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for the

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PRETORIA

South African Veterinary Medical Association Annual Meeting held at Onderstepoort, in September, 1938, under the presidentship of Mr. S. T. Amos, F.R.C.V.S.

PRESIDENT'S ADDRESS.

It is again my honour to present to you my presidential address for the past year. Your Association has made another step forward in its utility to the veterinary profession. The Council of your Association has met whenever necessary and has dealt with the many questions that have arisen and, whilst on this topic, may I again remind you of the debt of gratitude we owe to Council members for their keenness in carrying out this important work.

1. *Private Practice in South Africa:*

There has been a very marked expansion in private practice in the Union of South Africa in recent years. As an indication of this, as well as of the faith members have in private practice, one might mention that this branch of veterinary work is no longer merely confined to retired state veterinarians, but that several young graduates have within the last two years taken up private practice in preference to accepting remunerative posts in the State and Municipal services.

It is now becoming evident that private practitioners are realizing that it is only by co-operation and loyalty to one another that their interests can best be safeguarded, and the example of the private practitioners of Johannesburg is a very excellent one; for in a letter of the 6th September, 1938, signed by practically all practitioners in that area their acquiescence in the acknowledgement of these principles was agreed upon.

The private practitioners do express appreciation at the manner in which the Department of Agriculture has honoured its agreement whereby state veterinarians, whose real function is to attend to scheduled diseases, are not permitted to do private work in areas where there are private practitioners. We have also recently obtained a definite assurance from the Department that, except in those areas where there are no private practitioners, animals will only be admitted for treatment at the medical and surgical clinic at Onderstepoort, if accompanied by a veterinary certificate.

2. *Foot-and-mouth Disease Outbreak in Natal:*

I think everyone connected with our profession feels very proud of the manner in which this most serious visitation to farmers has been dealt with, and, without being unduly egoistical, we must honour those veterinarians

upon whose shoulders this terrific work was placed. First of all we must be proud of the higher officials whose duty it was to decide on the method of dealing with the outbreak, and then of those men who were called upon to show an enormous amount of tact, and of physical and mental energy in carrying out the policy laid down. Those of us who have had to deal with farming communities in the crises of great economical loss, and have had to apply irritating but necessary restrictions, can very well appreciate what the campaign has meant, firstly to those farmers so seriously affected, and secondly to the veterinary department generally. One must not forget that the native population of our country was also included in the large list of cattle owners, and the smooth manner in which the outbreak was first of all located, and then dealt with, will ever stand to the credit of the veterinary department. I personally am prepared to pay the greatest tribute to all those whose duty it was to deal with this outbreak, and where the results have been so excellent it would be quite invidious on my part to mention any one name, although I am very sorely tempted to do so. They and we must share in the tribute that has already been paid to the veterinary profession of the Union of South Africa.

3. *13th International Veterinary Conference at Zurich, 1938:*

The South African Veterinary Medical Association was ably represented at this Conference by Drs. P. J. du Toit and J. Quin. It is with a good deal of pride that we record the fact that a draft of an International List of Animal Diseases, which was compiled by the veterinary staff of the Research Institute at Onderstepoort was presented at this Conference. Great appreciation of this work was expressed, and the list was submitted to the various countries for further consideration and discussion at the next Conference. To those who have not had the opportunity of seeing this list I can assure them that it is one that called for a tremendous amount of thought and, to use the words of Dr. du Toit, our Director of Veterinary Services, " The list which is hereby presented to the International Congress is intended to serve merely as a basis for further discussion. It is a first attempt to classify the diseases of our domestic animals. Many of its defects are obvious. It has been compiled in the first place for the use of English-speaking veterinarians; other national committees will have to adapt the list for use in their own countries, but whatever the shortcomings of the list the hope may be expressed that the labour of the compilers will not have been in vain. Perhaps this undertaking will in some small measure help in bringing veterinarians from all over the world closer together, and in promoting veterinary science." Personally, I consider this work of the greatest value to our profession, and a great honour to Onderstepoort and its zealous workers.

4. *The Journal of the S.A.V.M.A:*

This undoubtedly is an excellent agent for bringing the work of your Association to the notice of the veterinary world. The efforts of the present

and past editors and editorial committees of the Journal deserve our highest appreciation, for they have always maintained a very high level, and, as a result, the Journal is rapidly increasing in popularity, especially with overseas veterinarians and veterinary institutions. It is only necessary for me to bring to your notice that this high standard can only be maintained by the full co-operation of all our members. The cost of providing it must be met from subscriptions and advertisements, and I therefore appeal to all members to pay their subscriptions promptly and to support, whenever possible, those who advertise in our Journal.

5. *General:*

Your membership today numbers 176 and we have to regret the death of two members during the past year.

I cannot conclude without expressing my great appreciation of the work of the Council Members and all the co-opted members on the different committees. They have made the working of the Association a very pleasant one indeed.

To your Secretary, Mr. Van Rensburg, and to his assistant, Mr. Van der Wath, I tender my sincerest thanks for the great work that they have done, and would remind members again of the debt we owe to such officers.

Seventh World's Poultry Congress and Exposition.

The usual Triennial Conference will be held this year in the United States of America. There will be two venues — Washington D.C. from July 25 – 27, and Cleveland, Ohio, from July 28 – August 7.

Everything points to this Conference surpassing all previous ones. It is not difficult to understand why this should be so, seeing that nearly all poultry research work in the world is centred in the United States.

The Lungsickness Campaign in the Caprivi Zipfel.

By G. McINTYRE, Government Veterinary Officer, Aliwal North.

On the 1st June, 1938, I received instructions to proceed to the eastern Caprivi Zipfel to assist in an inoculation campaign against lungsickness. The letter contained the note that no supplies were obtainable in the Caprivi and stocks would have to be obtained at Livingstone. This did not sound too good and I tried to obtain some information on the area, but could find no one who knew anything about it. In fact, few people knew where it was. On communicating with my Senior Veterinary Officer I was informed that I would be camping, and that camp equipment would be supplied. To be on the safe side I packed everything I thought would possibly be required; stretcher, bedding, cooking and eating utensils. It was as well I did, as on arrival at Livingstone I found that the official camp equipment was short of cooking and eating utensils and that the table, bed and bedding could not go in the aeroplane. Transport from Livingstone to the Caprivi was to be done by aeroplane. Two military planes from Roberts Heights were detailed for the work. I had to repack my kit into smaller packages and found in this connection that cardboard containers obtained from the stores were more suitable than wooden boxes, as they were lighter and more easily handled.

Dr. G. Schmid, Government Veterinary Officer, from Windhoek, was in charge of the operations. I met him at Livingstone. He had made a preliminary survey of the area and arranged inoculation centres, building of crush pens, collection of cattle, and the construction of landing grounds.

The Caprivi.

The Caprivi Strip is a long narrow stretch of country lying between Angola and Northern Rhodesia on the north, and Bechuanaland Protectorate on the south. It is bounded on the east by the Zambesi River and on the west by the Okavango River. It is divided into two by the Kwando River, which flows from north to south through it. From the Kwando River westwards the country is uninhabited as it is waterless. This area was not visited so it is not dealt with in this article. The eastern Caprivi Zipfel is the area between the Kwando River and the Zambesi River. It is shaped roughly like a ham with the shank at the junction of the Zambesi and Chobe Rivers.

In its passage through the Caprivi the Kwando River is known by local names; thus where it cuts the Caprivi it is known as the Mashi River, where it turns east, as the Linyanti, and when it emerges from the swamp,

as the Chobe River. It is a big river with a strong flow of water. Its width varies from fifty to one hundred yards and its course is very tortuous.

The country is very flat and is subject to floods in the rainy season. The average rainfall is 36 inches per year and for most of the year the country is water-logged. Only for three or four months, in winter, is it possible to move about comfortably at all. The Superintendent of the area travels in summer by barge or canoe, and in winter by Scotch cart or on horseback. On the east the country is fairly open, with swamps and clumps of bush; but further back from the rivers the trees increase, until along the dry part in the north there is quite a forest. Tree growth stops wherever water lies for any length of time. These areas can be distinctly seen from the air. The trees are principally species of acacia, but there are other varieties of tree of which I do not know the names. These grow to a good size and provide the wood for canoes and domestic utensils. Their native names are Mupolota and Molombe. The open areas are covered with coarse grass which grows six or seven feet high and the vleis are covered with reeds. The soil is a light infertile sand on top, with a rich sub-soil underneath.

Wild Life.

The Caprivi is a natural game park. It is a very isolated area and therefore has not been visited by shooting parties. There is game in abundance. Antelope are plentiful on the east and south, but get scarcer in the drier parts. Herds of eland, reedbuck, and letchwe were seen. Elephant and hippopotamus are numerous. Giraffe are present. Lions and hyænas are plentiful, and take severe toll of the cattle in the area. Pythons are plentiful and are hunted by the natives. Crocodiles infest the swamps and rivers. At one vlei ten large dead crocodiles were seen. They had been killed by the natives as the vlei dried out. Along the flood area and near the gardens, there is an abundance of bird life. Some of these birds have wonderfully coloured plumage and are very pretty. Ants are numerous and are the real cultivators of the country. As they work and build their ant-heaps they bring the sub-soil up on top of the sand. They build enormous ant-heaps. These are like small hills being several feet high and several yards long and broad. Trees take root in these ant-hills and grow, and in time, due to the fall of leaf and the collection of sand round the trees, a distinct mound is built up.

Population.

The Caprivi is occupied by natives. It is fairly heavily populated on the east and south, but only sparsely west and north. There are three different tribes in the area — the Massubia, the Bafui, and the Maeie. As is usual with natives, the less civilised, the better they are. The Massubia occupy the east, the Bafui the centre, and the Maeie the south-west of the eastern Caprivi.

They live community lives in villages under a village headman. The villages are mostly sited just above flood level. The natives, as far as one could judge from the men who came with the cattle to the kraals, are a well built active type, and are of a cheerful disposition. The average height of the men was 5 feet 8 inches, but taller men were frequent. Their principal food is grain, (mealies and kaffir corn) and milk. They get meat only when a beast dies or a buck can be speared. A beast is seldom killed for eating. The flesh of the python and lion is eaten, that of the latter being considered a particular delicacy. A lion was shot near my Mashi camp and its carcass was cut up and eaten. Three oxen were killed at Linyanti to get raw hide to repair the crush. The meat was dumped near my camp on the night the animals were slaughtered and distributed the next day. Distribution started at 4 p.m. and did not finish till 6 p.m. There was so much argument and noise that I thought violence would be done before the business was finished.

The natives are poor. Their cattle are of little value and they are very far from the labour market. The price paid for the three oxen that were slaughtered was £1 10s. each. They were big oxen and would have been worth £10 on Union markets. The lobola system is in force and the exchange is one beast for a wife. Grain is grown in gardens in the flood area. Villages that are away from the flood area have their gardens there, and during the time of the cultivation, growing, and reaping of the crop, certain members of the village live at the garden. When grain is harvested, it is threshed on the spot and stored in large baskets that have been plastered inside and out with ground from ant-heaps and, when full, sealed with the same agent. These are kept on a raised platform and covered with a roof of reeds to protect them from the sun and weather. Gardens are made where ants have worked and brought the sub-soil up above the sand. The trees are cut down and burned and the ground among the stumps ploughed and planted. Native tools consist of spears, knives, axes, and adze. These they mostly make themselves. They are very clever with them.

Their principal musical instrument is the tom-tom. They also get music out of an instrument made up of six or eight steel strips fastened to a piece of flat wood and held over a gourd to increase the sound.

Canoes are used on the rivers. These are cut from one solid log and are from twelve to twenty feet long and from twelve to eighteen inches wide. They looked difficult things to handle. When a canoe is no longer of use in the water it is used as a sledge. One day at Linyanti I visited one of the native camps and found other natives showing great interest in a portion of a canoe that was being used as a sledge. I inquired what was the cause of the interest and was informed that this was a portion of a canoe that had been made by Makoloil people who no longer existed, having been destroyed in native fights.

Natives had to bring their cattle to the inoculation centres and camp with them there until the inoculations were finished. They made kraals and shelters for themselves from thorn trees and kept fires burning all night on account of lions and hyænas. When any of these gentry were about nobody slept, as the natives kept up a constant noise shouting, beating tom-toms, and cracking whips. They had good reason to be afraid of lions. A lion attacked the kraals the second night I was at Mashi. It was later shot by a native hunter and the news was received with great celebration. I made a post-mortem examination of this animal, and found it to be a lioness with three fully developed cubs almost ready to be born. She was very fat and had two tapeworms. On another occasion, when we went to work in the morning, the spoors of three lions were found at the kraals, where they had evidently passed the night. I had difficulty in getting the natives to work as they all wanted to go and cut wood for fires. That night there was no rest and the noise created was terrible. It was sufficient to scare everything within miles.

The natives concentrated at Mashi were very primitive. They wore no clothes, though it was winter and the weather cold, and seemed to have very little of anything. They were a well-built lot of men and worked well. At Linyanti there was a mixed lot from two tribes. Some were of the Bafui tribe, partially civilised and lazy, and others of the Maeie tribe, natural and workers.

There is only one small store in the Caprivi — at Katima Molilo. The principal trading centre is at Shesheke in Northern Rhodesia. The Caprivi natives do nearly all their trading there, though it means a four or five days' trek for most of them. It was from there that they introduced lungsickness into the Caprivi.

Cattle.

The cattle I met were a very mixed lot. I thought that in such an isolated area a special type of animal would have developed, but such was not the case. No village had one type of cattle and there seemed to be no endeavour made by owners to keep to a certain strain. As usual with native herds, there was a good percentage of bulls.

Vegetation.

As was to be expected in such an area with its abundant water and tropical climate, the vegetation was luxurious. Grasses grew so thick and long that it was difficult to get through, and the seed stems were very thick and strong. The long grass made shooting difficult and dangerous. The inoculation centres were chosen where there was plenty of grass and water away from the ordinary grazing grounds of the villages. Many of the trees are evergreen or carry their leaves till late in the season. I did not recognise any of the trees except the acacias.

Lungsickness.

Lungsickness was introduced into the Caprivi from Shesheke in Northern Rhodesia, where it is practically endemic. It spread through the villages on the trade route on the east of the Caprivi, where travelling cattle were kraaled for the night. The disease spread from the Caprivi to Bechuanaland Protectorate through an affected ox ridden by a native when he went visiting in that territory. In 1937 a large number of infected and in-contact cattle in the eastern Caprivi were destroyed and there was good reason to believe that the disease had been stamped out. I did not find any affected cattle during the inoculation and none was found in any of the other areas. Several carcasses were examined, but no signs of the disease found, so that the type of lungsickness that affected the area cannot be described.

Inoculation.

The inoculation campaign was carried out as a protective measure and to assist the Bechuanaland people to dispose of their cattle. A preliminary census had been made and it was found that there were about 24,000 head of cattle in the eastern Caprivi Zipfel. The country was divided roughly into three areas each having about the same number of cattle. In these areas certain inoculation centres were prepared and owners instructed to bring their cattle to these places for inoculation. They had to keep them there until the inoculations were finished. This was why these centres were selected away from the villages and where water and grazing were plentiful. When put into practice very little adjustment was required and the scheme worked well. What adjustment had to be made was due to delay of the owners in producing the cattle, or to a discrepancy in numbers. A week was allowed for the inoculation at each place. At Linyanti, the place at which I inoculated in the second week, both these troubles arose. There was delay in producing the cattle and the number that were brought exceeded the estimate by 1,227. Fortunately, I was able to communicate with Dr. Schmid, and he was able to take over the third place in my area.

Crush pens, with entrance and exit kraals, had been built at each place of inoculation. The crushes were works of art ! They were a forest of poles with saplings as side rails and the whole lashed together with strips of bark and portions of raw-hide. The poles were not set deep in the ground and as the ground was sandy there was no strength in the crush. The crushes held from twenty to twenty-five head of cattle, and these could not be packed in tight. The crushes were being constantly broken and damaged, and one native had to be kept on repairs. In my area, cattle were brought forward by villages and not by individual owners. The largest number owned by a village was 360 and the smallest three, the average being 99. The two centres at which I worked were known as Mashi and Linyanti. At Mashi 32 villages brought 2,891 head, and at

Linyanti 32 villages brought 3,427 head to the first inoculation. There was very little increase. The natural increase that did take place was offset by losses. At Mashi the increase and loss between the first and second inoculations was 43 and 40 respectively, and between the second and third inoculations 51 and 46 respectively. At Linyanti the increase and loss between first and second inoculations was 76 and 16, and between second and third inoculations 67 and 22, respectively.

The vaccine was a pure culture of the pleuro-pneumonia organism in Martin's broth plus serum. It was prepared by the Kabete Laboratory, Nairobi, and was issued in bottles containing 200 cc. It was of an amber colour, felt "harsh" to the touch and had a strong but not unpleasant odour. It did not keep long and had to be used within twenty-one days of issue. The dose was one cc. and three injections had to be given; the first two in the tail, and the third in the shoulder. I saw several cattle that had lost their tails; and these got the three injections in the shoulder without any apparent ill effects. Vaccine was carefully handled and syringes were frequently cleaned and sterilized, but the inoculation site was not disinfected.

The vaccine was sent from Nairobi to Livingstone by airmail, and was distributed through the Caprivi by aeroplane. It came out every Friday. Inoculations were given at intervals of three weeks. At Mashi area I found 30 per cent. of adult cattle reacted to the first inoculation, but no appreciable reaction to the second inoculation. At Linyanti the reactors were more numerous, being 40 per cent., and occurred among tollies as well as adult cattle. No reactors were found among calves, and very few among cows with thin tails. The reaction was a thickening of the tail through oedematous swelling of the subcutaneous tissues. No reaction necessitated operation on the tail, and no deaths occurred that could be definitely attributed to the effects of the vaccine. I left before I got any reports of the effects of the third inoculation. There was a surplus of vaccine at the third inoculation so most adult cattle at Mashi, and many at Linyanti, were given a dose of 2 cc. Calves born between the first and second inoculations were given 2 cc., and those born between second and third inoculations were given 3 cc. of the vaccine.

As the cattle were inoculated they were branded 1 for first, 2 for second and 3 for third inoculation. Owners had been warned that if the police found any cattle not carrying the proper brands, after the inoculation, they would be destroyed without compensation. At Linyanti two cattle were brought to the second inoculation that had not been at the first, and they were immediately destroyed.

