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CIRRHOSIS OF THE LIVER IN STOCK IN CAPE
COLONY, PRODUCED BY TWO SPECIES OF
SENECIO (SENECIO BURCHELLI AND SENECIO
LATIFOLIUS).

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DISEASES in which cirrhosis of the liver is the most pronounced *post-mortem* lesion are met with over the greater part of the world, and in certain countries have assumed the proportions of an epizootic amongst stock. In the great majority of cases the cause has been traced to the ingestion of some irritant in the food or water. As examples of these outbreaks we remember Winton's Disease in New Zealand, Pictou Disease in Nova Scotia, and Bottom Disease in South Dakota.

The Chief Veterinary Surgeon of this Colony, Mr D. Hutcheon, has long noted the prevalence of chronic atrophy and chronic hardening of the liver in horses and in cattle in Cape Colony, and has noted how such disease was, in many cases, one of locality. In the Report of the Chief Veterinary Surgeon for 1903 he says: "Sporadic cases are met with all over the Colony, but in some districts it is very prevalent, accounting for a large percentage of the mortality amongst stock, and particularly on the same veldt. Cases have occurred in animals which have never been in a stable in their lives, and on *post-mortem* the liver was atrophied, tough, and dense in texture."

The clinical symptoms usually shown (in the case of the horse) are those of stomach staggers.

A farmer in the Kokstad district says: "My experience was obtained in the middle districts of Natal and some parts of East Griqualand, where horses will often thrive and increase for a few years. Then in the early summer a close observer will notice that some do not cast their winter coats, which remain dry and unthrifty during autumn. By this time the horses have become dull-looking, soon hanging their heads, stagger about, and lean their heads against a wall or anything. This state continues for a week or more, when the animal is found dead, having stumbled into a ditch, or blindly staggered over a krantz."

Further, when referring to the treatment of this complaint, Mr Walker says: "The only successful plan we found was, immediately the horse was observed to be affected . . . to take him up, put him in a stable, and feed him day and night on dry forage—hay and mealies, give ordinary condition powders till the skin got right, then feed as the other stabled horses were fed. Having been housed, fed, and ridden for a year, the animal generally got into perfect health, and could be turned out on to the veldt for a few years again. We became accustomed to look out for the disease and to counteract it in this way, managing to rear a few horses to ride, as no animal that I remember took the disease till it was pretty well full-grown."

From the above and a great deal of evidence of a similar character, collected by myself and other officers of this Department, it is very evident that, in addition to the coarse innutritious vegetation and the alternate condition of poverty which the animals experience, there is something in the vegetation itself which appears to exercise a slow, irritating effect upon the liver, inducing the chronic hardened condition of that organ so frequently met with.

Further, it is a well recognised fact that this disease is much more prevalent on some farms in the same district than on others which are situated at the same elevation. It is even more prevalent on certain parts or camps of the same farm than on certain others, which clearly indicates that there is something special in the vegetation of that particular part.

During the years 1905-06 the attention of certain officers of this Department was directed to a similar disease which had been for some time prevalent in the Molteno district and adjoining areas of the Stormberg amongst cattle and horses, showing certain well-marked and characteristic clinical symptoms of dulness, straining accompanied with diarrhoea, mental aberration, and death, and on *post-mortem* well-marked and constant lesions in the liver, and frequently in the fourth stomach. This disease became so prevalent as to be alluded to as "straining-sickness" or "Molteno cattle and horse disease."

A letter was drafted in the form of a series of questions and issued as a circular to the stock-farmers of the area, asking, Whether it was most prevalent during a drought or when there was an abundance of food? Whether it affected cattle recently introduced or those which had been over a year on the farm? Questions were also asked respecting the symptoms and *post-mortem* lesions observed. There were some slight differences of opinion expressed on minor points in the replies received; but there was a general agreement on the following: It affected animals of all ages, but full-grown and old

cows the most. It attacked animals introduced as well as those which had been born and bred on the farm, but it was rare that an animal became affected before it had been a year or more on the farm. Only one gentleman had seen it in cattle which had been less than a year on the farm, and these cattle came from the Dordrecht district, most probably from similar veldt. This appeared to indicate that the disease was not due to any irritant or virulent poisonous plant which the cattle eat, as strange cattle would be most liable to graze on these plants soon after their arrival, before they had acquired any experience of their dangerous character. The length of time that the disease appeared to take to develop into an acute form indicated also that, whatever the exciting cause was, it produced its action very slowly. Another important fact which all were agreed upon was that no really typical case of this disease recovered.

