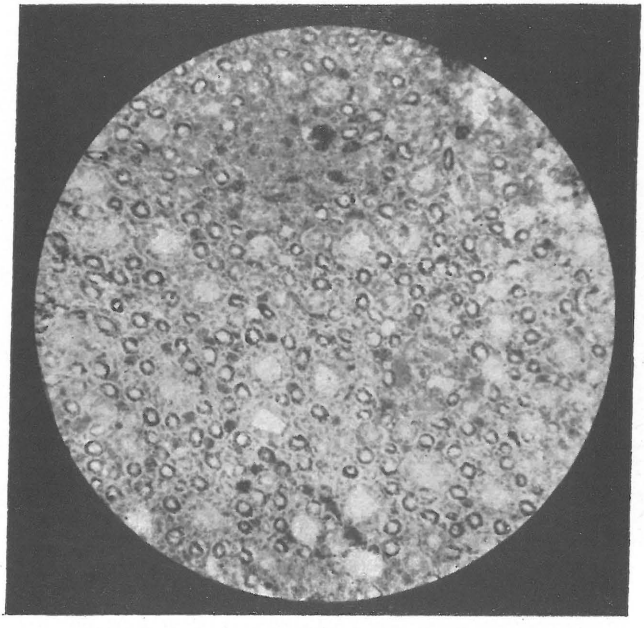
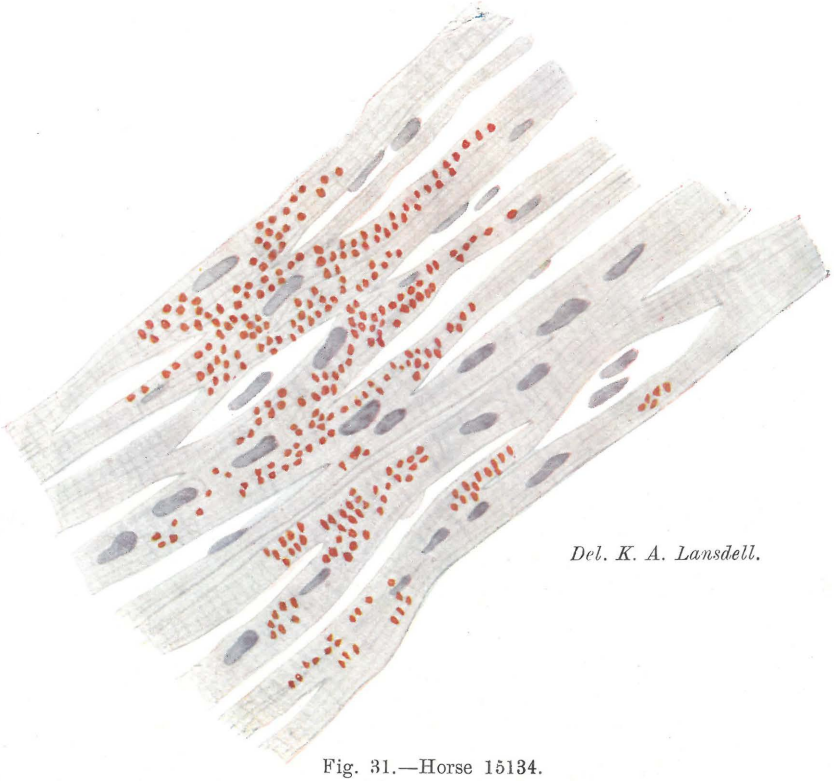
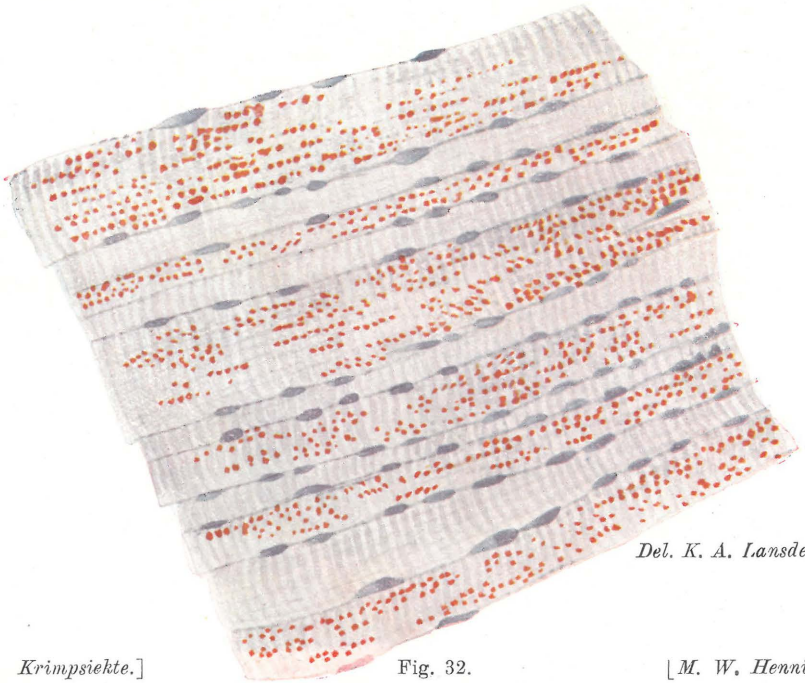


Fig. 30.
Horse kidney. Magnif. 80 ×.



Del. K. A. Lansdell.

Fig. 31.—Horse 15134.



Del. K. A. Lansdell.

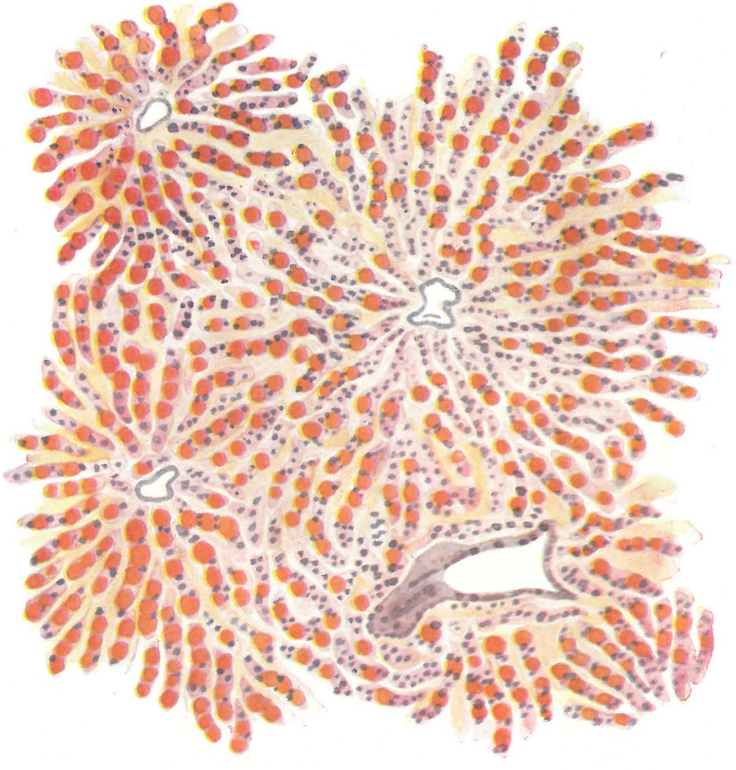


Fig. 33.

Krimpsiekte.] [*M. W. Henning.*