Before leaving Livingstone we met Mr. M'Cauley, veterinary officer at Maun, in Bechuanaland Protectorate, and had a most interesting chat with him. He informed us that the inoculation of cattle in Bechuanaland

had just been completed, and that there had been no reactions, and that an extra inoculation of 3 cc. had been given in the shoulder. There had not been time to get reports of the reaction to that inoculation. The inoculations in the Caprivi were more successful, as at all places reactors were found. I noticed that the number of reactors varied in different herds, but cannot account for this.

Handling the Cattle.

The natives are very rough with their cattle. The cattle had never before seen a crush and there was great difficulty in getting them to go into it. The natives used to get them bunched up at the entrance and thrash them unmercifully. There would probably be one animal standing across the entrance and jammed there unable to move, and no one would think of clearing it. I had constantly to be telling them what to do, and the difficulty was that when one lot was clear, the same thing would happen with the next one. Men from a village worked with their own cattle, then cleared off, and when the next lot came along the same instructions had to be repeated. I was given three native assistants at each crush. They did the branding and assisted in checking the cattle, and held them during inoculation.

Other Diseases.

There seemed to be very little other disease among cattle in the Caprivi. What there was, was mostly due to parasites.

Anthrax broke out in one area during the inoculation and the description given of cattle deaths in other areas would indicate that the cause was anthrax. The natives speak of a spleensickness which is probably anthrax.

Aphosphorosis is troublesome and affects growth, but is to be expected in country of this nature.

Lungsickness: This disease has been and gone and I hope will remain a memory.

Anaplasmosis: From some examinations made of the entrails of young stock that died I think this disease must account for some loss among them.

Fascioliasis: Of the internal parasitic diseases this is the most severe. It occurs mostly in the east, where the country is very wet, and swamps and vleis never dry out.

Lice: Haematopinus eurysternus was very common and did considerable damage. It was found confined to the tail, and caused a dermatitis which thickened the skin and made it dry and hard. The hairs of the brush were frequently found as stiff as wire, being coated with the "glue" with which the eggs had been fastened to it.

Ticks: Cattle were heavily tick-infested. Many suffered from abscesses due to tick bites. The common ticks were *Boophilus decoloratus, Hyaloma aegyptum impressum* and *Rhipicephalus evertsi.*

Deaths from Disease: Deaths from disease were not frequent and lions and hyænas accounted for more deaths than anything else.

Treatment of Disease: The favourite method of treatment of a sick animal seemed to be by branding it over the place where it was thought to be sick. Circles were found branded round the eyes, over the heart and joints, and squares on the chest and abdomen.

Castration is carried out at weaning time. The scrotum is opened, the testicle pulled out, and the cord severed with a sharp knife.

Cattle are decorated with knobs of skin at the nose and over the eyes. These are produced by stripping a piece of skin above the place to be decorated, leaving it attached at the place, and allowing the wound to heal. I saw some cases where the skin had been stripped off from the whole length of the nasal bone, and a hard horny growth had filled the wound.

General.

Transport: There are no roads and therefore no wheeled vehicles in the Caprivi, except the scotch-cart of the Superintendent. The natives use sledges when it is possible to get about. Aeroplanes were used on the lungsickness campaign. Two Wapiti aeroplanes from the South African Air Force at Roberts Heights were detailed for the work and stationed at Livingstone. Landing grounds had been prepared at various centres. These were rough and sandy and not very large, but proved very serviceable. I had not been in an aeroplane before so this was quite an adventure. I thought the instructions given me somewhat casual. They were: "Keep your head down if anything happens, and, if necessary to jump, take the parachute ring, jump, count one, two, three, and pull". What was to happen after that was not explained. Fortunately there was no need to practice, and I never felt that there was any need to keep my head down.

As I have stated earlier I had to repack my kit into smaller packages, and in this connection would mention that for such transport, kit should be done up in rolls, if possible, or in small cardboard containers. The aeroplanes took us to our stations, brought out the supplies of vaccine, and moved us from one place to the next when required. There was no breakdown in this service, which was eminently satisfactory. This was our only link with civilization, and the day of the aeroplanes' visit was *the* day of the week. The Air Force personnel were very cheerful and obliging and did much to help the time to pass. I wish to put on record my appreciation of their unfailing kindness and assistance.

The aeroplanes were of great interest to the natives who kept a constant look-out for them. One of the police "boys" assisting at Mashi had to come to assist at Linyanti. He did not care to fly, so, the first time, he walked the fifty miles through the bush rather than fly. He went back to Mashi with the aeroplane, however, and was annoyed because he was not provided with a flying cap.

The Chief at Linyanti had misbehaved and one day the Superintendent with his police boy arrived by aeroplane to arrest and remove him. The Chief got away, so the police boys were instructed to find him and arrest him, and the planes would come back in two days to collect them. They caught the Chief, but he preferred to walk to Katima Molilo than to fly.

Climatic Conditions: I found the weather extraordinarily cold at nights. During the day the climate was not bad, till the winds started and the dust got stirred up. It was frequently so cold in the morning that the natives were slow in turning out to work, and I had to send the police boys to fetch them. One morning at Mashi there was ice on the water that had stood overnight in the dixie. The nights were so cold there that I could not get warm, though I had six blankets and an eiderdown quilt. Mashi was much colder than Linyanti, but I do not know why. From the middle of July strong winds blew that stirred up dust and made working conditions very uncomfortable.

As all days were alike to the natives, I worked every day of the week to get finished. Work started at 8 a.m. and went right on till 5 p.m., without a break. I found it impossible to get owners to turn out earlier. Camp servants were a difficulty. I could not get one who knew much about cooking. I got one at Mashi and one at Linyanti, but they knew very little and I had to instruct them and supervize the cooking myself. They live very simply themselves and have not any opportunity of learning other methods. All water used in camp was boiled as a precautionary measure against malaria and bilharzia. There was a small black mosquito at Linyanti that bit most viciously. Malaria is prevalent there. Linyanti is where Dr. Livingstone came through from Bechuanaland on his exploring journey, but there is no memory or legend of his visit among the natives to-day.

Equine Colic.

By WAKEFIELD RAINEY, Bloemfontein.

In a mechanical age a new article on this old subject may seem out of date, and a treatment of the matter on orthodox lines would be uninteresting to at least ninety per cent. of modern veterinarians. But in a world that yet contains millions of horses distributed through nearly every country, equine colic is still a universal disease which claims a place in the onward march of veterinary science.

An editorial article not long ago in a well-known English veterinary journal commented with interest on the fact that veterinarians still seemed to be concerned about equine colic. We should be so concerned because, until we can master the pathology and rationalise the treatment of so common a malady, we have no business to bewail the trespasses of the quack.

It is hoped to discuss colic here in a way that will be new and that may help to renew our ideas of the treatment of other common maladies with which familiarity has bred contempt. This will not be a classical dissertation in the text book style; there is no need now to add to the long and dreary lineage of recitations that pre-date Youatt and will survive this article.

We all know what we mean by " colic " and if there are any who will be more precise, half a hundred text books will supply as many definitions. All that the writer would do is to turn, if he can, the light of reason, of truth and of humanitarianism, on to the treatment of this disease, a disease that is, more than almost any other malady of animals, submerged in darkness, irrationally handled, misconstrued, and whose treatment has often meant cruel suffering to animals.

It is generally agreed, and generally true, that a fairly definite knowledge of the cause must precede the rational and useful treatment of disease, and right here, on the threshold of our essay, we discover the reason for our failure, as a profession, to deal rationally with colic. We do not know the essential cause of the disease, or of the group of symptoms constituting the disease we call colic. We know a number of the concomitant factors of the cause, but not the cause itself.

Etiology.

We are aware that the average case of common colic is what we see as the result of spasm of the muscles of the intestines or stomach, due to disordered function, which may, in its turn, be due to indigestion, or fatigue,

or displacement, or a number of other contributory factors. But we do not know exactly why at that moment, which may not differ much from many other moments in the life of the body, such violent spasms, causing excruciating agony by compression and stretching of nerves, should occur. Nor, as a corollary of that ignorance, can we tell why at some moment, in most instances, the spasm ceases and the animal recovers. Very recently the amazing patience and skill of the physiologist and the biochemist have given us what may prove to be a glimpse behind this veil, in their reported discovery of the part played by certain ferments in gastro-intestinal activity. A ferment called acetylcholine is said to be secreted or at least liberated at the nerve endings when the parasympathetics are stimulated, and it is claimed that acetylcholine excites bowel function both of secretion and movement. Another ferment, esterase, is said to have a somewhat opposite effect. From the practical point of view the matter is still vague, but if these ferments exist, and if they have these effects, it is likely that there are other ferments having other similar effects, and that a profound disturbance of their interaction might speedily produce a disease of derangement, such as colic.

There are diseases such as cancer, eczema, and rheumatism, in which the quest for a first cause seems to take the hunter right back to the cell, where the quarry goes to ground and is lost in that infinitely small labyrinth. Vaguely we perceive that the essential cause of such diseases, of which equine colic may well be an example, is an interaction between a disordered cellular life and a disorderly environmental influence. That, at present, is as far as we can get, but it is enough to make a thoughtful worker extremely disinclined to believe that so obscure a disease as colic can be "cured" by so clumsy a means as the customary colic "drench."

Why should colic, as far as is known, be found only in man and equines, and in the latter only among stabled working animals as a rule? Again the search for the true answer takes us back to the cell. In the horse and man above all other mammals the pace of metabolism has been speeded up to an unnatural extent. The modern working horse has been brought, by artificial selection and an unnatural environment, to a state of vital and mechanical imbalance that readily leads to over-strain or pathogenic fatigue. The many well-known forms of arthritis and exostoses, as well as "roaring" and "shivering" and "stringhalt" are, in the writer's opinion, obvious consequences of this tendency to cell-insufficiency, and it seems probable that colic is in the same class.

In the light of this possibly true explanation we may imagine colic as occurring rather suddenly, as the result of a temporary or permanent interruption in the tremendously speeded-up and highly complicated physico-chemical mechanism of the gastro-intestinal system. The interruption is permanent and fatal when it takes the form of an incarceration or displacement of the bowel or stomach. It is also permanent and fatal in those less

common cases where there is an acute enteritis or a "bowel apoplexy," with sanguineous oedema of the intestinal wall.

The interruption is temporary and recovery spontaneous when there is no incarceration, or displacement, or acute inflammation and when the tension or distension of the bowel has not led to rupture, asphyxiation, or syncope. What one might call "freak" causes of gastro-intestinal disturbance such as calculus may or may not occasion a fatal colic.

Some hold that incarceration or displacement commonly, and even usually, occur as a consequence of an otherwise temporary colic, but there is no experimental evidence on that point, or at least none that can be trusted. On the contrary it has been the experience of the writer, and of others, that in a fatal displacement there is sometimes the history of a slip, or a fall, or of excessive muscular strain with fatigue, a few hours before admittance to treatment for colic. Some text books allege that flatulence may be a cause of colic and that colic is often due to flatulent distension of the stomach. Again evidence is lacking to prove this allegation. The writer for many years passed the stomach tube in nearly every case of colic in a busy London horse practice, and in no instance was there a very forcible discharge of gas, as might be expected from a tympanitic stomach; nor in any instance did the mere passing of the tube soon relieve the colic, as it surely would do, had the flatulence been the cause. Similarly, the puncturing of a flatulent colon may lead to a very forcible discharge of gas, but the colic is not thereby speedily cured, as would be expected if the flatulence had been the cause of the spasms. All this clinical evidence suggests that flatulence is an effect rather than a cause of colic.

The writer has not been able to find out if inflation experiments have been carried out to ascertain whether flatulent distension can cause characteristic colic, but he has no doubt that such experiments would have negative results. Some writers discuss gastric tympany in the horse at length, and claim that this condition is a common cause of fatal colic. Abnormal and dangerous distension of the equine stomach with food is often found on post-mortem examination of animals that have died from ruptured stomach or asphyxiation, but the writer has never seen a really tympanitic stomach on the post-mortem examination of a horse or mule. This negative evidence has been obtained in the course of very many years of horse practice and after carrying out a large number of post-mortems, consequently it is reasonable to think that this condition, if it occurs at all, must be as rare as flatulent distension of the large bowel is common.

When colic occurs, in conjunction with and partly as the result of, indigestion from over-eating, or wrong eating, or eating when fatigued, the complete disorganization of bowel function leads very quickly to increased food fermentation and the excessive formation of gas. Thus when one is certain that flatulence must, for obvious reasons, always be a result of colic,

it seems unnecessary to advance the theory that colic is a result of flatulence. Excessive flatulence, and even moderate flatulence if localized, may greatly increase the danger and the distress of colic, and it is probably this fact that has led to the idea that flatulence is a common cause of colic.

Prevention of Colic.

Colic, as a rule, is a preventible disease. Where the stable management is perfect, and the work is not heavy, colic is rare, and fatal colic is almost unknown. The heavier cross-breeds, of the "vanner" type, are more susceptible, and the writer has never seen colic in a donkey. The main contributory causes of colic are wrong feeding, and over-fatigue or strain.

If the food is of the right sort, given in the proper amounts at the right time, if watering is regulated, and if over-fatigue or over-strain are avoided, colic seldom occurs. In the Southern Hemisphere the commonest food-cause of colic is lucerne, either green, or fed as so-called lucerne "hay." The writer, not being a local agricultural expert, cannot speak with certainty upon the point, but he believes that much of the very green lucerne "hay" on the market to-day is not hay in the proper sense of that word, but is unfermented dried green lucerne, and not always well-dried at that.

Before the Great War, in the Orange Free State at least, much of the lucerne "hay" on the market was genuine hay of a golden brown colour. For some years the writer was a member of a daily forage-board for passing or rejecting hay for the military purposes of a large number of mounted troops, and at first he rejected all the very green lucerne "hay" which was known to be an exciting cause of colic among the heavy artillery horses.

These horses received up to 14 pounds of lucerne hay a day: a dangerous ration if the hay has not been properly fermented in the making. Later on the proportion of very green and apparently uncured hay increased, so that its rejection became impracticable, and to-day it is not easy to get the well-made genuine hay. In military experience the excessive ingestion of sand may be a common cause of colic, and it may be necessary to keep canvas nosebags on animals picketed on sandy ground to prevent it.

Treatment of Colic.

There can be hardly any disease of animals, and there is certainly no disease of the horse, for which so many "cures" have been claimed, as for colic. Experience teaches the clinician that the greater the number of alleged "cures," the less is the probability that there is any real drug remedy for a given disease.

It stands to reason that if there had been any specific drug for equine colic among the many that are used, it would have been discovered long ago. We may be sure that the trained ingenuity that was able to discover the specific treatments for milk fever, red-water, actinomycosis, and several

other diseases would not have utterly failed as regards colic if there had been a specific cure in the pharmacopoeia. Most workers, while admitting that there is no single drug that may be regarded as a specific, hold that purgation in general, and some purgatives or intestinal "stimulants" in particular, help to cure colic. They believe that the cure of colic is, in the main, a matter of sweeping the more or less fermenting food out of the stomach and intestine by purgative means, and they think they can re-start the suspended gastro-intestinal mechanism by the same gingering-up drug-agency. In addition, many maintain, and with some it is almost a religious doctrine, that turpentine combined with a purgative or a fixed oil will arrest the fermentation by its antiseptic power, while the aloes or oil sweeps the bowel clean.

There is a great deal of statistical evidence to suggest that these theories are not well founded. Colic in the British army, long before and during the Great War, was treated with just as much success as in any known civil practice without turpentine or a fixed oil, and often without a purgative at all. When a purgative was given, it was a four drachm aloes ball, strongly encased in gelatine, slow to dissolve in the stomach, usually given by a soldier, and often found afterwards on the stable floor. Aloes is not, in conventional ball form, a quickly acting drug and it is likely that in most if not all instances the fate of the animal had been decided long before the aloes had the effect imagined by the purgative school of physicians. The extent and evidential value of this military experience should not be under-estimated. No other body of statistical experience on this subject can compare in volume, at least, with the records of the British Army. The writer treated hundreds of cases of colic in a London practice without giving any purgative drug, unless half a pound of common salt in two gallons of water via the stomach-tube can be so described. Few deaths occurred.

In the early part of this article an effort was made to show that the first cause of colic must be looked for in a disorder of the chemistry of the cell, and it is hard to see how a rough and ready purgative could restore to the cell, in such a short space of time, the order that is needed to effect the commonly quick recovery from colic. As a clinician, one very often sees a horse in violent spasm and agony, and in about two or three hours — long before a purgative drug could act — a complete recovery. Here, quite obviously, the recovery has been spontaneous, due to the restored activity or harmony of the chemistry of the cell and the consequent resumption of normal gastro-intestinal function. How can one expedite this wholly unknown metamorphosis by means of any drug known, and, if one can, why is it that, after more than a hundred years of recorded clinical observation all over the world, there is no proof of any drug's efficacy? The writer has tried nearly every known sort of drug treatment for colic, and he also used the saline solution of Lang by means of the stomach tube for several years. He now affirms that, in his hands, it has not mattered

whether a drug was used or not. The results have always been about the same; from ten to fifteen per cent. of deaths from incarceration, or displacement, or enteritis, or rupture, or syncope (very few of the last), and from eighty-five to ninety per cent, of recoveries.

Quite recently, Gunning (1938), practising in England, has claimed success from the use of acetylcholine (half gramme doses in 5 cc. solution) injected once or more, at two-hourly intervals. The results given by Gunning, fifteen per cent. deaths, are not unusual.

Gunning claims, in effect, that acetylcholine is a specific remedy for a type of colic which, he is convinced, primarily arises from an impacted small intestine and which, he says, may cause death of itself, without displacement or rupture. He describes the post-mortem on one of two such cases he treated before he began to use acetylcholine. The stomach was greatly distended with food and liquid, which included the medicines he had given per os. Impaction of the small intestine with dry ingesta was observed for some feet, starting at the ileo-caecal valve. There was no rupture. Since he has used acetylcholine, that is for the three years 1934 to 1937, he has made nineteen post-mortems without finding a similar condition of the stomach and small intestines.