I should like to add that stomach staggers in the horse is invariably attributed by the Boer and back veldt farmer to "Bots," or, as they are termed in the Colony, "Paapjies" (G. equi). Mr Chase, M.R.C.V.S., who was stationed in Molteno for the purpose of investigating the nature and cause of the prevalence of cirrhosis of the liver in stock in that area, had an opportunity of observing many cases of the disease and of making a great number of *post-mortems*.

The following is a brief summary of Mr Chase's interim report :—

The disease makes its appearance after the spring rains have fallen, about October, and continues until the end of March.

During the rest of the year the animals appear to be free from it. It attacks animals of all ages, from eighteen months onwards, and no breed of cattle appear to be immune.

Symptoms in Cattle.—In a few cases the affected cattle have been noticed to be poor and unthrifty for some time previously; in other cases the cattle appear to be in the best of health in the morning, and later in the day are found to be affected.

In cows the outset of the disease is marked by a greatly diminished milk supply, no desire whatever for food, and a dry, staring coat.

Often, however, the first noticeable symptom is a chronic diarrhœa of a most persistent type, which continues in spite of remedies which, in the ordinary way, would stop it. Fluid fæces are at first passed, but are soon replaced by a darkish-brown liquid. Straining now commences, with or without the passage of any fluid matter. The straining efforts are at first slight, but they rapidly increase both in intensity and frequency, until in the last stage I have timed them to occur fourteen times to the minute.

As a result of the excessive straining the rectum often gets everted, and the blood vessels in that region are ruptured.

As soon as straining commences the animals appear in great pain. In some cases they will lie down and groan, with the head stretched straight out in front of them; in others they remain standing, get into a frenzied condition, and attempt to charge any one approaching them.

All affected animals become rapidly emaciated, and generally die in from two to three days after the straining commences and after passing through a comatose stage, which lasts for a few hours.

Post-Mortem Appearances.—The most marked changes are seen in connection with the liver and gall bladder. In almost all cases the

liver has been found in a state of chronic cirrhosis. It has a leathery feel, and when cut into is found to be very tough; generally it is smaller than normal and of a slate blue colour, and its edges have lost their sharpness and become rounder. In one case the liver was much reduced in size and hob-nailed. The gall bladder is always found to be greatly distended with bile which has a characteristic viscid consistency, like treacle. The first, second, and third stomachs are usually normal, but the mucous membrane of the fourth stomach shows a number of pin-head petechiæ over its whole surface, and the folds of mucous membrane are thickened and soft from the presence of a semi-gelatinous fluid in the submucous tissue (*Chief Veterinary Surgeon's Annual Report*, June 1904). The three following cases in detail are from Mr Chase's report on the outbreak.

Case I.—Subject an aged cow, said to have been in low condition for four months, first seen 11th January. I found her to be very emaciated and weak, purging badly but feeding well. On the 18th the animal fell down on the veldt, and, being unable to rise, was killed on the 20th.

Post-Mortem.—Lungs, heart, stomachs, and intestines normal. Liver in an advanced stage of cirrhosis, the bile ducts being enlarged, thickened, and hardened. The external appearances resembled fluke cirrhosis, but no flukes were present. The organ was tough through its whole extent except at a few isolated centres on its edge, where it showed signs of fatty infiltration (which may have been caused by wasting). The gall bladder was enlarged, thickened, and attached to the liver; it contained half a pint of black viscid bile and a few growths on its inner surface (Section of Liver, Fig. 1.).

Case II.—Subject a fourteen-months-old calf, first seen on the evening of the 19th, when I found it purging and straining badly, feeding, but very weak, coat staring. Appearance depressed and dull, no yellow colour of the mucous membranes, which were somewhat anæmic. On the 20th and 21st the animal appeared worse, unable to rise, head drawn round to the opposite side on which he was lying, grunting and in great pain; died evening of the 21st.

Post-Mortem.—All the organs healthy except the liver and gall bladder; the liver was cirrhotic, cutting with a crisp harsh sound, and it was slightly smaller than normal. The gall bladder was thickened, enlarged, and full of viscid dark brown bile.

Case III.—Subject a six-years-old ox, first seen on the 26th, purging slightly but otherwise normal. On the 27th seemed better, and on the 1st started to purge again. In a few hours straining commenced, and the animal became mad and unapproachable; the straining by the evening became excessive, averaging fifteen violent strains to the minute. On the 2nd he died.

Post-Mortem.—All organs healthy with the exception of the fourth stomach, liver, and gall bladder. Gall bladder distended and full of thick dark brown bile, viscid like bird lime. Liver was cirrhotic but not markedly so. The lining membrane of the fourth stomach was thickened and œdematous, and showed discrete areas of extravasation.

From these cases the general run of the clinical symptoms and *post-mortem* appearances in the case of cattle can be seen.

Symptoms in Horses.—In cases of chronic cirrhosis in the horse,

or stomach staggers, to use its common name, the most common symptoms are:—The horse is dull and drowsy-looking, and will be observed to gape and yawn frequently; he continues to eat, but without that keen relish for his food which a healthy horse manifests, and he may fall asleep with his head leaning on the manger and his mouth full of food. He may then manifest symptoms of uneasiness and abdominal pain, he hangs his head in an extended position, and if loose he will move about in a sleepy heedless manner, and with a weak straddling gait in his hind quarters. If his mouth is examined, by inserting the hand and smelling it when withdrawn, it will generally have an offensive sour smell, more especially if the horse has been fed on grain food. These symptoms gradually become more pronounced, he becomes more restless, moves his legs uneasily, or paws with his fore feet, rests his head on the manger, or stands pressing it against the wall, or any obstacle near him. If let out, he will wander on with an unsteady staggering gait, his head hanging down, and an unconscious glare about his eyes. By-and-by he becomes more excited and delirious, and plunges about in a violent manner, with trembling of the muscles and apparent blindness. If on the veldt in this stage, he invariably wanders on until he tumbles into some sluit, when he continues his mad struggles until he dies exhausted, or if he is within reach of the homestead he may return, and force himself right through the door, head foremost into some house. Or he may become quite comatose, and die without a struggle.

Post-Mortem Appearances.—The organs with the exception of the liver are generally normal. The latter organ is generally atrophied, of a dull grey colour, and a tough leathery consistence. I may add here that I am well aware that stomach staggers can be produced by distension of the stomach with *any kind of food*, but the fact that so many horses suffer from stomach staggers in certain areas of the colony is indirectly due to the ingestion of some irritant acting on the digestive tract and producing the cirrhosis of the liver with the attendant train of symptoms.

Microscopical Appearances of Liver.—Veterinary Surgeon Chase when stationed at Molteno forwarded me numerous specimens showing various stages of the disease, from acute venous congestion to advanced cirrhosis with such a development of fibrous tissue as to almost render the liver functionless. In the stage of acute venous congestion, the interlobular veins are immensely distended, and the liver cells are in places vacuolated and contain fat granules.

In the more advanced cases, when cirrhosis sets in, the proliferation of the connective tissue is not limited to the interlobular region, but invades the lobules themselves in circumscribed areas. There is a great variation in the size of the lobules, some being almost obliterated. The invasion into the lobule may be traced in several stages, from a simple increase in the interlobular connective tissue, and with or without a rearrangement of the normal columnar grouping of the hepatic cells, to the complete substitution of dense connective tissue for the parenchyma.

These lesions and microscopical appearances correspond with those presented by the livers of cattle which have died from "Pictou disease," described by Wyatt-Johnson in Nova Scotia, who says:

"Pictou disease is limited to two countries, where it has been known for thirty years. It is not contagious, but destroys all the cattle living on certain farms within a space of three years." This also corresponds with the Molteno disease.

Pictou disease is ascribed to a weed, "ragwort."

The parenchyma of the liver is affected with degeneration and fatty changes; in two or three months cirrhosis appears and the animals finally succumb.

Dr Adami, in reporting on the disease to the Canadian Minister of Agriculture, finds "the main lesion an extreme condition of cirrhosis, the fibrous tissue not only being along the vessels between the lobules (periportal), but extending in between the individual cells, the

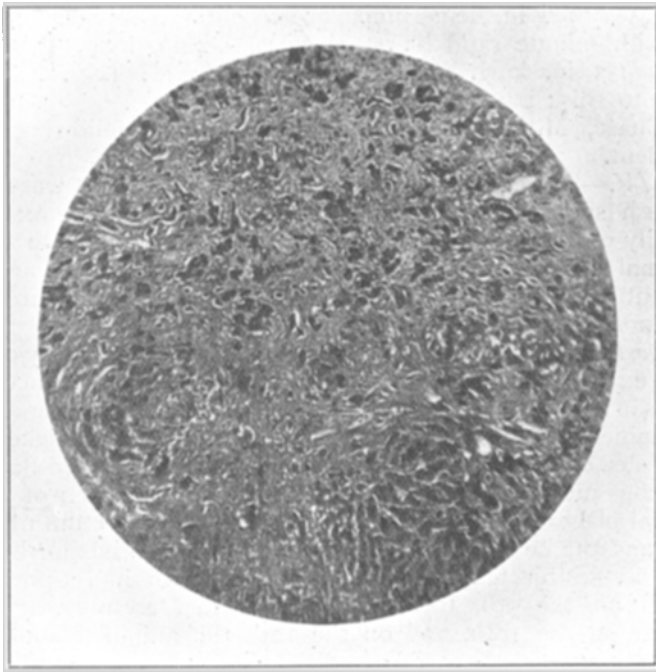


FIG. 1.