In my experience the type of fatal case showing a greatly engorged stomach with apparently no onward passage of solid or liquid ingesta to the small intestine is rare, unless a rupture is present. One may easily make many more than twenty post-mortems in succession without encountering such a cause of death. Apart from Gunning's evidence not being sufficient to substantiate a claim for the specificity of acetylcholine, his observations are complicated by his using, on the same cases, several other drugs, such as linseed oil, turpentine, arecoline and aloes. If further trials are made with acetylcholine, other drugs should not be used at the same time. It may well be that in acetylcholine we have the key to the future treatment of equine colic, but this hormonic drug must be tested, by itself, with adequate controls, before any useful opinion of its efficacy can be formed.

The writer has observed with satisfaction, and he regards it as a fortunate fact, that narcosis does not seem to retard recovery, or to increase the mortality. Consequently, it would seem to be the humanitarian duty of everyone who treats equine colic to use narcotics to dull the pain and lessen the spasms. For this purpose, chloral hydrate intravenously and atropine subcutaneously, usually in conjunction with morphine, are known to be often satisfactory. In view of the entire absence of proof of the remedial value of such drastic intestinal stimulants as arecoline and eserine, veterinarians ought not to use these potentially torturing drugs. It is submitted that no veterinarian who himself had colic would agree to having these violent agents tried on him and that he should give to his animal patients the same consideration he would demand for himself. This is especially true of

arecoline, which has often caused ruptured stomach, according to the experience of the writer and of others who have used it in the treatment of colic. It has been urged that very small doses of arecoline or the like are harmless, but it is also common knowledge that men, when faced with what they think is a "kill or cure" disease like violent colic, will not be always content with small dosage. Moreover, there is, as already stated, no proof that arecoline and the like, in any dosage, will help to cure colic. It should be a scientific and moral rule that dangerous drugs are not to be used for treating a disease condition, where spontaneous recovery will occur in the great majority of instances if nature is given a chance.

Obviously, the veterinarian who wishes to treat colic rationally should pass the stomach tube whenever possible: even in the most violent cases, when narcotics are given, there is usually a quiet phase when this can be done. It was observed above, on the evidence of the stomach tube, that excessive flatulent distension of the stomach is rare, and that, possibly, it may not exist at all. Nevertheless, while there is doubt there is always a chance that relief may be given by the emission of gases, however small the volume. A slight release of gas has often been observed. After passing the tube, the writer always pumped into the stomach two gallons of water containing half a pound of common salt in solution, but he has no evidence to show that this procedure helped to "cure" acute colic. In sub-acute colic with impaction of the large bowel, cases have always recovered within three days of this saline therapy, and there has been less subsequent disturbance to health than when more drastic and less satisfactory means of purgation have been used.

Some continental authorities have advised gastric lavage (repeated injections through the tube and syphoning off), but the writer has found, in practice that this cannot be done effectually and safely in a severe case of colic, and that it is not necessary in slight cases. An English clinician, Gunning (1938), recently observed that the addition of a large bulk of saline fluid to an already tense stomach or intestine in a state of frequent spasm must increase the pain, and said that he had seen pain increase because of that procedure. The writer has not observed an obvious increase of pain after injecting two gallons of saline fluid, but the possibility exists, and further observations are desirable.

When there is marked flatulence of the colon, the trochar and canula should be used invariably, at the most prominent part of the distension, high up on the right flank, without hesitation and without fear. Forcible exercise should never be given, but the animal should have every freedom to roll or lie as it likes, subject to measures of protection against self-injury through the violence of the spasms.

It is often claimed in text books — especially Continental and American ones — that twist of the large bowel can be certainly diagnosed, and corrected,

either by manipulation from within the rectum, or by forcibly rolling the animal. The writer frankly does not believe in this, excepting as a rare bit of luck, or coincidence, because he has tried the procedure in scores of apparently suitable instances, without any success.

Morphine, in the writer's experience, does not seem, if given by itself, to relieve the pain of colic, and it certainly has no curative effect. Recently, the writer had to listen, in social circumstances that precluded comment, to an account given by an eminent medical man of how he "saved the life" of a valuable race-horse on a Union-Castle liner by giving, subcutaneously, two grains of morphine, and thus "curing" the horse of a violent attack of colic. The horse in the spasms had become violent, and had smashed up the deck loose-box, and the Captain had agreed to its being shot. During the long discussion that preceded this solemn decision, the spasms had subsided, and the medical man asked to be allowed to try the effect of morphine before the execution was carried out. The morphine was given, the spasms did not recur, and the medical man was the hero of the hour. This is how "cures" are born, because no one could dispute the conclusion, based on such clear experimental evidence, of a famous medical man!

A disease like colic in which Nature, in at least eighty per cent. of cases, will effect a dramatic recovery in a short time, is the quack's delight, because, in most cases, he can hardly go wrong with his prescriptions.

Oil and Turpentine Drenches.

It is necessary for the sake of our reputation as scientists, and for economic as well as humanitarian reasons, to deal at some length with the superstition that a mixture of linseed oil and turpentine will "cure" colic. The almost universal belief in this mixture — a belief that is more strongly held in South Africa than anywhere else — is a remarkable instance of the survival of empiricism in veterinary science. The belief is based on a pseudo-scientific assumption that turpentine effectually arrests food fermentation in the stomach and intestines, while the linseed oil acts as a purgative and prevents further food fermentation.

The average dosage is two ounces of turpentine and ten to twenty ounces of oil, but these amounts are nominal, because in practice, as a rule, more or less of the nauseating mixture finds its way on to the person of the operator, on to the ground, down the windpipe, or remains in the bottle. A dosage of more than two ounces of turpentine, indiscriminately prescribed without regard to the body-weight or physical condition of the animal, is dangerous, as is an early repetition of the usual dose.

In the aggregate, large numbers of equines are killed every year by turpentine-poisoning, or by pneumonia due to the maladministration of the conventional turpentine and oil drenches.

On the first day of the Annual Meeting of the South African Veterinary Medical Association at Onderstepoort in October, 1938, it was officially

reported to the conference that several horses had died recently at Graaff-Reinet and elsewhere soon after being given a linseed oil drench containing four ounces of turpentine. It was suggested that these fatalities might have been caused by an impurity in the locally bought turpentine and that all would have been well if pure turpentine had been given. In the writer's experience, and in the common experience of fellow practitioners, consulted by the writer in Britain, more than three ounces of turpentine, of whatever quality, may be a lethal dose for small animals in poor or diseased condition.

If the turpentine and oil drench has, as is widely believed, a curative action on spasmodic colic, the effect can only be due to the turpentine. A fixed oil like linseed, which has no active purgative principle acts, as a laxative, too slowly to exert any curative power on a normally short disease like colic. The value of the mixture, if any, must be in the turpentine. It must be agreed after such vast opportunities for observation, during more than a hundred years of universal use, that if turpentine had any specific curative action on colic it would long ago have been proved. Thus turpentine can only help to cure colic by its supposed chemo-therapeutic action in arresting food fermentation. But the idea that anti-zymotic drugs given by the mouth will rapidly arrest food fermentation in any really beneficial way is practically exploded. Far more efficient anti-zymotic drugs than turpentine have been tried in a much more appropriate setting than in an oily mixture, without satisfactory result.

If the matter is considered rationally it will be clear that a safe dose of turpentine, largely diluted, and obstructed, by admixture with a heavy oil, cannot effectively arrest food fermentation in the vast, capacious and complicated equine stomach and intestines.

The bacteria and moulds that cause fermentation are not like nematode worms, floating freely in the intestinal lumen readily accessible to chemical action. They are buried in food particles, encased in mucus, and generally protected from attack by the large globules of the oily mixture. Moreover the mixture of fixed and essential oil is not being poured into a normal stomach or intestine with normal churning and peristalsis whereby it may be diffused throughout the ingesta. In such circumstances, amounting to what one might call a "traffic block" of the intestinal transport system, one would expect an oily mixture, artificially introduced, to float to the surface, or to become localized, rather than to mix with what is already there. Post-mortem evidence in some instances has actually shown this localization of recently given drugs. The writer can recall one case in which drugs given twenty-four hours before death could be identified at the surface of the food-mass in an over-gorged stomach. There is, in short, no reason to think that the turpentine can act at all beneficially, and certainly not to the extent needed to restore normality to a deranged gastro-intestinal mechanism, in an hour or two. In the opinion of the writer, the suggestion that such a crude means can "cure" so mysterious and rapid a malady as

colic is scientifically absurd, and there is, to his knowledge, no experimental or rational justification for the idea.

Summary.

1. The customary empirical attitude towards a universal disease of the horse is deplored, and an effort is made to show that a rational approach by practitioners to such a common pathological problem must precede the triumph of veterinary science over quackery.

2. It is suggested, in the light of recent physiological research, that the first cause of colic is the disordered reaction of certain ferment-secreting cells to adverse environmental influences.

3. The treatment of colic is discussed and reasons are given to account for the fact that no known drug-treatment, so far, has been proved to cure a disease in which spontaneous recovery is the rule, and death, due to drug-incurable complications, the exception.

4. The invariable use of narcotics and the passing of the stomach tube and the employment of the trochar and canula, when indicated, are urged.

5. A rational attack is made on the worst empirical survival in veterinary practice, namely the abuse of turpentine and linseed oil drenches for all and sundry intestinal troubles of the horse, especially in South Africa.

6. Reasons are given for the abandoning of such dangerous and torturing drugs as arecoline and eserine, or at least for the suspension of their use until experimental proof of their value is available.

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The Significance of the Amino-Acid in Nutrition.

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It is self-evident that the proteins play an important rôle in the animal body. Just as the carbohydrates are the characteristic substances of the vegetable world, so are the proteins characteristic of the animal. In sheep, according to Lawes and Gilbert, they contribute 80 per cent. of the dry, fat-free carcass.

The animal is, directly or indirectly, dependent on plant proteins for its supply of protein. And since the plant proteins differ in constitution from the animal protein, the body must be able to transform the former into the latter. The initial transformation is to a large extent accomplished during the process of digestion. In the alimentary canal the proteins are broken up into amino-acids, the "building stones" of the protein molecule. The freed amino-acids are absorbed and carried away in the blood stream; from them the body selects what are necessary for the synthesis of its own proteins.

The conception of individual amino-acids as important factors in animal nutrition is a relatively new development in biochemistry. Although most of the amino-acids were already discovered by 1900, relatively little was known of their quantitative distribution in the various proteins; and the conception current before that time that all proteins were of equal food value — *i.e.*, were qualitatively similar — dit not favour an advance of knowledge. It is true that Magendi in 1841 emphasised the nutritive inferiority of the protein gelatine; but it is fair to say that in general attention was directed only to the quantity of protein necessary in nutrition, while the question of quality was neglected.

With the commencement of the twentieth century a new impetus was given to the study of the proteins by the methods for the analysis of the products of protein hydrolysis which the work of Kossel and Kütcher and Emil Fischer and his co-workers made available. Hereafter more attention was devoted to the chemical composition of the protein. It was chiefly this new stimulus to the chemical investigation of proteins that led to our being able to distinguish qualitatively between proteins from different sources.

The most important result of this research has been the discovery that many proteins are largely or completely lacking in certain amino-acids: *e.g.*, gliadine (the protein of wheat) contains only small amounts of lysine; zein (of maize) contain no lysine at all and only small amounts of tryptophane; gelatine lacks tryptophane, tyrosine, cystine, valine, isoleucine, and hydroxyglutamic acid (Dakin, 1920).

At least 36 amino-acids have been isolated from the hydrolysates of various proteins. Of these, the biological importance of some 22 has been determined more or less completely, while relatively little is known regarding the significance of the remainder.

Until it was possible successfully to replace the protein in a ration by mixtures of purified amino-acids, the most contradictory opinions were expressed regarding the indispensibility of the different amino-acids. After more than 30 years of research little more could be said than that tryptophane, lysine, and histidine were essential amino-acids, while on the other hand fairly strong evidence had been produced to show that the dibasic amino-acids were non-essential.

The position was considerably clarified by the discovery in 1935 of the indispensibility of threonine, an advance which made possible the successful substitution of protein by mixtures of pure amino-acids. By this means Rose and co-workers (1938) were able to arrive at the following classification of the amino-acids:—

TABLE I.

Classification of Amino-Acids according to their effect on growth.

Indispensable Amino-acids.	**Growth-promoting Isomeric.**	**Dispensable Amino-acids.**
Tryptophane	d– and l–	Glycine
Histidine	d– and l–	Alanine
Phenylalanine	d– and l–	Serine
Methionine	d– and l–	Norleucine
Lysine	d–	Aspartic acid
Leucine	l–	Glutamic acid
Isoleucine	d–	Hydroxyglutamic acid
Valine	d–	Proline
Threonine	d–	Hydroxyproline
Arginine	unknown	Cittrulline
		Tyrosine
		Cystine

According to Rose there are thus ten amino-acids which are essential for normal growth. That a mixture of these ten can promote normal growth has been proved by Meyer and Rose (1938).

The following amino-acids deserve special consideration:

Methionine and Cystine. — In spite of having for more than 20 years been regarded as essential, cystine is now classified by Rose as a non-essential amino-acid. Doubt was first cast on the status of cystine by the discovery of Jackson and Block (1932) that methionine can at least partially replace cystine in a low casein ration. The problem was cleared up by the discovery of Womack and Rose (1937) that methionine alone produces just

as good growth as when combined with cystine: it is thus able to replace cystine entirely. On the other hand cystine in the absence of methionine has no growth-promoting effect, as was shown by the fact that rats receiving cystine alone lost weight just as rapidly as controls receiving neither amino-acid. This work affords definite proof that methionine is essential and further that cystine in the complete absence of methionine has no stimulating effect on growth. It should, however, be remembered that in the presence of small amounts of methionine the addition of cystine greatly augments the nutritive value of a ration which is deficient in these two amino-acids. This fact emphasises the danger of drawing conclusions unless the absolute composition of a ration is known.

Valine. — Previously, very little information was available regarding this amino-acid. Considering its simple structure, one might be inclined to expect the animal body to be able to synthesise it. That such is, however, not the case has been shown by Eysstein and Rose, who have given attention to this substance during the last two years. Deficiency of valine in a ration causes rapid loss of weight, anorexia and finally death. Unlike other amino-acid deficiencies, valine deficiency produces a characteristic symptom-complex. The rats are abnormally sensitive to being touched and their movements lack co-ordination, the gait is staggering and there is a tendency to turn in circles until exhaustion supervenes. The pathogenesis of this syndrome has not yet been determined, although the opinion has been expressed that it is of nervous origin.

Arginine. — It is an interesting observation that on an arginine-free ration rats increase in weight 70 to 80 per cent. more slowly than controls. This is in contrast with other examples of essential amino-acid deficiency, in which the animals lose weight and eventually die. This observation that growth is only slowed is in accord with the earlier conclusion of Scull and Rose (1930) that rats are able to synthesise arginine. Classification of arginine as an essential amino-acid is thus a matter of definition of an essential amino-acid — one necessary for normal growth, but in whose absence growth still occurs, although more slowly.

Amino-acid Requirements for Functions other than Growth.

The nutritive requirements of an animal may be distinguished as follows:—

1. Maintenance requirements.
2. Growth requirements.
3. Reproductive requirements.
4. Milk-production requirements.

Maintenance Requirements. — Little is known of the amino-acid requirements of the adult animal. Data derived from the study of growth indicate clearly that maintenance requirements are distinct from growth requirements. It has repeatedly been observed that a ration insufficient for growth might

nevertheless be capable of satisfying maintenance requirements.

Osborn and Mendell observed that rats were able to live for months on a gliadine ration, but that no growth took place unless lysine was added. Similarly, zein supplemented with tryptophane can fulfil maintenance requirements, but growth does not occur until lysine is added.

From these two observations it appears that for maintenance lysine is necessary at most in small quantities. Hopkins (1916) found that rats receiving only tyrosine, lysine, tryptophane, and histidine lose weight very slowly and may remain for long periods in apparent good health.

It is believed that the study of amino-acid requirements for maintenance has much to contribute to the problem of endogenous nitrogen metabolism of the body.

Reproduction Requirements. — Regarding the effect of an amino-acid deficiency on reproduction, we can again lay claim to but little knowledge. However, there are indications that amino-acids play an important rôle also in this connexion. Thompson (1934) observed a pronounced reproductive superiority of rats receiving a 17 per cent. protein ration over those on a 15 per cent. ration. Eckles and co-workers (1935) noted irregular oestrus cycle in one of their aphosphorosis experiments and ascribed this not to the phosphorus deficiency, but rather to the proteins in the ration. In New Zealand, Cunningham and Hopkirk produced sterility in rats by means of a 65 per cent. protein ration. Their work convinced them that amino-acid deficiency is a cause of sterility. Last year they succeeded in producing testicular atrophy with a 70 per cent. maize or oatmeal ration, which was otherwise well balanced. This sterility was not prevented by vitamin E. But if these maize or oatmeal proteins were replaced by those of wheat, rye, or barley, the testicles developed normally. The authors ascribed these results to an amino-acid deficiency.

More definite proof that the amino-acids play a rôle in reproduction was provided by Pearson (1936). He found that rats placed on a gliadine ration exhibited but two or three normal oestrus cycles before this function lapsed into total abeyance. Normal cycles were restored after a mixture of the hexose bases (lysine, histidine and arginine) had been substituted for an equivalent amount of gliadine. The same result was obtained by administering 0·6 per cent. of lysine in place of the hexose bases. The work of Seegers (1937) still further emphasises the importance of proteins in reproduction. If pregnant rats are placed eight days before term on a protein-free ration, the foetuses suffer nutritional disturbances. Even a 10 per cent. gelatine ration caused a similar nutritional disturbance of the foetuses.

Milk-production Requirements. — Very little is known regarding the extent to which the various amino-acids stimulate milk-production. Morris and Wright (1933) found that a deficiency of lysine and tryptophane caused a fall in milk-production. There is a remarkably close relationship

between the milk-production value of a given protein and its content of lysine and tryptophane.

TABLE II.

	% Lysine per total N.	% Tryptophane per total N.	Biological value for milk-production.
Blood meal	10·04	6·51	73
Pea-meal	7·04	2·56	64
Peanut-meal	4·46	5·20	59

Daggs and Lidfeldt (1938) have shown that the sulphydryl amino-acid cystein or its potential forms (cystine or methionine) stimulate milk production. The effect of other amino-acids on milk production has not yet been determined.

The foregoing survey should leave no doubt of the importance of the amino-acids for the normal functioning of the animal body. However, a wide field lies open for investigation before all the effects of amino-acids on the body are determined.

The Amino-acid Content of Foodstuffs.

So important are the individual amino-acids in nutrition that it is imperative for us to know the amino-acid content of the various food-proteins. In the past a large number of proteins have been isolated from the commoner foodstuffs and their amino-acid content determined. But the proportion in which these isolated proteins occur in the foodstuffs is not known. Consequently there is no basis on which the quantity of a given amino-acid in a foodstuff may be calculated. Furthermore such data would be still more complicated by the free amino-acids or those combined as peptides, since these are not included in such analyses.

Unfortunately it is at present still impossible by chemical analysis to determine the absolute amino-acid content of a foodstuff. It is for this reason that biological methods of investigation still enjoy preference. By these methods the following amino-acids deficiencies of foodstuffs have been determined:

TABLE III.

Foodstuff.	Known amino-acid deficiency.	Known amino-acid sufficiency.
Maize	lysine and tryptophane	——
Oats	lysine	——
Peanut-meal	——	lysine, tryptophane and cystine
Linseed meal	cystine	——
Copra meal	——	cystine and lysine
Lucerne	cystine and methionine	——
Soya beans	cystine	
Sesame meal	cystine	

The abovementioned amino-acid deficiencies of maize and oats were determined by Mitchell and Smuts (1932), while the remaining determinations were made according to the methods of Mitchell. From the table (III) we see that a considerable number of plant proteins are deficient in cystine. This fact assumes especial significance when we remember that the sulphydryl amino-acids are important factors in milk-production. A mixture of such deficient protein is of course no better suited to the requirements of the animal as regards its amino-acid complex. For this reason it is important to know the amino-acid deficiencies of the various food-proteins and to take cognizance of this when composing rations, so that the best use may be made of the foodstuffs employed.

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The Occurrence and Pathogenicity of *Nematodirus* Species in Arid Areas.

By W. J. RYKSEN, Beaufort West.

Amongst the disease-producing nematodes, *Nematodirus* species have received scant attention in veterinary literature. In that area of the Union of South Africa called the Great Karroo, this parasite has been frequently encountered and has revealed hitherto unsuspected pathogenic effects. In this region, helminthiasis of stock is mitigated by climatic factors, types of vegetation, and by the systems of animal husbandry practised.

The low average rainfall of six inches per annum occurs as short spells of rain in summer which, as a rule, are followed by periods of dry, cloudless weather of several months' duration, without dew. Under such conditions larval forms of the common nematodes appear unable to survive in the absence of dense vegetable cover close to the soil.

Secondly, a system of extensive grazing of sheep in jackal-proof camps is practised. About four to six acres of pasturage per sheep are required and camps are seldom less than 1,000 acres in extent. Under such conditions, larval forms, even if hatching conditions for worm eggs are suitable after rains, have a small chance of obtaining entrance to a host.

Thirdly, the type of pasturage, consisting of bare stems, thin leaves and twigs, well above ground level, affords little shelter to larvae. The vegetation consists of shrubs of *Pentzia* (Karroo bush), *Tripteris* (draaibos), *Othonna* (springbokbos), *Chrysocoma* (bitterbos), *Eriocephalus* (kapokbos), *Mesembryanthemum* (vyebos), and *Galenia* (kraalbos). The shrubs are browsed, and as the stock do not graze close to the ground, the chances of obtaining larval forms must be small compared with the chances on grassveld. In addition the available food supply is highly nutritious. Analyses of mixed samples of vegetation show an average composition of protein 10%, carbohydrate 40%, fibre 30%, and soluble ash 10%. Although the inorganic phosphorus content may vary from 0·15% in times of drought to 0·3% under favourable conditions, aphosphorosis rarely occurs and sheep do not voluntarily take licks containing bonemeal or salt. The high nutritive value of the grazing is reflected in the excellent condition and rapid growth of young stock, which in themselves are well known to increase natural resistance to helminthic invasion.

In times of drought, when thousands of flocks are moved to the Orange Free State and other well watered parts for pasturage, such sheep may become heavily infested with *Haemonchus contortus, Oesophagostomes,* or

Chabertia ovina. Infestation with these nematodes, which include in the life-cycle a delicate, free-living larval stage, is lost in about six to twelve months on Karroo pasturage. On the other hand *Nematodirus* species, so commonly found, maintain themselves on account of the resistance to desiccation and strong negative geotropism of their infective larvae.(1)

In the vast majority of post-mortems undertaken as routine investigation of deaths from any cause, *Nematodirus* is the only nematode found in Karroo sheep. Apparently this species does not survive on account of exceptional powers of multiplication. *Nematodirus* eggs are never found in great numbers in faecal samples, even when samples are obtained from severely infested sheep.

Occurrence.

Although *Nematodirus* is extremely common, its presence in sufficient numbers to cause mortality depends on the topography of the farm. On farms where sheep are watered from open dams or vleis, its effects may be seen on the young stock in the absence of any other helminth. In times of drought, when hand feeding has to be resorted to for a couple of months and sheep are kept under conditions of overcrowding, in small paddocks, severe losses may occur in animals up to two years old. Lucerne hay and maize are fed, but loss of condition naturally leads to increased susceptibility.

Symptoms.

Marked emaciation and weakness, without the diarrhoea commonly associated with helminthiasis, are the outstanding features. Visible mucous membranes are pale; the pulse is weak; the temperature is often elevated due to co-existing patchy pneumonia as the result of debility.

Post-mortem Examination.

Hydropericard and/or hydroperitoneum accompanied by gelatinous infiltration are always found in advanced cases. Liver and kidneys show slight fatty changes, and the former is often quite friable. The small intestine, especially its anterior portion, may be packed with *Nematodirus* worms. Its mucous membrane may be dark red in colour or quite pale, if anaemia is very severe. Varying degrees of tumor splenis are seen, associated with a varying extent of pneumonic changes. All stages of red and grey hepatization, indicating chronic pneumonia of lobar type, may be present.

Diagnosis.

It is of importance to differentiate between *Nematodirus* and *Geigeria* or *Bunostomum* in view of the similarity of symptoms shown. *Nematodirus* is a thin-necked pink worm, apparently lying loosely coiled in the lumen of the intestine, whereas the hookworms are thicker, firmly attached, and

straight. Under the microscope the double genital tube of *Nematodirus* affords instant differentiation. The poorly developed mouth capsule and striated neck of this species are also of value in distinguishing it from the hookworms.

Treatment.

Tetracholorethylene mixed with equal parts of medicinal liquid paraffin, in doses up to 20 cc. for adult sheep, has been found most effective. 2·5 cc. of a ten per cent. bluestone solution is given, and is immediately followed by the anthelminthic which is administered slowly by syringe.

The tetrachlorethylene emulsion "Tetram" is as effective as the above mixture, and preferable.

Two treatments at monthly intervals are usually advised, and these are followed by rapid improvement of the condition of the flock.

Control Measures.

Pans and vleis should be camped off, if practicable, and the sheep should be watered from concrete troughs. Under Karroo conditions, two treatments during the autumn will dispose of infestations accumulated during the summer months and will enable the sheep to pass through the winter months in prime condition.

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Gastritis Traumatica and Rumenotomy in Bovines.

By G. KIND, Johannesburg.

Ingestion of all sorts of indigestible matter, especially pieces of metal, such as wire, nails, and even table knives, caused more losses than any other disease on the Rand, when I started private practice in 1924. Agitation, and claims against agents, caused a considerable decrease in these losses. Still, traumatic gastritis is one of the most frequent ailments in cattle and deserves the veterinarian's special attention.

Symptoms.

A variety of pathological changes may be caused by the foreign body:—

1. It may be a blunt object causing an atony of the rumen.
2. It may be a sharp piece of metal slightly damaging the walls, as the reticulum contracts.
3. It may penetrate and cause a localized or a diffuse peritonitis.
4. It may damage the spleen, liver, heart, or lungs.

Accordingly the symptoms vary. Ruminal movements may still be heard, but may cease abruptly as if stopped by a pain; or their peculiar sound may indicate too much free liquid in the rumen; or they may have ceased completely. Cessation or marked decrease of defaecation or urination is often a typical symptom. Pain is obvious in most cases and may be determined by palpation in the region of the reticulum. Pain on the diaphragm causes the animal to hold its breath. Localized trembling of the anconaei indicates a pericarditis. Stretching of the neck and head, a hunch-back and disinclination to hollow the back, may be obvious symptoms. One can be sure that there is *no* traumatic gastritis when an animal stretches itself comfortably and hollows its back. Severe signs of toxaemia are present when the liver, spleen or lungs are affected, or when the animal is suffering from a general peritonitis. Disturbances of the circulation are obvious and well known in traumatic pericarditis.

Indications for Rumenotomy.

One's decision whether to operate or not is influenced mainly by economic considerations. Gastrotomy is usually indicated when a diagnosis of traumatic gastritis without pericarditis or toxaemic complications is made. It may also be indicated in apparently incurable and recurrent stomach disorders, in case a blunt foreign body is present, or purely for diagnostic reasons. Animals of high breeding value may be operated on even if the prognosis is unfavourable.

Rumenotomy.

It is easiest to carry out the operation on the standing animal, if its temperament allows this. Thirty to fifty grams of chloral hydrate, in a drench, eliminates hampering ruminal movements. The tissues must be infiltrated with a local anaesthetic. A twelve-inch cut is made vertically between hip and ribs, to the peritoneum, but great care should be taken to leave the peritoneum intact. Then the intact peritoneum is stitched to the rumen with a continuous catgut suture to enclose an oval area about 8″ x 2″. Inflation of the rumen is a great help in preventing aspiration of air in case the peritoneum is opened accidently before it is stitched to the rumen. For this purpose I use a Cermak mouth gag, a rubber stomach tube, and an oxygen cylinder.

Then the rumen is opened. The wound may be kept clean with an oilcloth stitched to a wire loop which is inserted into the wound, or with wide retractors on both sides and a narrow one at the bottom. Offensive food masses, sand, stones, after-birth rags, conglomerations of string and food, and sharp foreign bodies, besides an occasional coin, may have to be removed from the rumen and reticulum. Large abscesses which cannot be reached directly from the outside are opened into the stomach. I do not close the abscess wounds, as recommended by others. One case in which an abscess the size of a child's head in the spleen region was opened from the reticulum recovered permanently.

After a thorough search, the rumen is closed with a catgut suture catching the edges of the wound through the muscularis, and by a second Lambert suture catching the parietal peritoneum and the ruminal wall outside the circular continuous suture and bringing the needle out inside that suture, and continuing in reverse sequences on the other side of the wound. Muscles and skin are sutured separately; the first with catgut and the second with Michel clips. There is always a certain amount of soiling of the wound and this must be well cleaned with disinfectants. Iodoform powder is then dusted freely into the wound. Collodium is used for protecting the skin wound. Rarely, a small abscess forms which has to be opened some days after the operation, but usually no after treatment is necessary; which is important in this country of long distances and hot weather.

Other workers pull the rumen out of the wound and hold it with the aid of tape; and others again stitch it to the outside skin; and some to the peritoneum, after opening it. All these methods have the disadvantage that the rumen collapses as soon as the peritoneal cavity is opened, and air streams in. The rumen is fairly heavy, and tape or stitches are liable to tear out of the wall of the rumen. In some cases the rumen is filled with large quantities of semi-liquid, decomposed masses, which are difficult to control when the animal strains or coughs. There are bound to be occasional accidents that prove fatal, in cases where the peritoneal cavity is not sealed

effectively. It may also rarely happen that the stomach wound opens in spite of the double stitching; this results in a stomach fistula which gradually heals when the rumen is effectively stitched to the parietal peritoneum, whereas otherwise it would take a fatal course. Never have I expeirenced ill results due to adhesions. I have found that these gradually work loose, and, in time, hardly a trace of them is left.

Operation for Traumatic Pericarditis.

The various methods for the treatment of traumatic pericarditis are, in my experience, unsatisfactory. Moussu's method gives temporary relief and reduces the oedema, but hardly enough to make the meat on the fore-quarters fit for human consumption. Rib resection does not give proper drainage, and may lead to a pleural infection. And as it is usually impossible to remove the foreign body through the pericard, a gastrotomy has in any case to be done. I tried trephining the breast bone, a method which gives better drainage. Out of six cases, one recovered completely.

Rumenotomy in Peracute Tympanitis.

In cases of peracute tympanitis when food masses and gas are mixed so that the trocar is of no use, a far cruder method of rumentomy, without shaving or disinfecting, has to be applied: make a cut three to four inches long; the stomach contents will shoot out with tremendous force. Let two attendants hold the wall of the rumen to the abdominal wall. Then stitch the rumen to the muscles with catgut in a continuous suture, and leave the wound open. The stomach fistula will heal gradually.

RESULTS.

Statistics of results are not of much value. They would be unfavourable if all cases of traumatic gastritis, without regard to complications, were operated on. Practically all losses after gastrotomy are due to complications which were caused by the foreign body, before the operation; and which were not diagnosed. In my experience no losses should occur as a sequel to the operation as described. Cases operated within a few hours of the onset of symptoms show nearly 100 per cent. recoveries. One rarely gets these cases, because the farmer usually uses 101 remedies before calling a veterinarian.

When I first did the operation as described in Frick's text book on operations (1912), two out of six cases died as a sequel to the operation. From 1926 to date, I have lost two out of three hundred and forty-four as a direct sequel to the operation. In both cases the peritoneum was torn due to a faulty technique and a general peritonitis set in. This could have been prevented under better conditions, and with more care.

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SUMMARY.

Rumenotomy for the removal of foreign bodies has been practised on the Witwatersrand since 1925. 344 head of cattle were operated on in the way described above, and only 0·6 per cent. died as a result of the operation. The rumen is stitched to the parietal peritoneum before opening the latter, to prevent the peritoneal cavity being filled with air, and to prevent the rumen from collapsing. All the stitching is done with No. 3 or No. 4 catgut. The skin wound is completely closed with silk or Michel clips. Usually no after treatment is necessary. Pericardiotomy by trephining the breastbone, in combination with gastrotomy, has been done, with one permanent recovery out of five animals operated on.

CORRESPONDENCE.

Comment on a Paper by Prof. F. K. Kleine

By Sir ALDO CASTELLANI, K.C.M.G.

23, Harley Street,
Cavendish Square,
London, W.1.
December 6, 1938.

The Editor,
Journal of the South African Veterinary Medical Association,
Onderstepoort, Pretoria, South Africa.

Sir, — A paper by Prof. F. K. Kleine published in the Theiler Memorial Number of your Journal (December, 1936, Vol. VII. No. 4) with the title "Remarks on Sleeping Sickness," has only just been brought to my notice.

With all respect to Prof. Kleine for whose work I have a great admiration, the history he gives of the elucidation of the aetiology of sleeping sickness is completely incorrect.

I beg to quote verbatim and in chronological order certain extracts from the Reports of the Sleeping Sickness Commission of the Royal Society which he has mentioned.

Extract 1, from Report No. 1, by Dr. Aldo Castellani (dated Entebbe, Uganda, April 5, 1903): "I would suggest as a working hypothesis on which to base further investigation that sleeping sickness is due to the species of trypanosome I have found in the cerebrospinal fluid of the patients of

this disease, and that at least in the last stage there is a concomitant streptococcus infection which plays a certain part in the course of the disease."

Extract 2, from Report No. 1, by Colonel David Bruce and Dr. David Nabarro, p. 11 (dated Entebbe, Uganda, May 29, 1903): "The Commission (viz., Col. Bruce and Dr. Nabarro) arrived in Entebbe, Uganda, March 16, and were met by Dr. Castellani, a member of the Sleeping Sickness Commission sent out by the Royal Society in May, 1902.

"Dr. Castellani informed us of the work he had done, one especially interesting observation being that he had discovered trypanosomes in the cerebrospinal fluid in five out of fifteen cases of sleeping sickness. Dr. Castellani remained in Entebbe for three weeks after the arrival of the new commission, and during this time he examined twenty-nine further cases for trypanosomes, with the result that 70 per cent. were found to contain these parasites. Dr. Castellani, we presume, has already published these results. After his departure the commission continued to pursue this line of work."

Extract 3, from Report No. 4, by Col. Bruce and Dr. David Nabarro: "This most interesting discovery of Dr. Castellani's, which was due to his introduction of centrifuging the cerebrospinal fluid in his search for his streptococcus, has been of the utmost possible value to the present commission. It put them at once on the right track and led to the rapid and easy elucidation of the etiology of this hitherto mysterious disease. Without a knowledge of his observation they might have worked for months in the dark, and in truth, they might even have returned to England still ignorant as to the true cause of the disease."

The first Sleeping Sickness Commission consisted of three members: Dr. G. C. Low, Dr. C. Christy and myself. Dr. Low and Dr. Christy have always given me full credit for my researches on the etiology of the disease. Dr. Christy, for instance, in a letter to the Evening Standard, London, September 2, 1931, wrote: "Sir Aldo Castellani was the real discoverer," and adds: "As I was a member of the first Sleeping Sickness Commission (Low, Castellani and Christy) to Uganda in 1902-3, I am in a position to know the facts as they are."

The second Commission consisted of two members: Colonel David Bruce and Dr. David Nabarro, both members arriving in Uganda on the same day by the same boat. Dr. Nabarro therefore is in an unique position to know the inner history of the investigations and I may quote a paragraph from his letter to the British Medical Journal, December 15, 1917:

"In conclusion, I should like to repeat here what I have always stated in previous publications — namely, to Castellani goes the credit of having first discovered the trypanosomes in the cerebrospinal fluid of sleeping sickness patients, of having first connected it with the etiology of sleeping sickness, and of having first published it."

Sir Ronald Ross, in the Journal of Tropical Medicine and Hygiene, October 1, 1926, in a paper with the title "Simian Journalism," wrote the following:

"There cannot be any doubt that the fundamental discovery in the elucidation of the etiology of sleeping sickness was Castellani's observations of trypanosomes in the spinal fluid of sleeping sickness patients." He also wrote: "Considering that Castellani made certain conditions before acquainting Bruce with his discovery, and that this has been publicly confirmed by Nabarro many times (British Medical Journal, October 6, 1917), how can it be said that he (Castellani) attached no importance to his findings and did not see the possible relationship between the trypanosomes and sleeping sickness?"