Micro-photograph of section of liver of an ox, showing cirrhosis from senecio poisoning.

organ being enlarged." (Dr Adami rather infers a bacterial cause for the disease).

In the *Annual Report of the United States Bureau of Animal Industry*, 1891-92, Dr E. C. Schroeder describes a disease associated with a form of cirrhosis of the liver in the horse, which occurred in Vermont, South Dakota, and which in clinical and *post-mortem* appearances closely resembles Pictou and Molteno Disease.

The disease in South Dakota is termed "bottom disease," from its prevalence along the marshy side or bottom of the Missouri. The clinical history is one of a gradual running down in condition, and on *post-mortem* there is found an enlarged firm friable liver, with a mottled appearance on section, and microscopically showing areas of

proliferating fibrous tissue, with degeneration of the adjacent or enclosed liver cells. The ranchmen attribute it to a plant of the leguminosæ, *Crotalaria Sagittalis*.

Mr Gilruth, Chief Veterinarian of New Zealand, in his reports for 1903-04 and 1904-05 describes fully a disease of stock associated with chronic cirrhosis of the liver, which is termed "Winton's" disease, and possesses many of the clinical symptoms and the majority of the *post-mortem* appearances met with in the Molteno disease.

The micro-photographs in Mr Gilruth's report show a marked resemblance to those published with this article: "In the more chronic cases there is observed an intense increase of fibrous tissue, chiefly interlobular and capsular. This tissue may consist of very

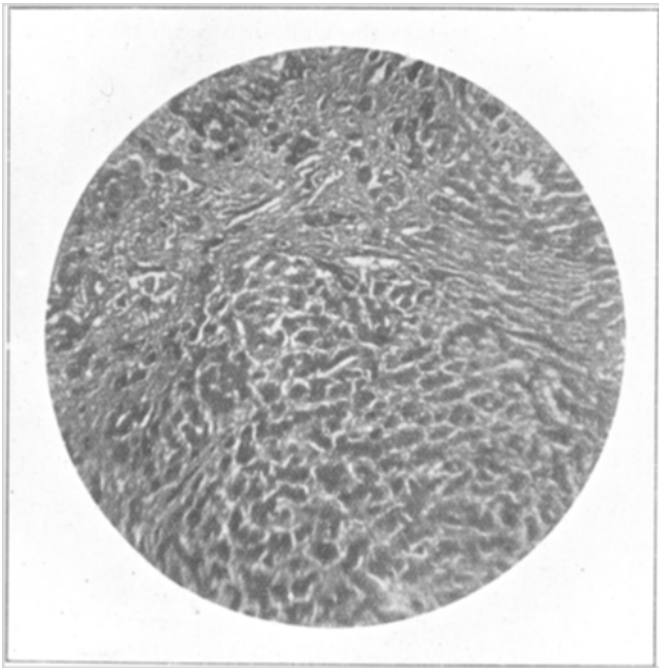


FIG. 2.

Same as Fig. 1, but cirrhosis more advanced

broad bands, which are more pronounced in the interlobular lesion, and send processes into the lobules, between the liver cells. In the very early stages there is naturally little formation of new fibrous tissue. The chief characteristic is intense portal and hepatic congestion, with an accumulation of new connective-tissue cells . . . Fatty degeneration may be noticed . . . New bile ducts may be formed in the new tissue."

FEEDING EXPERIMENTS WITH *SENECIO BURCHELLI*.

After certain inoculation experiments, all of a negative character, the bulk of the evidence pointed to some irritant in food or water as a

cause, and (bearing in mind Mr Gilruth's work with Winton's disease) suspicion fell upon a member of the *compositæ*, which Professor M'Owen, the Government Botanist, identified as *Senecio burchelli*.

This is a plant much resembling the ordinary ragwort, tansy, St. John's wort, or staggerwort, of Great Britain.