Sir Ronald Ross, Sir Patrick Manson, Sir Frederick Mott, Laveran, and Golgi in their publications have always given me the credit of having made what they call the fundamental discovery in the elucidation of the aetiology of sleeping sickness; and Paul Ehrlich in a letter to Dr. Albert Chalmers wrote the following: "Die Leute sich auf den Kopf stellen müssen um das Gengentheil zu beweisen."

Yours faithfully,

(Sgd.) *Aldo Castellani of Kisymaio.*

NEWS.

Mr. R. Paine, Officer-in-Charge, Allerton Laboratory, retired on pension in January, after completing nearly 37 years' service, and is starting out in private practice in Pietermaritzburg.

After qualifying in London he joined the Cape of Good Hope Veterinary Division in 1902 and served in various parts of the Cape Province and Transkei till 1934, when he was transferred to Allerton.

Throughout his career Mr. Paine has been an able and enthusiastic worker on behalf of public servants and of the veterinary profession. He was one of the prime movers in the formation of the old Cape Veterinary Medical Association and was the first secretary of this body, serving it in this capacity during the most critical years of its brief existence.

After Union in 1910 he and Mr. J. W. Crowhurst, of Cape Town, who is still an active member of our Association, were chiefly responsible for the Cape Association's sinking its identity and amalgamating with the old Transvaal Veterinary Medical Association, to form the South African Veterinary Association, which was subsequently to become the South African Veterinary Medical Association. These two members were also instrumental

in transferring the funds (a substantial sum) of the Cape Association to the newly formed national body.

At present Mr. Paine is President of the Natal branch of the S.A.V.M.A.

* * *

Mr. A. S. Canham has been transferred from Pretoria to Allerton as Officer-in-Charge while Mr. M. M. Neser, late Senior Veterinary Officer of South West Africa, succeeds him as Senior Veterinary Officer, Transvaal.

* * *

At the Third International Congress of Tropical Medicine the Nocht gold medal was conferred on Dr. P. J. du Toit. The presentation of the medal was made by Prof. Nocht himself, and he referred enthusiastically to the excellent work that is being done at Onderstepoort.

* * *

Captain W. S. B. Clapham, who sustained serious injuries when he crashed in a glider three months ago, is making rapid progress towards complete recovery, and hopes to assume duty in his new post in Basutoland within the next month.

ABSTRACT.

Recherches sur la nature chimique et sur les propriétés biologiques des antigènes somatiques et des toxines des bactéries, by A. Boivin and L. Mesrobeanu, VIe Congrès de Chimie Biologique, Lyon, 11 – 13 Octobre 1937. (*Research on the chemical nature and biological properties of somatic antigens and bacterial toxins.*)

This article reviews some of the work of B. and M. who have carried out extensive research on the isolation of antigens from bacteria.

According to immunologists, the O or somatic antigen (the endotoxin) of Gram-negative bacteria is the important one in producing virulence and immunity. It is attached to the bacterial body and is liberated with some difficulty in culture media. *S. typhi* and *V. cholerae* are examples of two microbes which produce endotoxins. On the other hand, bacteria such as *C. diphtheriae* and *Cl. tetani* produce exotoxins which diffuse easily into the surrounding culture. Exotoxins differ from endotoxins in being more toxic, more antigenic, and less heat stable.

From the pneumococcus, Heidelberger and his colleagues isolated a polysaccharide, not itself capable of acting as an antigen, but precipitable, specifically, by antisera prepared in an animal immunized with the " whole "

pneumococcus. Landsteiner named such substances "haptenes." Thus the complete antigen, as it exists in the bacterium, is the result of the union of the polysaccharide responsible for specificity and some other substance, the loss of which deprives the polysaccharide of antigenicity. Heidelberger considers the "other substance" to be a protein, but B. and M. point out that such a carbohydrate-protein complex, endowed with the properties of an O antigen has not yet been isolated from any bacterium.

From *S. aertrycke* the authors (1933) isolated a substance corresponding to both the O antigen and to the endotoxin. This material, chemically pure or nearly so, contained no protein but was a glycolipoid complex,* made up of the union of a specific polysaccharide and fatty acids. Raistrick and Topley independently, but by a different method, isolated the same substance from *S. aertrycke.*

To obtain the substance, the bacterial bodies are washed and then treated with trichloracetic acid. This treatment coagulates the proteins which are removed by centrifugation; the polysaccharide-fatty acid complex remains in solution. The trichloracetic acid is removed by dialysis, and finally the antigen is purified by precipitation with alcohol or acetone. Raistrick and Topley removed the protein by tryptic digestion and then purified the antigen by precipitation with alcohol.

The antigen is the union of a polysaccharide with fatty acids and also with acetic acid and phosphoric acid. These two latter acids make up only five to ten per cent. of the antigenic molecule, whilst the polysaccharide constitutes from one-half to two-thirds, and the fatty acids about one-fifth to one-quarter. Heating the complex in a feebly acid medium splits it into fatty acids and the polysaccharide, and this latter may be decomposed by the use of a more strongly acid medium.

Immunologically the complex is the same as the O antigen and the endotoxin. It stimulates the formation of O agglutinins; is precipitated by O agglutinins formed by immunizing an animal with bacterial bodies; is toxic (mouse and rabbit) and is neutralized by antibodies formed by immunizing an animal with the complex. The polysaccharide is neither toxic nor antigenic but is precipitated by "anti-complex" serum.

By the trichloracetic acid technique, B. and M. were able to isolate the complex only from Gram-negative bacteria (*e.g.* typhoid and paratyphoid, dysentery and proteus) and not from Gram-positive organisms (*e.g.* anthrax, staphylococcus and diphtheria). From these the polysaccharide only could be isolated.

Exotoxins do not dialyse easily, are precipitable by protein precipitants, and are more or less rapidly destroyed by ferments, *i.e.* they act like proteins. Further, the most highly active preparations always contain nitrogen. By the trichloracetic method, diphtheria toxin may be brought to a state bordering on purity. In this case the precipitate and not the

supernatant contains the active material. By other purification methods, Eaton and Pappenheimer obtained results almost identical with those of B. and M.

Shiga's dysentery bacillus is an example of a microbe which forms two toxins, an exo- and an endo-toxin. By the trichloracetic acid method, these may be separated.

J. H. M.

* un complexe glucidolipidique.

REVIEW.

The author states: "This publication* is designed primarily for the use of veterinary practitioners, students, and others interested in the identification of the internal parasites of domestic mammals. It is meant to bridge the gap between the very complicated science of taxonomy of parasites and the man in the field."

The publication is in the form of a key, based primarily on the host, organ and lesion specificity and secondarily on gross morphological characters. In most cases, however, it is possible to identify the parasites without the aid of a microscope.

The author has undoubtedly given much time and thought to the compilation of this key and has admirably succeeded in producing a very practical guide which will aid in making parasitology a more popular subject with veterinarians.

It is stated that "Whenever possible aberrant forms have been included in the keys. However, when their inclusion would result in confusion with the more common forms they have been omitted as have most of the species which have not been reported from North America." Certain parasites which are of importance in South Africa and other countries are therefore not included, such as the Schistosomes, eye-worms of cattle (*Thelazia*), *Onchocerca* species of cattle, *Gastrophilus pecorum*. Young stages of parasites migrating in the body and of pathogenic significance might have been included, especially those which are well visible, such as immature stages of *Paramphistomes* in the intestine, nymphs of *Linguatulidae* and immature *Cysticercus tenuicollis* in the liver. The latter will usually be taken for flukes and probably identified as *Dicrocoelium lanceatum*. In spite of a few possible breakdowns of this type, however, the key is most useful and can be warmly recommended.

H. O. M.

* *Practical Identification of Endoparasites for Veterinarians*, by J. H. Whitlock, D.V.M., M.S. 1938, pp. III + 35. Burgess Publishing Co., Minneapolis, Minn., U.S.A.

Publications of the Imperial Agricultural Bureaux.

A conference was held in 1936 to discuss the work of the various Imperial Agricultural Bureaux and it was decided to do everything possible to make the publications of the Bureaux more widely known, with a view to recruiting more subscribers. To further this end the prices of the publications have been specially reduced for countries participating in the Bureaux system. The Union is a participant in this system. Below is a price list of the publications as well as a list of the present subscribers in South Africa. Any member of the Association who wishes to become a subscriber should send his address and subscription direct to the Secretary, Imperial Agricultural Bureaux, 2, Queen Anne's Gate Buildings, London, S.W.1, and also advise the Editor of this journal.

Names and prices of the regular Journals of the Bureaux and Institutes.

	Rate to Empire Subscribers.
List of Publications relating to Soils and Fertilizers	£1 0 0*
The Veterinary Bulletin	£2 0 0*
Index Veterinarius	£5 0 0
Nutrition Abstracts and Reviews	£2 2 0*
Plant Breeding Abstracts	£1 0 0*
Herbage Abstracts	£1 0 0*
Herbage Reviews	£0 15 0*
Horticultural Abstracts	£1 0 0*
Animal Breeding Abstracts	£1 0 0*
Helminthological Abstracts	£1 10 0*
Bulletin of Entomological Research	£1 10 0
Review of Applied Entomology, Series "A"	£1 10 0
Review of Applied Entomology ,Series "B"	£0 15 0
Review of Applied Mycology	£1 4 0

Note. — The items of cost starred (*) make up the sum of £11 7s. referred to as the price of one complete set of "Bureau publications." Index Veterinarius issued on sale terms only.

List of Subscribers to Bureau Publications, Union of South Africa.

1. **Soil Science.** (The Imperial Bureau of Soil Science).

List of Publications relating to Soil and Fertilizers.

Librarian, Public Library, Market Square, Johannesburg.
University of Witwatersrand, Johannesburg.
J. Lintner, Esq., P.O. Box 2033, Durban.
Messrs. Safco Ltd., Umbilo, Durban.
H. Klintworth, Esq., Department of Agriculture and Forestry, Pretoria.
Ch. Horticulturist, African Realty Trust, Ltd., Johannesburg.

2. **Animal Health.** (The Imperial Bureau of Animal Health).

Veterinary Bulletin.

The Principal, Stellenbosch-Elsenburg Agricultural College, Stellenbosch.
The Municipal Library, Kerk Street, Johannesburg.
Dr. E. M. Robinson, P.O. Onderstepoort, Pretoria.
The Librarian, University of Pretoria, Pretoria.
Messrs. J. L. van Schaik, Ltd., Church Street Central, Pretoria, Transvaal.
G. B. Purvis, Esq., F.R.C.V.S., 21 Park Road, Grahamstown, Cape Province.
Chief of the General Staff, Department of Defence, Pretoria (two copies).
The Librarian, Witwatersrand Medical Library, Hospital Street, Johannesburg.
The Senior Veterinary Officer, Windhoek, South West Africa.
Director of Veterinary Services, Onderstepoort.

3. **Animal Nutrition** (The Imperial Bureau of Animal Nutrition).

Nutrition Abstracts and Reviews.

Messrs. Tiger Oats Co., P.O. Box 120, Maitland, Cape Province.
The Principal, Grootfontein School of Agriculture, Middelburg, Cape Province.
The Principal, Stellenbosch-Elsenburg College of Agriculture, Stellenbosch, C.P.
E. E. Buettner, Esq., 40, Kimberley Road, East London, Cape Province.
The Librarian, University of Cape Town Medical School, Mowbray, Cape Town.
The Librarian, University of Witwatersrand, P.O. Box 1175, Johannesburg.
The Officer in Charge, Government Chemical Laboratories, Box 1080, Johannesburg.
The General Manager, Messrs. Safco Ltd., Umbilo, Durban.
Messrs. Tomango Ltd., Private Bag, Durban, Natal.
The Principal, School of Agriculture, Glen, Orange Free State.
The Principal, School of Agriculture, Potchefstroom.
Dr. J. H. Kellermann, Veterinary Research Laboratories, Onderstepoort, Pretoria.
The Veterinary Research Laboratory, Onderstepoort, Pretoria.
The Librarian, Division of Plant Industry, Department of Agriculture, Box 994, Pretoria.
The Librarian, University of Pretoria, Transvaal.

4. **Plant Breeding and Genetics.** (The Imperial Bureau of Plant Breeding and Genetics).

Plant Breeding Abstracts.

Jacq. Sellschop, Esq., P.O. Box 37, Upington, C.P.
T. D. Hall, Esq., Messrs. African Explosives and Ind. Ltd., Chamber of Mines Building, Holland Street, Johannesburg.
J. M. Hector, Esq., Dept. of Agric. Botany, University of Pretoria, Pretoria, Tvl.
Librarian, Cotton Breeding Station, Barberton, Transvaal.

5. **Pastures and Forage Crops.** (The Imperial Bureau of Pastures and Forage Crops).

Herbage Abstracts — Herbage Reviews.

Agric. Adviser, Messrs. African Explosives and Industries Ltd., P.O. Box 1122, Johannesburg.
Messrs. African Explosives and Industries Ltd., Umbogintwini Factory, Umbogintwini, Durban, Natal.
Chief Horticulturist, African Realty Trust Ltd., P.O. Box 3044, Johannesburg.
Chief, Division of Plant Industry, Dept. of Agriculture and Forestry, Pretoria.
Prof. R. Lindsay Robb, Faculty of Afrigulture, University of Pretoria, Transvaal.
J. P. J. van Vuuren, Esq., P.O. Box 13, Bothaville, Orange Free State.

6. **Horticulture and Plantation Crops.** (The Imperial Bureau of Horticulture and Plantation Crops).

Horticultural Abstracts.

Chief Horticulturist, African Realty Trust Ltd., Johannesburg.
Technical Book Co. Ltd., Cape Town.
Citrus Research Officer, S.R.C.C.C., Addo, Cape Province.
Chief Fruit Inspector, Cape Town.
Chief, Division of Plant Industry, Dept. of Agriculture, Pretoria.
R. H. Marloth, Esq., Nelspruit, Eastern Transvaal.
Miss K. Murray, Cape Province.
I. A. Dicey, Esq., Laurensford Estate, Somerset West, Cape Province.
South African Potash Co., Balfour House, Cape Town.
South African Book Store, P.O. Box 1016, Pretoria.

7. **Animal Breeding and Genetics.** (The Imperial Bureau of Animal Breeding and Genetics).

Animal Breeding Abstracts.

L. L. Roux, Esq., Vet. Res. Laboratory, Ermelo.
School of Agriculture, Natal.
Director of Veterinary Services, Onderstepoort.
G. N. Murray, Esq., Pretoria.
College of Agriculture and Forestry, Stellenbosch.
University of Pretoria, Transvaal.

8. **Parasitology** (Helminthology). [The Imperial Bureau of Agricultural Parasitology (Helminthology)].

Helminthological Abstracts.

Entomologist, Cape Explosive Works Ltd., P.O. Dynamite Factory, Cape Province.
A. B. Purvis, Esq., F.R.C.V.S., 21, Park Road, Grahamstown, Cape Province.
Director of Veterinary Services, Onderstepoort.

Jl: S.A.V.M.A.,
X(2): 44 – 55.
1939.

Some Observations on Dourine.

By G. de KOCK, Section of Pathology, E. M. ROBINSON, Section of Bacteriology, and B. S. PARKIN, Section of Medicine, Onderstepoort.

Before describing the experimental work which has been done on dourine in recent years at Onderstepoort, a brief reference will be made to some of the earlier work on the disease in South Africa. Theiler (1918) describes the earlier observations made on the existence of dourine in South Africa. The first were by Lyons in the Hay district in 1914, and in 1916 Andrews found the disease to be very widespread in the North West Cape area. The symptoms appeared milder than those in other parts of the world. A number of mares were brought to Onderstepoort and kept under observation for long periods, but no trypanosomes were ever found in them. In 1917, in order to determine whether the disease was really dourine, sera were sent to Watson in Canada, and all were returned as positive to the complement fixation test. Walker (1919) inoculated puppies with blood from a case of dourine and one of them showed trypanosomes on one day, but the strain was lost.

Routine testing by means of complement fixation was commenced on a small scale in 1923. An imported strain of *T. equiperdum* was used for the preparation of antigen. No further articles have been published on the disease in South Africa except one by Schulz (1935), who recorded his experiences with dourine in the Griqualand West area during the eradication campaign which had been undertaken there. Schulz made some valuable observations to which reference is made in the section of this paper devoted to observations on foals.

Before describing the experimental work which has been carried out, a few explanatory notes on the complement fixation test will be given.

Before using sera for the test they are heated to a temperature of 60° to 62°C for half an hour in order to destroy the complement which might be present in fresh sera, and certain bodies found in sera which interfere with the reaction of complement fixation. In the test itself it has been found by experience that if the serum of an animal causes a positive complement fixation in an amount of 0·2 c.c. the animal must be regarded as infected. In some cases the antibodies are present in large amounts, and the serum may give a positive reaction in as small a quantity as 0·001 c.c. In the case of mule and donkey serum there is a tendency for the serum itself to cause complement fixation even when no antigen has been used with it. This fixation of complement in the absence of antigen (trypanosome emulsion) is called a non-specific reaction.

Before tests are made, the complement is carefully titrated and the exact quantity to be used is determined. In the routine tests on sera sent in from the field, these are only tested in amounts of 0·2 c.c. as it is only necessary to divide positive from negative sera, but in the case of the sera from experimental animals, these have been tested monthly, and from time to time a determination of the actual amount of antibodies in them carried out.

In sera from the field where an incomplete fixation is obtained with 0·2 c.c. a retest is asked for about a month later.

(1) *Observations on Foals of Infected Mares.*

Few observations on dourine infection in foals have been published. Hutyra and Marek (1938) mentioned that foals might become infected from mares with dourine, through the milk. Boes in a discussion on an article by Cinea (1933) mentioned that in Belgium in 1921-22 he observed that thirty per cent. of foals from infected mares reacted to the complement fixation test. Infection was considered to be through the milk as the foals ceased to react when they stopped drinking the milk of the mares.

Schulz (1935), in referring to the infection of foals from mares with dourine, stated that the percentage of infection was higher in foals from clinically infected mares than from those not showing clinical symptoms. In exceptional cases foals from temporarily non-reacting mares reacted. Sucking foals, though infected, showed no clinical symptoms. He thought some cases might be considered as having a latent infection, seeing that they reacted to the test for a few months. The observation was made that a positively reacting foal might become negative in the course of a few months. He thought that dourine could be transmitted to foals by the milk or by material from the udder soiled by vaginal exudate. Infection via the placenta or via the conjunctiva he considered unlikely.

In the present series of experiments the reactions of a number of infected mares and their foals were studied. A number of abortions occurred and a number of foals were born dead. From all these blood was obtained but no positive reactor was found. Of fourteen foals born in 1937, four survived and are still alive, and one lived for a few months. Thus an opportunity was offered for the regular testing of foals. The other nine foals either died at birth or lived a few days.

The fact that no reactions were obtained from newly born foals or in foetuses has a parallel in other diseases such as contagious abortion in cattle, in which diseased foetuses and newly born calves from infected cows always give negative reactions. Similarly lambs of ewes immunized against bloedpens have no antitoxin in their blood at birth.

With these observations in mind, the reactions of the foals were followed up, and it was found that a similar type of reaction occurred in

the animals examined at Onderstepoort. The first two foals were examined for the first time at three weeks, when they gave positive reactions. One foal, 21479, remained with its dam until weaned and reacted positively until four months old when it became negative, and remained so for a year. It was then inoculated with vaginal washings of a dourine mare showing trypanosomes, and again became positive. It has remained positive, but all attempts to demonstrate trypanosomes in it have failed. It is quite probable that the foal was reinfected with the inoculated material.

The other foal 21472 lost its mother when a week old. It was brought up by hand and gave a positive reaction at the first test carried out when it was three weeks old. It remained positive for three months in spite of being fed on cow's milk. It then ceased to react and remained negative until its death at eight months. It has been shown by Mason, Dalling and Gordon (1930) that in lambs born of ewes hyper-immunized against bloedpens, the antitoxin persists for about two months in the blood even if the lambs are removed from the ewes soon after birth. The antibodies of a homologous type are absorbed in large amounts from the colostral milk and are only slowly eliminated. It is probable that the persistence of antibodies in foals can be explained in the same way.

This year, two foals from reacting mares, both of which reacted positively within twenty-four hours of birth, were removed from the mares when a week old and are being brought up on cow's milk modified to resemble that of the mare. Observations on these two foals should confirm or disprove the idea that passively transmitted antibodies persist long after a foal has ceased to drink milk containing antibodies. The reservation must, however, always be made that the foals are possibly infected. In that case no explanation of the reaction within a few hours of birth or of the subsequent disappearance of the positive reaction can be given.

On two farms where testing was carried out on a big scale, foals nine months to a year old, the progeny of infected and reacting mares, reacted strongly. In one case a reacting foal seven months old was brought to Onderstepoort and kept under observation for five months, when it died. It remained positive constantly.

These results in the field are difficult to reconcile with those obtained at Onderstepoort, but one may perhaps assume that in the field-cases referred to actual infection had occurred. Schulz (1935) mentioned that in some of his observations the foals lost their positive reactions.

The table at the end of this paper summarizes the results obtained with all the foals under observation. It will be seen that three of the foals, 21622, A, and B, died at birth and gave negative reactions. One, 21561, lived for twenty-four hours, but did not suck and gave a negative reaction after death. It was only possible in two cases to follow the reactions from birth. In one of these, 21555,

a negative reaction was obtained at birth, but at twenty-four hours the reaction was strongly positive and remained so for three months, when it became negative and has remained so for a year. In the other case, 21556, a negative reaction was obtained at birth, but at twenty-four hours it was positive, though weak, and gradually became weaker until it disappeared by the fourth day. The foal was weak at birth and, though it did suck at first, became gradually weaker and died on the twelfth day.

In the case of another foal, 21632, a positive reaction was obtained eight hours after birth, but the reaction only persisted for six weeks, after which it was negative until death occurred at four months.

Another foal, 21578, was born to a mare in the low-antibody group. As explained in the section of this paper devoted to the observations on infected mares, this group consisted of mares which were regarded as giving reactions just within the titre accepted for judging animals as positive. The foal reacted negatively at birth and never became positive. The colostral milk of this mare showed a low concentration of antibodies, and apparently the foal obtained insufficient to cause a positive reaction to develop.

Further tests must be carried out on the concentration of antibodies in colostral milk and in milk produced during full lactation. Further attempts must be made to demonstrate *T. equiperdum* in the milk of infected mares, as van Rensburg and Parkin (private communication) found trypanosomes in the udder of a dead infected mare at Onderstepoort.

(2) *Observations on the Onderstepoort Troop of Dourine-infected Horses.*

In 1934 a commencement was made with the building up of a troop of horses infected with dourine. About fifty mares in all were obtained from Griqualand West, though owing to deaths there have not usually been more than forty on hand at any one time. Over a period of four years, mares have been obtained from different farms and at present there are only twenty-two left, some of which have been under observation for the whole period. It is interesting to note that no animal has recovered, as judged by the results of the complement fixation tests. Some mares bought four years ago react as strongly now as they did when purchased.

It was found that a few animals gave weaker reactions than the others. In these cases the reactions could be regarded as positive, but only just. It is usual for the majority of the positive reactors to give reactions in a dose of 0·2 c.c., of serum down to 0·001 c.c. at least. The weak reactors which give reactions in 0·2 c.c. but rarely lower, are termed low antibody cases. They remain consistently low and their reactions do not vary much. Before the improvement in the preservation of the antigen was introduced (preservation at temperatures of –12°C instead of at about 4 to 10°C),

there was a tendency for the low antibody cases to give incomplete positive reactions even in 0·2 c.c. when the antigen was over three weeks old.

The animals in the dourine experiments have been kept in the open in camps and fed daily. At present the only food they can get is that supplied to them. Most of the horses have remained in fair condition and in the summer months some have attained excellent condition, in spite of the infection. It is difficult to judge the relation of the infection to the condition of the animals as no uninfected controls have been kept under similar conditions. There is no doubt, however, that some horses died as a result of dourine infection. Nine died from horsesickness and ten were killed on account of cachexia. Other causes of death were impaction, dystokia, pleuritis, fractures, etc.

In order to make observations on the infectivity of mares with dourine, two stallions were allowed to run with the main troop of mares at different times. Both stallions became infected and reacted strongly to the complement fixation test. One of these stallions, 21400, developed enlargement of the penis and testicles and has lost much in condition. The other, 21398, has remained in excellent condition and up to the present has shown no symptoms.

The infected stallion 21400 was placed in a separate camp with five susceptible mares. Within five months, three of these mares commenced to react to the complement fixation test. One of them, 21483, became very thin and had to be killed. Immediately after death numerous trypanosomes were found in saline washings from the vagina. Vaginal washings, milk, amniotic, and allantoic fluids were injected into white mice, but no infection of these animals occurred. Vaginal washings of the other two positively reacting mares were then examined and trypanosomes were found without difficulty.

A large number of mice were inoculated with vaginal washings, but none became infected. It is of interest to note that although further and repeated examinations of both mares were made within the three to six months after trypanosomes were first seen, no parasites were found. It is possible that these are only frequent in the vagina in the early stages of infection, as repeated attempts have been made to detect the parasites in the vaginal washings of old cases at Onderstepoort without success.

One of the mares, 21044, died about a year after commencing to react to the complement fixation test. At post-mortem immediately after death, vaginal washings were made and injected into twenty mice and two rabbits. The cerebro-spinal fluid and blood were spun and the deposit examined for trypanosomes, but none were found. The cerebro-spinal fluid was injected into twenty mice intraperitoneally. All the animals injected with the material taken at post-mortem remained healthy.

Another experiment was carried out in which a healthy stallion, 21372,

was placed with five mares which were grouped in the low-antibody class. The experiment has been continued now for about eighteen months, but so far the stallion has not become positive to the complement fixation test. The five mares must be considered infected as they react to the test. Two have foaled recently and two others appear to be in foal, so the stallion must have served the mares. This experiment will be continued and regular monthly tests made on the animals in it and the main troop of infected horses to see whether recoveries ultimately take place and to study the reactions of foals.

Further transmission experiments will be made in the attempt to isolate a South African strain of *T. equiperdum*. Great difficulty has been experienced in most countries in infecting laboratory animals with trypanosomes obtained from natural cases and some workers consider rabbits and dogs should be used instead of mice or guineapigs. Fresh susceptible mares have been placed in contact with an infected stallion in order to obtain early cases for transmission work.

Recently (20·10·38) a susceptible mare 21176, which had been placed in contact with an infected stallion 21398 on 1·9·38, commenced to react to the complement fixation test. The vaginal mucus showed *T. equiperdum* in fair numbers. In view of previous failures to infect laboratory animals, a fresh attempt was made. Ten rabbits, three puppies, and thirty mice were inoculated with the material, but without success. The mare showed a few trypanosomes in the blood.

(3) *Observations on Dourine in Donkeys.*

Since the commencement of routine testing for dourine at Onderstepoort much difficulty has been experienced in interpreting the reactions given by mules and donkeys, but more particularly by the latter. The main difficulty is that sera of mules and donkeys sometimes give non-specific reactions. These have been explained in the notes on the complement fixation test. This non-specific factor was encountered by Meyer (1909-10) in his complement fixation tests on mules and donkeys for glanders; and he recommended inactivation at 62°C for half an hour instead of the usual 58° to 60°C. All sera have been inactivated at 62°C for half an hour as a routine procedure, but this has not completely eliminated non-specific reactions, nor has the same temperature for periods of forty-five minutes to an hour. Temperatures above 62°C are liable to coagulate the sera and destroy the antibodies responsible for the reaction.

In samples received from the field for testing, most trouble has been experienced with sera that have had to travel some distance. Those from South West Africa have given a high percentage of non-specific reactions. In the block tests in Bredasdorp and Swellendam districts a number of mule and donkey sera have been included, but so far no difficulty has been

experienced in interpreting the results of tests made with them. In the case of non-specific reactors one can, by using a double dose of complement, usually eliminate the non-specific factor, but this still leaves a doubt as to whether a weak positive reaction is being dealt with.

Some donkeys have reacted positively at one test, then at subsequent tests given partially positive or even negative reactions. There have been donkeys that have done this over a period of some months, giving alternating positive, partially positive, and negative reactions, finally becoming negative permanently. One is at a loss to interpret these reactions and it is difficult to say whether the animals were once infected and have since lost their infection.

There are two donkeys at Onderstepoort that have been infected with *T. brucei* for some years. These animals always give positive reactions and there is no suggestion of non-specific ones. Animals infected with *T. brucei* always react positively with an antigen made from *T. equiperdum*, the reaction being a group one shared by a number of species of trypanosomes. The tests with the donkeys suggest that unless the reaction is constantly positive the animal is not really infected, and it is possible that uninfected donkeys may give an occasional definite or partially positive reaction.

Further experiments on the sera of mules and donkeys are being carried out to eliminate non-specific reactions. The importance from the eradication campaign point of view is very great, as inaccurate results are liable to reduce the confidence of the farmer in the value of the tests.

Treatment and Artificial Transmission Experiments.

The utilization of treatment in the control of dourine, even if successful, is unlikely to be a practical procedure. Little attention has thus been paid to it. A few preliminary trials have been performed and more are at present being carried out. For the purpose of comparison, treatment has been tried in fully susceptible horses infected with a strain of *T. equiperdum* imported from Europe and used in the preparation of antigen for the complement fixation tests, and in natural cases of dourine inoculated with this strain, to see if any immunity to it would be shown.

The horse infected with the imported strain was successfully treated with a combined naganol-arsenic therapy, but this treatment was unsuccessful in natural cases. These latter showed improvement in condition following treatment, but the infection was not eliminated as the animals continued to react to the complement fixation test. If a horse affected with dourine acquired naturally be infected with the imported strain, it develops an infection of the blood stream and shows symptoms indistinguishable from those seen in artificial *T. brucei* infection of a susceptible horse. The symptoms produced are an intermittent fever and a rapid loss in condition. There is a drop in the red cell count of up to 60 per cent., and numerous

trypanosomes are seen in blood smears. The occurrence of such a superimposed infection is possibly due to variations in the parasite resulting from its adaptation to laboratory animals. This superimposition on natural cases should be attempted with the South African strain of *T. equiperdum* if it should be isolated. Treatment of the superimposed infection produced a permanent disappearance of the parasites from the blood, a marked improvement in the animal's condition, elimination of abnormal temperatures, and a return to normal of the blood count. The complement fixation test, however, remained positive. The introduced trypanosome had therefore been eliminated, but the previous natural infection remained unaffected.

Horses are very susceptible to the imported strain of *T. equiperdum.* Even the infusion of infected blood into the apparently undamaged vagina produces the form of the disease with a marked infection of the blood, the parasites appearing in as short a time as five days, accompanied by a rise in temperature.

The possibility of producing positive reactions to the complement fixation test by inoculating horses with positive serum or whole blood free from trypanosomes has been explored, but so far positive results have not been obtained. The experiments were suggested by the occurrence of positive reactions in foals after drinking the colostral milk of infected mares.

The Campaign in the Field from the Laboratory Point of View.

It is not proposed to review the various attempts made to carry out systematic tests for dourine in South Africa, but simply to mention some points of interest about tests done at Onderstepoort.

From 1923 to 1931 only a small number of tests were done and no systematic testing was undertaken. Subsequently, limited eradication schemes were undertaken in the North West Cape areas and large numbers of tests had to be carried out.

Much difficulty was encountered in obtaining satisfactory sera from the field for tests. At first sera were decanted from the clots and sent with 1 per cent. carbolic acid solution as preservative. At present the method used is to collect the samples in 10 c.c. bottles to which boracic acid solution has been added, so that when the bottles are filled with blood the final concentration of the preservative is 1 per cent. This method has proved satisfactory even in the summer months. Coagulation is not interfered with, and perfect sera are obtained which only commence to hæmolyze after about two weeks at summer temperature.

Since the commencement of the block tests in the Western Province last year, about eight hundred samples a week have been tested. The number of samples to be tested has been known approximately so that arrangements for dealing with them could be made. Improvements have

been made in the preservation of the antigen which is now stored at – 12°C and retains its full potency for at least a month. Previously when kept at about 10°C in an ordinary refrigerator it lost potency after about two weeks.

All positive sera are retested, the duplicate being used for the retest. In case of doubt both samples are retested.

Conclusions.

1. The results of the complement fixation test in the diagnosis of dourine in horses are very consistent as judged by continuous observation on a troop of infected animals.

2. In the case of donkeys and mules further observations are necessary in order to study non-specific and irregular reactions.

3. Foals of infected mares are negative to the test at birth, but they may give positive reactions shortly afterwards. These reactions are usually lost after a few months. The question whether actual infection of the foals occurs requires further investigation.

4. The sterilization of the blood of horses infected with the antigen strain of *T. equiperdum* has been accomplished.

5. Judging by the complement fixation test, no transference of antibodies from an infected to a normal horse has been noted.

6. Superimposition of the antigen strain of *T. equiperdum* on natural cases of the disease has been demonstrated.

7. In natural cases of dourine, therapy has not influenced the reactions of the animals to the complement fixation test.

8. Mares have been infected with dourine by infusion of blood containing trypanosomes into the undamaged vagina.

9. A number of mares have shown a positive reaction to the complement fixation test for four years or longer without any clinical manifestations of the disease. They were able to infect stallions and so become a factor in the spread of the disease.

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TABLE I.

Foal.	Date of Birth.	Dates of Tests and Results.
21472	25.1.37	17.2.37 ††
		4.3.37 ††
		31.3.37 ††
		3.5.37 ††
		22.6.37 –
		22.7.37 –
		8.9.37 –
		16.10.37 –
		died
21479	2.2.37	17.2.37 ††
		4.3.37 ††
		31.3.37 ††
		3.5.37 ††
		22.6.37 –
		22.7.37 –
		8.9.37 –
		10.12.37 –
		Became positive again when inoculated with infected vaginal washings.
21507	26.2.37	31.3.37 ††
		died 26.4.37
21561	26.7.37	26.7.37 –
		27.7.37 –
		died
		Never sucked
21556	16.7.37	17.7.37 –
		18.7.37 †
		19.7.37 †
		20.7.37 –
		died 28.7.37
		(weak foal)
21555	15.7.37	15.7.37 –
		17.7.37 ††
		22.7.37 ††
		23.8.37 ††
		16.10.37 ††
		10.12.37 –
		11.2.38 –
		23.3.38 –
		23.3.38 –
		Still negative to date.

TABLE I — (continued).

Foal.	Date of Birth.	Dates of Tests and Results.
21578	6.8.37	6.8.37 – 11.8.37 – 8.9.37 – 16.10.37 – 10.12.37 – 11.2.38 – 23.3.38 – Still negative to date. Foal of low antibody mare.
21622	22.10.37	22.10.37 – Died at birth.
21632	8.11.37	8.11.37 † 8 hours after birth. 10.12.37 †† 11.2.38 – died 23.3.38
21610	28.9.37	10.12.37 †† 11.2.38 † 23.3.38 – Still negative to date.
Foal A	7.8.37	Died at birth (dystokia) –
Foal B	9.5.37	Died at birth –
21616	6.10.37	7.10.37 – died 7.10.37
21626	23.10.37	24.10.37 †† 10.12.37 †† 11.2.38 – 23.3.38 – Still negative to date.
21621	Came to Onderstepoort when 7 months old	22.10.37 †† 10.12.37 †† 11.2.38 †† 22.3.38 †† died.

†† = strongly positive. † = weakly positive. – = negative.

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The Health of Wild Animals.

By Lieut.-Colonel J. STEVENSON-HAMILTON, Kruger National Park.

(Read at Annual Meeting of S.A.V.M.A., 1938.)

I have been asked to say a few words in connection with wild life ecology as observed in the Kruger National Park. I wish to refer particularly to the peaks and depressions that appear to have affected different species of animals during the time I have been in charge of the area, because it is possible that these effects were caused by diseases.

In addressing an audience of this kind, I am naturally diffident about putting forward my views and therefore shall speak only of certain observations; the elucidation of their causes must be left to those better qualified than myself.