Description of S. burchelli.—Stem suffruticose, erect, scabro-pubescent or glabrous, striate; leaves half-clasping and minutely eared and toothed at base, linear, entire or denticulate, with revolute margins; fl. branches nude or sparsely leafy, loosely corymbose or paniced; pedicels elongate, scaly; inv. of about 12 scales, glabrous, calyced; disc.-fl. about 40, rays 5-7, flat; achenes minutely puberulous.

A scrubby, half-woody bush, with the habit of *S. rosmarinifolius*, but much looser inflorescence, the heads of twice larger size, on long pedicels, very few in the corymbs, or subsolitary. Stem more or less rough with minute rigid hairs. Leaves, 1-2 in. long, 1 line wide. Pedicels 1-3 in. long. *S. linariafolius*, Drege! (Hb. Sd.) partly belongs to this; partly to *S. debilis* (Hb. Hk.).

Mr Chase, M.R.C.V.S., then carried out the following feeding experiment.

Case IV.—Subject, a young healthy ox. The plant was obtained in a fresh succulent state just after the rains, and the ox received 4 oz. daily on the 12th, 13th, 14th and 15th October. On the 15th the animal was noticed sick, showing uneasiness, diarrhœa, and straining (eventually the rectum became everted), and it died at daylight on the morning of the 16th.

The *post-mortem* was very similar to the cases of naturally occurring disease, except that the lesions were rather more pronounced. The liver was in a state of acute venous congestion. Gall bladder distended and inflamed. Leaves of fourth stomach very much thickened and œdematous, due to the presence of a clear straw-coloured fluid under the mucous membrane, and over the surface were numbers of petechiæ, in several places approaching to ulceration. The rectum much congested, and the heart showed epi- and endo-cardial hæmorrhages.

Case V.—Subject, a healthy ox. Fed with half-a-pound of *S. burchelli* on the 9th, 10th, 11th, and 12th December. Diarrhœa appeared on the 11th, and on the 12th the animal commenced to strain, and continued to do so all day, death supervening on the 13th.

The *post-mortem* showed inflammation of the fourth stomach and thickening of its leaves by a submucous effusion; the red areas of extravasation were larger than in any other case. The gall bladder was distended, and its inner surface showed petechiæ. The liver showed lesions of acute venous congestion.

Case VI.—Subject, a calf. It was fed with small quantities of *S. burchelli*, extending over a period of two months, and it became so emaciated that it was slaughtered when unable to stand.

On *post-mortem* the liver had a shrunken appearance, slate-coloured, tough to the touch, and firm on section. The gall bladder contained 6 oz. of a dirty orange-coloured, thick and sticky bile, in fact it could be pulled out like bird-lime (Borthwick). The fourth stomach showed a few petechiæ, but no œdema. Other organs normal.

From a consideration of the above notes of Mr Chase's work there would seem to be little reason to doubt that the cirrhotic disease of

the liver of cattle in the Molteno area, with its accompanying train of symptoms, is due to the ingestion of *S. burchelli* in the food, and that the plant is most deadly in the young state.

It may be argued that the liver lesions met with in the experimentally produced cases were not so typically cirrhotic as in the cases naturally contracted on the veldt, but this may be and probably is due to the fact that the animals on the veldt only pick up a little of the plant daily, and thus the irritation though more lasting is less severe.

With the view of ascertaining to what extent the cases of stomach staggers (with cirrhotic liver as a prominent lesion) occurring in horses in the Molteno district were traceable to the action of the *S. burchelli*, Mr Chase started a feeding experiment on a young horse.

Case VII.—Subject, a healthy young horse. It was in good condition and during the course of the experiment it was carefully attended to and regularly fed and watered. On 24th November it received from 1½ to 2 oz. of the plant *S. burchelli*, and the same amount daily until the 6th December. On that date the animal refused to eat the plant in the forage, and so it was daily balled with that amount.

On 8th December it was noticed to be falling off in condition, and it continued to do so though the appetite remained good.

On 28th January the horse died, and, unfortunately, the *post-mortem* was rather delayed, and putrefaction had somewhat masked the pathological changes.

The liver was bile-stained throughout, firm, and indurated. On section distinct traces of new fibrous tissue were observed. The other organs were normal.

FEEDING EXPERIMENTS WITH *SENECIO LATIFOLIUS*.

As a result of the work in connection with Molteno disease we received numerous samples of plants for identification as the *S. burchelli*. One farmer persistently sent another variety of the *S.* family, which was identified by the Museum authorities as *S. latifolius* D.C., and many observers in Molteno assured me that this was far the commonest *senecio* there, and occurred thickly on farms where the cattle disease was very prevalent. We suspected the *S. latifolius* from its close alliance to *S. burchelli*, and I carried out the following feeding experiments.