Before proceeding further, I wish to express my thanks to Dr. A. D. Thomas for his kindness in supplying me with copies of his reports on the external and internal parasites affecting wild animals in the Transvaal Low Veld. I found his remarks exceedingly illuminating and interesting; indeed, his list of the living creatures which inhabit the interiors of even the most healthy antelopes was to me so terrifying that I have since conceived a distaste for game meat of all kinds. Were Dr. Thomas's reports more widely known, the South African public's taste for buck-meat and biltong would be considerably lessened.

I will confine myself to giving you a short digest of what I have noticed since I first took over control of the Sabi Game Reserve in 1902. Since that time I have lived more or less continually within the area, and during the first 25 years of the period, most of my time was free to observe and study wild life in the veld. The fluctuations in the prosperity of species in a wild state — a subject of long investigation in some of the American national parks — appear to occur here as well, although the relatively short period of my observations (36 years) does not perhaps justify definite conclusions. In America it was found that peaks of abundance of individual species occurred at regular intervals of years, with intervening periods of decreasing and increasing numbers. The same seems to apply to our animals. Certain species have become very numerous, then declined suddenly, and at a later period revived again. Certain factors, not yet fully studied, the study of which must be left to trained scientific observers, cause the life of any given species to proceed, not in a straight line, but in a succession of upward and downward curves. A sharp upward curve is often followed directly by a steep downward one, and this again, after a nadir of long or short duration, by a slow recovery.

In 1902, when I was first introduced to the Sabi country, all species of wild animals were scarce compared with the numbers there now, and the proportions between species differed, in some cases considerably, from those obtaining at the present time. My observations were mainly confined to the country south of the Olifants River, since owing to the slow means of transport in those days, I could pay only occasional and fleeting visits to the country north of it. In 1902, although game generally was relatively scarce in the Reserve, the reedbuck, a definitely local animal, was numerous compared with other species within its particular habitat. At that time the country was ravaged by large numbers of wild dogs (*Lycaon pictus*) which roamed in packs of 50 and upwards, and preyed largely, perhaps chiefly, on impala and reedbuck. Aided no doubt by a considerable reduction of the wild dogs through the efforts of the game reserve staff, reedbuck greatly increased so that they existed in large numbers in every vlei and other suitable spot in the Reserve. It was almost impossible to camp near water without hearing their strident whistling right through the hours of darkness. This condition persisted until I went overseas in 1914.

I may perhaps here stress the very important and useful part played by carnivora, great and small, in checking and possibly even in preventing the spread of disease — the result of overcrowding or other causes — among the ungulates. Deprived of these natural checks, every species would quickly outrun its food supply, possibly with disastrous results. In wild nature no animal can long survive, once sickness, age or injury has limited its powers. A herbivorous animal deprived of its full powers of escape has small hope of long survival. A carnivorous creature having once lost his full power of pursuit and capture cannot exist for long. Nature is hard. In the wilds there are no doctors nor hospitals nor charitable institutions. Eat or die is, in fact, the rule, and if a wild animal cannot get its food for itself, it can expect no help from others. So we get the survival of the fittest, for no animal either carnivorous or herbivorous having once fallen below a certain standard of health, has any hope of survival. Fang and claw deal quickly with the latter, while a more lingering death from starvation is the fate of the sick lion, leopard or wild dog.

Were the natural checks eliminated, I feel sure that wild animal problems of health over any large area where artificial restriction by human beings was not resorted to would quickly form a matter for most serious consideration.

On my return a few years later I at once noticed the complete absence of reedbuck in their former haunts. Enquiries from the staff elicited different reasons. Some put the decrease down to wild dogs, others to native poachers. But I reflected that in spite of the staff having been reduced between 1914 and 1919 the killing of wild dogs had still proceeded; and I remembered further that up to 1902 both native poachers and wild dogs had been far more numerous than they ever became later, and yet the

position of reedbuck as compared with that of many other species was then very favourable indeed. In spite of a lesser toll having been exacted from them during the years 1914-19, wild dogs in 1920 could not be compared in numbers with those in 1902. Some natives whom I asked about it said the reedbuck had been "killed by ticks," and this gave me the idea that there might have been some cause, other than those suggested to me, of the almost complete disappearance of the reedbuck; especially as impala, which have always formed the staple prey of wild dogs, had actually increased considerably in numbers.

Between 1914 and 1919 there were several dry seasons, with one (1917) in which abnormal rain fell and much of the country was under water. Before 1914 there was annually a great deal of grass burning, in pursuance of the policy of hunting carnivorous animals, and this may have resulted in the overstocking of the damper ground of the vleis with reedbuck. It is only within the last four or five years that reedbuck south of the Olifants River have again become sufficiently numerous to be seen without having specially to be sought for.

The Olifants River forms a definite faunal barrier. North of it the reedbuck had no set-back, but increased from very small numbers in 1903, when I first visited the country, to quite large numbers today.

Steenbuck had had much the same history as reedbuck. They were exceedingly numerous in 1914. After 1920, for a good many years, they were hardly to be seen at all. During the last 10 years they have been increasing rapidly, and may now be nearing a peak.

Although the favourite country for sable antelopes was always the western portions of the old Sabi Reserve which were excised in 1923, there still remained considerable, though migratory, herds in the eastern portions between the Sabi and Olifants Rivers. In 1925-26 commenced a series of drought years which, with slight intermissions, continued until 1936. Early in 1926 I noticed a reduction among the sable, especially among the calves. After 1930 the decline became still more marked and I put it down at the time to the sable's having trekked to more suitable country. This, indeed, is a solution which cannot be ruled out, since both the areas lying outside the present Park on the west, and Portuguese country to the east are better sable countries than the Park south of the Olifants River. Sable are comparatively delicate feeders and would no doubt leave any country temporarily denuded of their favourite grasses. Still one cannot overlook the fact that there have been, within the last few years, several reports concerning sable being found dead in their more favourite grazing grounds to the west of the Park, and I think it quite possible that between 1925 and 1935 some parasitic complaint may have affected them.

In 1916 many wildebeest, waterbuck, duiker, and warthog, all species which had been increasing progressively since 1902, died, after a long

drought, in considerable numbers in the neighbourhood of waterholes. In 1922-3 wildebeest and waterbuck died during heavy rains apparently from some complaint affecting the legs. In October, 1923, during a period of heat and drought just before the rains, I examined a newly dead wildebeest bull. There had been nasal discharge, and the intestines were inflamed. About the same time I came on a newly dead waterbuck bull suffering from a gangrenous condition of the chest and softer parts of the body which gave out a most offensive smell. These, however, appeared to be isolated cases and I noticed no signs of sickness among the game in the vicinity. There was a rapid and uninterrupted general increase among blue wildebeest up to about 1932, when so high a peak was reached that much of their favourite grazing country became overstocked, and the pasture was so much destroyed by overgrazing combined with drought, that it has not yet recovered. Since that time there has been a visible decline in the numbers of wildebeest in the eastern areas of the Park though in the western ones they are as numerous as ever, and I am inclined to attribute this decline to migration due to overgrazing rather than to any pathological cause.

Waterbuck have a somewhat similar history. After several years they appear once more to be increasing in the eastern areas. I should, however, remark here that in early 1937 reports reached me of waterbuck — especially the younger animals — being found dead in the country west of the Park, " from the effect of ticks." In the absence of any scientific examination it is of course impossible to say what was the actual cause of death: but I have always noticed that grass ticks swarm in greatly increased numbers on to any wild animal which is in bad health or poor condition.

Impala have been on a steep upgrade for some years and have, in the absence of their chief enemy — the wild dogs — reached an unprecedented and possibly dangerous peak, so far as the health of the species is concerned.

Zebra have also climbed steadily, without any set-back, for a great many years past. They are, of course, essentially migratory animals.

The virtual disappearance of the wild dogs is as interesting as that of the reedbuck. I have mentioned their history up to 1914. Early in 1920 the ranger in charge of the Shingwedsi country reported the appearance of a virulent type of disease among domestic dogs, which he called " pulmonary distemper " and which he thought had come from Rhodesia. It had spread from domestic to wild dogs and he said most of the latter had died. In fact, even today wild dogs are scarce over all that country, though they have recently begun to show signs of revival. Up to 1926 wild dogs (*Lycaon*) still roamed the country south of the Olifants River in packs of up to 20 individuals. About that year a virulent and seemingly new disease (possibly in the light of present knowledge, rickettsiosis) appeared among domestic dogs south of the Olifants River. The disease was characterized by extreme wasting, but there were many variations, and the ultimate cause

of death was usually pneumonia, and I think about 90 per cent. of the animals attacked died, in spite of attention.

From about 1928 wild dogs began to be less and less in evidence, until between 1931 and 1935 none were seen in the Sabi area, although formerly, on account of the abundance of impala, this had been their favourite hunting ground. This absence had nothing to do with shooting, as after 1931 no more were killed by the staff. In 1935 a small pack of eight, including three adults and five half-grown young ones, appeared for a week or two near the Sabi, but disappeared. Today I believe only one pack of perhaps a dozen exists south of the Olifants River in the Park, and is usually found near the Crocodile River. Unfortunately I have been unable to get specimens or smears from dead or sick wild dogs as, like other wild animals, they always crawl away to hidden places, or perhaps down holes, to die.

Hyenas were amazingly numerous in 1902, but between 1912 and 1930 something detrimental to them evidently occurred, and at one time they seemed to be almost extinct south of the Olifants River. During the last few years they have once more increased in numbers.

During the severe drought in the south-west corner of the Park at the end of 1935, a great many buffaloes died. I hardly think that local lack of grass was sufficient, by itself, to account for the mortality, since both pasture and water was obtainable within a reasonable distance. In any case, with the advent of rain and the springing-up of the young grass the mortality, which had continued for about two months, suddenly came to an end.

Similar variations in numbers have been noticed among the smaller mammals. On several occasions during the past 30 years abnormal but temporary increases among rodents have been reported. There was a notable peak about 1908 in a portion of the Reserve, and native crops suffered greatly from the attacks of rodents, and the kraals and storehouses were invaded. During the summer of 1936-7 the neighbourhood of our Lower Sabi camp was infested with enormous numbers of rodents, chiefly if not entirely, the multi-mammate mouse (*Mastomys*). They came into the camp in thousands, ate their way through the door frames, and did a considerable amount of damage to camp furniture and stores locked up in the huts. Various steps were taken against a possible further visitation in the following summer (1937-8), but the rats seemed to have disappeared, and the camp remained quite free from them. 1937-8 was rather a wet summer, and the following an unusually dry one. I do not know whether the cause of the great infestation and subsequent disappearance was a local migration due to climatic changes or not. No reports of any unusual number of dead rodents during 1938 were received.

Within the past few years there seems to have been a noticeable increase of the grey-footed squirrel (*Paraxerus cepapi*). This species has

a vast number of enemies, both on the ground and in the air, and there has been no noticeable decrease among any of them.

It is unfortunate that we can only guess at the reasons for many of these fluctuations among species. For the nature of the country, the habit of wild animals of concealing themselves when about to die, and the ubiquitousness of scavengers makes it a matter of extreme difficulty to discover remains of any animal, sufficient for investigation. A few scattered bones are often all that are left within 12 hours of death; and it is therefore sometimes hard to know whether the animal's death was due to disease or to violence.

Before closing these remarks I should like to say a few words regarding the tsetse fly and its remarkable, and in some cases permanent, disappearance from many of its old haunts, after the rinderpest epidemic at the end of the last century. Rinderpest is supposed to have first broken out in Eritrea, on the Red Sea coast, among cattle imported from Asia Minor, where the disease was endemic. It appeared in Abyssinia in 1890 and travelled slowly southwards until it reached the Zambesi, whence it progressed at the rate of the ox waggons which then formed the main transport of the country. It pursued its course in a rather erratic manner. Here a range of mountains diverted it, there it swept down one bank of a river and left the other untouched. Sometimes a comparatively large area was left unvisited, while the surrounding country was ravaged. The Barotse Valley in N. Rhodesia, which held many thousands of cattle at that time, was unaffected though the areas all round were nearly denuded of stock. It swept down the west side of Lake Nyassa leaving the country between the eastern shore and the sea untouched, and it stretched out its tentacles across Northern Rhodesia to Angola. Having crossed the Zambesi at many points it pursued its southerly trend into the Transvaal, via the main waggon roads from Rhodesia, ravaged the High Veld, and was apparently introduced into the low country bordering the Portuguese frontier by the oxen of winter trekkers and hunters in 1896. It raged during the middle and latter part of that year in what is now the Kruger National Park, but had expended its fury by the end of that year.

Prior to the rinderpest this area had contained a number of well-known belts of tsetse fly, understood to have been *Glossina morsitans.* In addition to several zones north of the Sabi River, practically all the country south of it, for some 40 miles west of the Lebombo Hills, was more or less fly-ridden, although I have been told by experts that it was probably only of seasonable occurrence over a portion of the area, and remained permanent in certain portions of the south-east and south which were, no doubt, fly sanctuaries throughout the year. The big game within the southern fly belts were then impala, wildebeest, zebra, kudu, and buffalo.

It is a popular fallacy that rinderpest was fatal to all kinds of game. On the contrary it attacked only or mainly the species of *Bovidæ* that possess a large naked rhinarium, and thus more nearly approximate domestic cattle:

buffalo, eland, kudu, and bushbuck and, to a lesser extent, duiker and reedbuck. Waterbuck were only partially affected. All horned ruminants which possess a hairy rhinarium, such as sable, roan, tsessebe, blue wildebeest, and impala, and are more nearly akin to the goats than to the cattle, seem to have been little, if at all, affected by the disease. Curiously enough, both warthog and bushpig died in large numbers. Not more than 15 buffaloes are supposed to have remained in the Sabi Bush. Of course, zebras were completely unaffected. During the latter part of 1896 those types of horned ruminants susceptible to rinderpest were dying fast from the disease, and were still doing so when the winter graziers and hunters left the Low Veld as usual about September, 1896. Fly was still in evidence, though some say not so numerous as usual. A hunter who knew the Low Veld well in those days once wrote to me as follows:– " Previous to August, 1896, the present Sabi Reserve was infested with fly, and in all that country buffalo were to be found only between the Sabi and Crocodile Rivers, and about the junction of the Sand River with the Sabi; though not so numerous as in previous years. One night in June, 1896, I was sitting up for lions just below the Sabi Bridge, when a kudu bull came down to drink. As it did not come up again from the water I went down at dawn to the river and found it lying dead where it had been drinking — dead from rinderpest. Again, in December, 1896, I was down at the Sabi River, and not a single tsetse fly was to be found, nor has one been seen since, to my knowledge. At the same time the fly disappeared from the other fly belts; all country that the rinderpest had ravaged."

When early in the following healthy season the hunters once more arrived in the Low Veld it was quickly noticed that the fly had entirely disappeared. It seems in fact to have done so during the short space of the last four months of 1896, both from the Eastern Transvaal and from all the adjacent territory of Portuguese East Africa, lying east of the Lebombo Hills. No tsetse fly has been seen, or heard of, now for more than 40 years, in any of these areas, and, as is of course well known, horses and cattle have remained in good health in all parts of the Kruger National Park and in the neighbouring Portuguese country, which formerly were tsetse fly zones.

Now how far was the disappearance of the tsetse fly due to a reduction of its food supply in the region in question? In the absence of any other destructive cause, the fact that even a few buffaloes, kudus, and bushbucks survived (supposing theirs to have been the only blood essential to the existence of the fly) must surely have permitted at least a few tsetse fly to survive, seeing that these animals preferred always the heart of the fly areas. The fly should then have again increased with the increasing herds. The buffaloes especially had been for many years so assiduously hunted and persecuted that they had become accustomed to confine themselves to the large area of thorn bush, which coincided with the fly belt, between the

Sabi and Crocodile Rivers. Seeing that the various tsetse-ridden zones in the Sabi country ran more or less parallel, and at no great distances apart, it appears rather improbable that all the animals which outlived rinderpest would have prolonged their absence from one or other of the fly belts for so long a time that all the flies died of starvation. But the fact was that the tsetse-infected country between the Sabi and Crocodile Rivers was fairly well stocked with wildebeest and zebra and was also the stronghold of the impala antelopes, animals which were practically untouched by rinderpest. Thus it was certainly not lack of blood food that caused the fly to vanish. All the above species, especially impala, were numerous in 1897 after both fly and rinderpest had disappeared.

The same phenomenon seems to have occurred in other places, for instance along the Chobe River, whence fly disappeared under much the same circumstances, during the same period. Two years after the rinderpest I had occasion to travel up the Chobe and Kwando Rivers in Northern Rhodesia for some four months. I was warned at Kazungula, before starting, that tsetse existed along much of my route and that I should leave my dog behind. However, as I did not intend to return by that route I decided to take the animal with me, and he suffered no harm from tsetse. In fact I never heard of nor saw one throughout my journey. All the buffalo and kudu had died of rinderpest and their bones lay everywhere. There were, however, large numbers of blue wildebeest, tsessebe, roan antelope, zebra and lechwe all along the route. Many years later I learned that fly had again appeared on the lower Chobe and enquiries made through the District Officers traced its origin to certain islands in the swamps to which the rinderpest had not penetrated and whence, in the course of years, the fly was carried back to the mainland by wild animals. I cannot say to what species this tsetse fly belonged, although I believe it was *morsitans*.

Now the important question arises. If the fly did not die of starvation during and after the rinderpest, what then caused its sudden disappearence from those areas where rinderpest had been most virulent?

Some experiments carried out in East Africa about 1911 on feeding tsetse flies on cattle suffering from rinderpest showed negative results. Yet it seems almost certain that some, as yet unknown, factor closely connected with the epidemic was responsible for the total and final disappearance of *Glossina morsitans* from thousands of miles of game country in the Eastern Transvaal and neighbouring Portuguese East Africa, between October, 1896, and May, 1897.

It seems a pity that so little attention should have been given to finding out the true cause of that very remarkable, sudden, and complete disappearance. Research work in that direction might perhaps have far-reaching results, could the basic reason be discovered, and the mind thus delivered of the probable fallacy that the fly perished from want of food. I hope

you will not think that I am suggesting artificially reintroducing rinderpest to this country for this purpose; but the disease still exists in endemic form in portions of Africa and it seems to me that some investigation along the line indicated might yield interesting results. I trust I, a layman, may be forgiven for thus bringing forward what has been in my mind for many years. I would not have ventured had not Dr. de Kock encouraged me to do so. There may possibly be nothing in my impressions; but I think research on these lines by trained biologists might be worth while.