By the kindness of a farmer in the Molteno area I have had delivered at the Experimental Station, Rosebank, a bi-weekly supply of this variety of *senecio*.

As supplied it is a tall (2 feet) herbaceous plant, with a rather woody stem supporting a few leaves, evidently the result of growing up amongst crowded vegetation; woolly on crown of the root, otherwise quite glabrous; stem erect, striate, tall, leafy, ending in a much branched, corymbose panicle; lower leaves oblong or obovate, acute or acuminate, tapering to the base, entire or remotely denticulate; upper numerous, cordate-eared and clasping at base, oblong or lanceolate, or linear-acuminate; partial corymbs many-headed, fastigiate, pedicels short, nearly nude; inv. of 5-8 glabrous, nerved scales, nearly nude at base; disc-fl. 10-12, rays 3-5; achenes quite glabrous.

Stem 2 feet high or more, leafy to the base of the panicle, the upper leaves diminishing in size. Leaves very variable in size, and in the



FIG. 3.

Senecio latifolius.

dentition of the margin, the wider ones penni-nerved, but even in the narrowest forms the upper leaves are amply cordate at base. Leaves in a/ 4-6 inches long, 1-2 inches wide ; in b/ 3-4 inches long, $\frac{3}{4}$ -1 inch

wide; in y/ much narrower. Panicle 6-10 inches across. Inv. 2-3 lines long, much shorter than the disc.

Case VIII.—Subject, a young ox (No. 63), bred on Cape Flats, and had been under observation for over six months. Started feeding experiment on 13th November, and 2 oz. of plant given daily in the food until 13th December, when the amount was increased to 4 oz. daily. On 26th December the ox started purging violently and straining, the fæces having a most offensive odour. The purging continued to the 30th, when the ox became unable to rise, lay straining violently, grinding the teeth, and twitching the eyelids. It died on the evening of the 30th.

Post-mortem.—Liver slightly smaller than normal, hard, firm, and cirrhotic; on section, the cut surface presented a general pale appearance with areas of congestion. The gall-bladder much distended, contained 2 pints of thick viscid bile with a consistency like bird-lime. The lining of the fourth stomach much congested and thickened, the mucous folds being distended with a clear fluid, and having a number of areas of inflammation on the mucous membrane. Intestines pale and quite empty. Rectum slightly everted and congested.

Case IX.—Healthy young ox (No. 64), bred on Cape Flats, and been under observation for six months. Started feeding on 13th November, and fed daily with 3 oz. of the senecio cut fine until the 13th December, when the amount was increased to 6 oz. Animal started purging on the 24th, strained most violently (everting lower part of rectum), fell down, was unable to rise or to stand when lifted, paddled with feet, eyes twitching, teeth grinding, frothing at the mouth. It died on the 25th.

Post-mortem.—Liver hard and cirrhotic. Gall-bladder distended, and full (3 pints) of pale-yellow, thick, sticky bile. Fourth stomach inflamed and congested, and folds thickened with clear straw-coloured fluid. The amounts of senecio given to the two oxen were:—

<i>Ox 63.</i> —2 oz. per diem for 31 days	3 lbs. 14 oz.
4 " " " 17 "	4 " 4 "
Total	8 lbs. 2 oz.
<i>Ox 64.</i> —3 oz. per diem for 31 days	5 lbs. 13 oz.
6 " " " 12 "	4 " 8 "
Total	10 lbs. 5 oz.

Case X.—*Ox 60.* Fed with 2 oz. of *S. latifolius* daily, increased for short time to 4 oz. 28th November 2 oz. daily until 13th December 4 oz. daily. 27th, animal fed badly; 29th, started purging; 30th to 4th January, purged badly; 5th January, reduced daily dose to 2 oz.; 21st February, found dead, no sign of struggling.

Post-mortem.—Carcase much emaciated, and subcutaneous tissue much bile-stained and of a pale straw colour. Mucous membrane of abomasum much congested, and its folds full of fluid and œdematous. Great pallor of membrane lining large intestines. Liver hard, firm, cirrhotic, cutting with a harsh sound, and smaller in volume than normal. Gall-bladder enormously distended, containing be-

tween 16 and 17 pints of thin watery bile (*see* Fig. 3). This distension of the gall-bladder would seem to be a prominent feature in senecio poisoning.

Horse No. 50.—Animal was a cast from the Police, and during the past four years had been fed on the ordinary horse ration of the force. 16th January, started feeding on *S. latifolius* (in dry con-

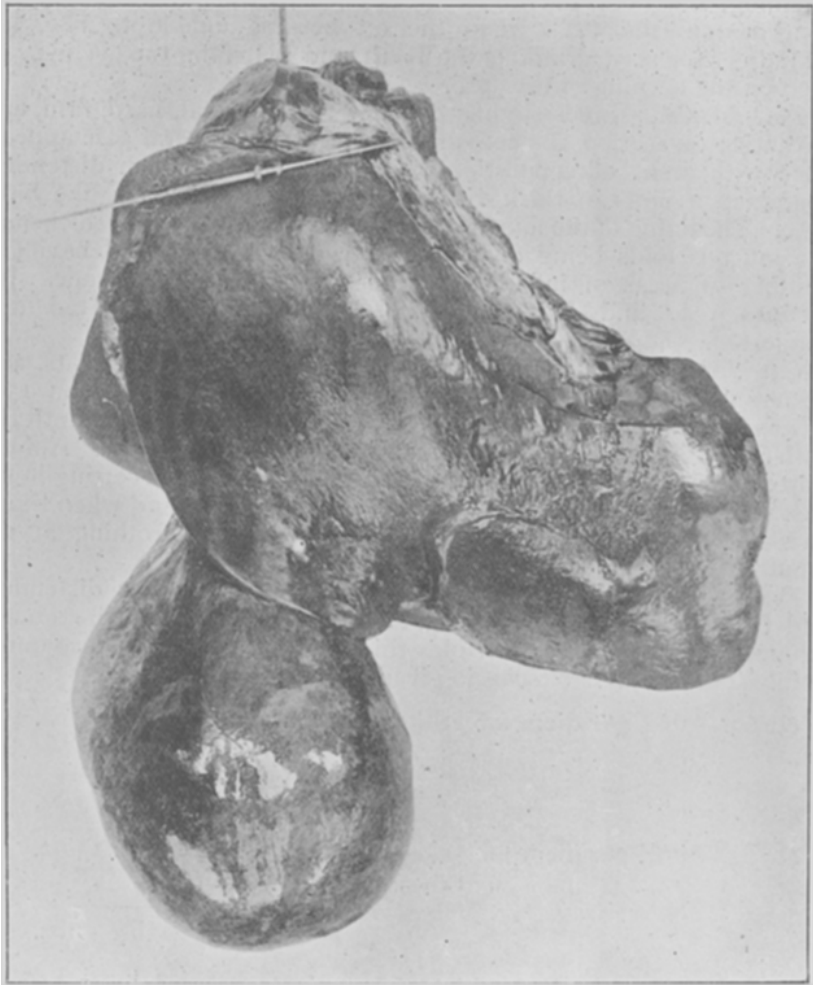


FIG. 4.

Liver of Case X., showing great distension of the gall bladder.

dition). He ate about 2 lbs. daily until the 17th February, when he refused all food. 18th February, seemed stiff and ill. 19th, not feeding at all. 20th, at 2 P.M. fell down suddenly, and got up with great difficulty. Nostrils distended, symptoms of acute abdominal pain, shivering, sweating; fell again suddenly at 4 P.M. At 10 P.M. was in very great pain, lying down, rolling, endeavouring to stand on his head and climb the walls. At 11 P.M. fell down as if shot, got

up, seemed quieter. At 11.45 pain commenced again at intervals of fifteen minutes, and continued until 3.30 A.M., when the horse died.

Post-mortem three hours after death. Rigor mortis well marked, subcutaneous tissue of body stained a deep yellow, not unlike malignant jaundice in the dog. Lungs, heart, and kidneys normal. Liver smaller than normal, firm, and cirrhotic in patches. Mucous mem-

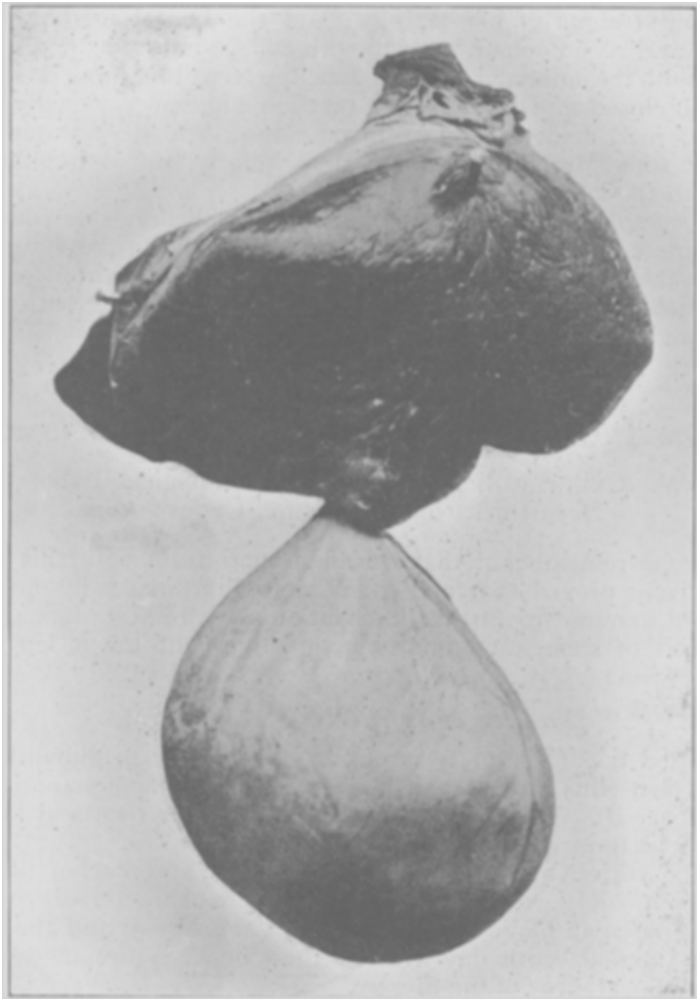


FIG. 5.

Same as Fig. 4.

brane of stomach slightly congested. Lining membrane of large and small intestines greyish in colour.

I have called particular attention to the sudden onset of pain in this case. Before 2 P.M. on the 20th February 1906 the horse, though not looking well, was in no pain. This peculiarity I have also noticed in cattle.

Horse No. 18.—28th December 1905, animal arrived in good con-

dition, save that it was suffering from injury to fetlock. 29th December, 4 oz. *S. latifolius* given and repeated daily until 2nd March 1906, when the animal, which had been gradually going off in condition and purging at intervals, show symptoms of colic; this colic appeared intermittently during the 3rd, 4th and 5th. On the 6th the horse refused food for the first time, appeared stupid, pulse slower than normal, temperature unaltered; the mucous membrane of the eye showed signs of bile-staining, and the buccal membrane was somewhat dirty in colour. On the 9th colicky symptoms again supervened, and the animal appeared much worse, breathing stertorous, eyes half closed and head resting on the manger, occasionally boring into a corner, evidently quite unconscious, no food or water taken. On the 10th March the animal died quietly, remaining standing almost to the last.

Post-mortem.—Great bile-staining of the subcutaneous connective tissue, mesenteries, and peritoneal covering of the diaphragm. A considerable amount of straw-coloured fluid in the peritoneal cavity. Heart flabby and pale. The liver small, hard, cutting with a harsh sound, and very cirrhotic. Hardly any healthy liver tissue left.

SERUM INOCULATION IN CANINE PIROPLASMOSIS.

By W. ROBERTSON, M.R.C.V.S., Bacteriological Laboratory,
Department of Agriculture, Cape Town.

It may be remembered that certain experiments with this disease conclusively proved that a dog salted to the disease by one attack remained immune to further inoculation or tick infestation, and that the blood of such an animal remained virulent for a lengthened period of time (*Agricultural Journal*, Vol. XX., No. 6).

Duration of Infectivity in Salted Dog.

A salted dog (No. 11) was obtained from the neighbourhood of Cape Town (this animal had had an attack of malignant jaundice and recovered). The dog was received November 1901 and its blood carefully examined, but without finding any parasite.

On the 16th November this dog was bled, and 6 cc. of the blood injected into dog No. 10; in eight days the characteristic pyriform inter-corporal bodies were noticed in the blood, and the animal died on the fourteenth day with all the clinical symptoms and *post-mortem* appearances of the disease.

This dog, No. 11, was first tested at short intervals, and then at longer periods:—17th November, 26th November, 3rd December, 17th December 1901; 6th January, 24th January, 3rd February, 25th February, 27th February, 13th March, 9th August, November 1902; 1st January, 1st April, 2nd June, 1st September, 4th December 1903.

In every case the inoculation of the blood from No. 11 produced death from malignant jaundice, *i.e.*, from 16th November 1901 to 4th December 1903, a clear period of two years. All this time the animal had been kept in a run where it was free from any infective influence.