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The Sensitivity of the Picrate Paper Test (Guignard Test) for Hydrocyanic Acid.

By DOUW STEYN, B.Sc., Dr. Med. Vet., D.V.Sc., Onderstepoort.

(Submitted for publication on 28.6.37)

Introduction.

As the picrate paper test for hydrocyanic acid is a very sensitive, expeditious, and simple one, it was thought advisable to determine its limits. The table published here might in certain cases be used in the quantitative determination of free hydrocyanic acid in materials which are known not to contain reducing substances such as aldehydes, acetone, sulphuretted hydrogen, sulphurous acid, etc., which also cause a reddish discolouration of the picrate paper. In the case of plants this test yields very satisfactory results. When specimens of animal organs are, however, analysed for the presence of hydrocyanic acid the only two reliable tests are the Prussian Blue and the sulphocyanate (ferric sulphocyanate, which causes a reddish discolouration). The method generally followed in the author's laboratory is to submit all specimens, especially animal organs sent in for analyses for the presence of hydrocyanic acid, to the picrate paper test and, if this proves to be positive, the ferric sulphocyanate test is done.

Waller (1910) describes what he terms "a new method for the quantitative estimation of hydrocyanic acid in vegetable and animal tissues." The method employed by him is as follows:– The minced animal organs (or blood) are acidified with tartaric acid and are then distilled into a solution of sodium picrate, which shows reddish discolouration if hydrocyanic acid is present. The estimations are made according to the intensity of the red colour. As there are substances other than hydrocyanic acid which yield a reddish colour with alkaline picric acid solution, unreliable results may, and will, be achieved when hydrocyanic acid is estimated by Waller's method in human and animal organs, especially when these are in an advanced state of decomposition.

Experiments.

Method.

Potassium cyanide solutions varying in strength from 1 : 1,000 to 1 : 50,000,000 were prepared with distilled water. Of each of these solutions 5·0 c.c. were pipetted into a small test-tube and 0·3 c.c. of 10 per cent. aequeous hydrocholoric acid added. A strip of filter paper moistened with sodium picrate solution was immediately inserted in the test-tube and fixed

TABLE SHOWING THE SENSITIVITY OF PICRATE PAPER TEST FOR HYDROCYANIC ACID.

	Quantity and concentration of Potassium cyanide.	Potassium cyanide in mgm.	Hydrocyanic acid in mgm.	Result.
1.	5 c.c. of 1 : 1,000	5·0	2·077	Typical reddish-brown discolouration of picrate paper commenced within 5 seconds
2.	„ „ 1 : 1,500	3·33	1·385	do „ 10 „
3.	„ „ 1 : 2,000	2·50	1·0385	do „ 20—40 „
4.	„ „ 1 : 3,000	1·67	0·692	do „ 40—50 „
5.	„ „ 1 : 4,000	1·25	0·52	do „ 50—60 „
6.	„ „ 1 : 6,000	0·83	0·346	do „ 1 —1½ minutes
7.	„ „ 1 : 8,000	0·625	0·26	do „ 1½—2 „
8.	„ „ 1 : 10,000	0·50	0·207	do „ 2 —2½ „
9.	„ „ 1 : 15,000	0·33	0·138	do „ 2½—3 „
10.	„ „ 1 : 20,000	0·25	0·104	do „ 3 —3½ „
11.	„ „ 1 : 30,000	0·167	0·069	do „ 3½—4 „
12.	„ „ 1 : 50,000	0·10	0·0415	do „ 4½—5 „
13.	„ „ 1 : 80,000	0·0625	0·026	do „ 5 —6 „
14.	„ „ 1 : 100,000	0·05	0·021	do „ 6 —8 „
15.	„ „ 1 : 200,000	0·025	0·0104	do „ 9 —10 „
16.	„ „ 1 : 400,000	0·0125	0·0052	Slight reddish-brown discolouration set in within 12—14 minutes and was very distinct after 15 hours
17.	„ „ 1 : 800,000	0·00625	0·0026	do 15—20 „ distinct after 16 „
18.	„ „ 1 : 1,000,000	0·005	0·00207	do 20—30 „ do 16 „
19.	„ „ 1 : 1,500,000	0·0033	0·00138	do 60—90 „ do 16 „
20.	„ „ 1 : 2,000,000	0·0025	0·00104	do 110—120 „ fairly distinct after 16 „
21.	„ „ 1 : 3,000,000	0·00167	0·0007	do ±140 „ do 16 „
22.	„ „ 1 : 4,000,000	0·00125	0·00052	do ±150 „ do 16 „
23.	„ „ 1 : 5,000,000	0·001	0·000415	Negative after 16 „
24.	„ „ 1 : 6,000,000	0·00083	0·00035	do
25.	„ „ 1 : 8,000,000	0·000625	0·00026	do
26.	„ „ 1 : 10,000,000	0·0005	0·000207	do
27.	„ „ 1 : 15,000,000	0·00033	0·00014	do
28.	„ „ 1 : 20,000,000	0·00025	0·000103	do
29.	„ „ 1 : 30,000,000	0·000166	0·00007	do
30.	„ „ 1 : 50,000,000	0·0001	0·000041	do
31.	Eight controls	——	——	do

by means of a rubber stopper. The tubes were then placed in a water-bath kept at a temperature of 55–60°C. After one-and-a-half hours in the water-bath at 55–60°C the tubes were removed and placed in an incubator at 37–38°C for a further sixteen hours. The sodium picrate solution was prepared by dissolving 5·0 gm. of sodium carbonate and 0·5 gm. of picric acid in 100 c.c. of distilled water.

This test was repeated six times with identical results.

Discussion.

From the table on the preceding page it is evident that the limit of the sensitivity of the picrate paper test lies at about 0·00125 mgm. potassium cyanide and 0·00052 mgm. hydrocyanic acid, that is, 5·0 c.c. of a 1 : 4,000,000 dilution of potassium cyanide.

It is obvious that the rate of reddish-brown discolouration of the picrate paper depends upon (1) the amount of hydrocyanic acid present. The greater the quantity of hydrocyanic acid present the sooner discolouration will set in and the more intense it will be. (2) Whether the hydrocyanic acid is in acid or alkaline solution. In acid medium liberation of hydrocyanic glucosides will take place at a quick rate, whilst no or very small quantities will be liberated from alkaline solutions. (3) The form in which the hydrocyanic acid is present. If hydrocyanic acid is present as cyanogenetic glucosides, an acid medium or specific enzymes will be required to liberate it. (4) The temperature at which the test is conducted and the length of the period of incubation.

In the tests conducted by the author it was found that the temperature at which the acidified potassium cyanide solutions were incubated played an important rôle in determining the length of the period which elapsed from the time the picrate papers were inserted into the tubes until these papers showed the reddish-brown discolouration.

The tests were made in duplicate, the one set of test-tubes being kept in a water-bath at 55–60°C for one and a half hours and then transferred to an incubator (37–38°C) for a further sixteen hours, whilst the second set of tubes was placed in a water-bath at 30°C for one-and-a-half hours and then left in the water-bath (15–20°C) in the laboratory for a further sixteen hours. From the results of these comparative tests it was evident that with the same dilutions of potassium cyanide discolouration of the picrate paper set in much sooner and was more intense at the higher temperatures. It was found that 5 c.c. of a 1 : 4,000,000 potassium cyanide still gave positive results after sixteen hours at the higher temperatures, whilst the same dilution yielded negative results at the lower temperatures. It is therefore advisable to incubate the tubes containing the materials to be tested at 37–40°C for at least sixteen hours before the result can be regarded as negative, because the test is much more sensitive at high than at low temperatures.

The increased sensitivity of the picrate paper test at high temperatures is due to the hydrocyanic acid being more quickly expelled from the material tested and also because the acceleration of the chemical reaction between the sodium picrate and the hydrocyanic acid results in the formation of sodium isopurpurate, which colours the strip of filter-paper reddish-brown.

Summary and Conclusions.

(1) The limit of the picrate paper test lies at about 0·00125 mgm. potassium cyanide (=0·00052 mgm. hydrocyanic acid). The test is more sensitive at high than at low temperatures.

(2) The picrate paper test yields very satisfactory results in the case of plants. In the estimation of hydrocyanic acid in animal and human organs and other materials which are likely to contain reducing substances, this test will, however, yield unreliable results. In such cases the most reliable and most sensitive test for hydrocyanic acid is the ferric sulphocyanate. According to Abderhalden (1923) and Autenrieth (1928) the limit of the sulphocyanate test for hydrocyanic acid is 1 : 4,000,000.

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X(2): 69 – 71.
1939.

Tattooing as a Means of Marking Stock.

By M. ZSCHOKKE, Dr. Med. Vet., Government Veterinary Officer, Mariental.

In South West Africa most of the karakul sheep are marked by means of ear-tags, which indicate ownership, identification-number, classification, etc. Although ear-tags are used all over the world, they have proved in many instances to be unreliable, especially for animals grazing on bushveld, because they are easily torn out and lost or in the case of valuable stud-stock they might be removed and substituted or interchanged without difficulty.

These disadvantages of the ear-tag system forced breeders in this country to search for a better and safer way of marking. As branding of sheep was impracticable, the idea of tattooing a convenient spot on the body was considered. Ears had to be excluded, because they are invariably black, in many instances very small, and may be cut off or disfigured.

After years of fruitless experiments with various aniline dyes, ranging from a bright yellow to red, kindly placed at our disposal by the Bayer firm, and after many experiments with specially constructed, complicated tattooing forceps made by the well-known firm of Hauptner, Berlin, the writer evolved, in November, 1936, a technique so simple that one wonders that its discovery was not made sooner.

Tattooing means to deposit in the corium a suitable dye, which will remain there, and will, after the wounds inflicted by the tattoo-needles have healed, be visible through the epidermis. If the dye is deposited into the subcutis, *i.e.* too deep, it will be absorbed by the body and in a very short time the tattoo pattern will disappear.

Owing to the black skin of the karakul, tattooing had not been considered in the past, yet every karakul sheep shows a few places on the body which are not pigmented, viz., a small spot behind the elbow, the groins, the inner side of the thigh, and the skin underneath the fat-tail. The tail is by far the most suitable site.

It occurred to the writer that the skin underneath the tail need only be lifted and drawn up into a fold to present an ideal field for an ordinary tattooing forceps as used for pigs or rabbits. (See fig. 1 and 2.)

Obviously the tattooing needles will pierce through both layers of the skin-fold and produce one correct and one inverted pattern (fig. 3).

In rubbing in the dye care should be taken not to stain the inverted mark. This can be avoided by using a soft leather shield. This oblong

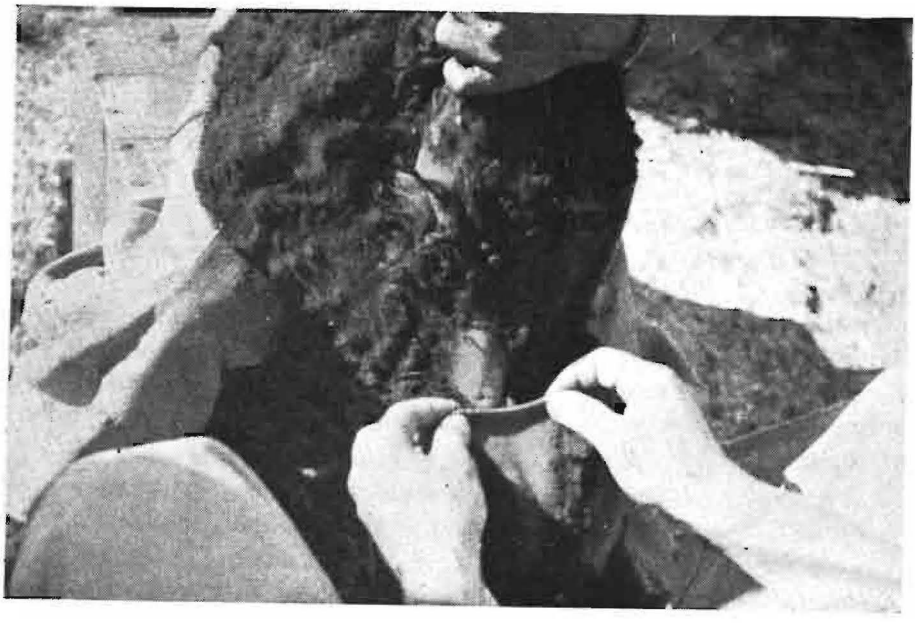

Fig. 1.

Fig. 2.

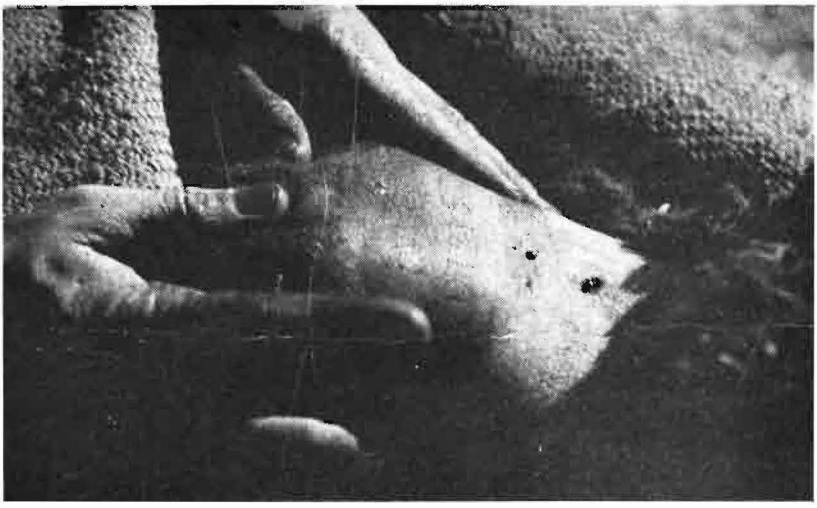

Fig. 3.

piece of leather should have a narrow opening in its centre slightly larger than the number to be tattooed.

By applying this shield with the opening over the correct pattern and rubbing in the dye through the opening, the inverted number is masked.

Tattooing has now been carried out on various karakul stud farms for more than two years. The results have been so satisfactory that there is a movement afoot to legalize this method of identifying karakul sheep.

Since the lack of a suitable technique was apparently the reason for this procedure of marking stock not being more extensively applied in the past, the experience gained here might also prove useful for stud cattle. This would obviate disfigurement of the skin by an ugly brand and provide an absolutely reliable method of identification.

(The photos have been placed at my disposal by courtesy of Miss Lizzie Voigts, Voigtsgrund.)

An Early Reference to Horse Sickness

(Cape of Good Hope Government Gazette; Friday, September 17, 1830. No. 1288.)

The following is published for general information.

Colonial Office, 10th September, 1830.

By Command of His Excellency the Governor,

(Signed) JOHN BELL,

Secretary to Government.

GRAAFF-REINET, 22nd July, 1830.

In the last article of the Instructions for District Surgeons, it is commanded that they shall, from time to time, report to Government touching Epizootic Diseases, and other medical business of their District; and as a large portion of the Horses of this District have died this year, of a Disease not generally known, I think it my duty to convey to you, for the information of His Excellency the Governor, and for the public good, the result of nine years' experience of the disease in question.

In the year 1819, the *Horse Sickness*, as it is usually called by way of eminence, on account of its great fatality, raged in this District to such a degree as to occasion much apprehension to those Persons who turned their attention to the Breeding of Horses; and on my arrival in this District, in the year 1821, I was requested by the then Landdrost, Captain STOCKENSTROM, to observe the Disease whenever it should appear; but no opportunity occurred till the year 1824, when the Disease again appeared, but not to the same extent as on the last occasion. I then took the earliest opportunity of observing the Disease while life continued, and of dissecting the body after death.

The first Symptoms of the Disease, as it has hitherto appeared in this District, are general torpor in the animal; he coughs and hangs his head; shows a great disinclination to motion; and refuses his food: there is generally some swelling and perspiration about the eyes, and occasionally of the whole head; the veins of the neck are distended, and the breathing is invariably quick and oppressed. These symptoms continue to increase till death ensues, which is seldom protracted to the fourth day, unless some remedy is used. Motion invariably accelerates the termination of the Disease, and persons occasionally ride the animal in its early stages, sometimes under the impression that a little exertion will prove useful, but generally from not suspecting that anything is wrong; for in the beginning of the Disease the mildness of the Symptoms does not lead to suspicion,

which is to be attributed to the insensible nature of the part affected.

If the animal is rode during the Sickness, or urged by driving or otherwise, to any degree of speed, he falls at once, literally suffocated by the quantity of frothy matter which fills his trachea and issues in abundance from his nostrils; — this was the case with the very first horse I dissected which had died of the Disease. The nature of the Disease was then so buried in obscurity — I mean in this District — and such contradictory reports prevailed, that I thought it advisable to get the assistance of two intelligent individuals at the dissection, and Messrs. KIFT and BAIN were kind enough to accompany me. The horse belonged to the last-mentioned gentleman, and fell under him while riding in the neighbourhood, without the slightest suspicion that the animal was diseased, and died almost immediately; he had not been stabled.

I shall give the result of the examination as it agrees with the results of numerous subsequent examinations, with a few exceptions only, which I shall note hereafter — and as being a document authenticated by the signatures of the gentlemen above-mentioned.

" The skin being removed, a more than natural determination of blood to the neck and shoulders was observed, but no other external indications of disease.

" On opening the Chest to examine the state of the lungs, the superficial veins were seen distended with blood; both lobes of the lungs were highly inflamed throughout more than half their bulk; the inflamed parts appeared of a dark colour, approaching to purple, and on cutting into their substance it was found to be filled with a yellowish frothy matter, similar to what came from the nose of the animal immediately before death. This froth exuded abundantly from the lungs when pressed — showing very satisfactorily the nature of the disease, and the origin of the discharge from the nose.

" The Trachea was entirely filled with the same frothy matter, which, by stopping respiration, appeared to be the immediate cause of death.

" The inflamed parts of the lungs were easily torn and punctured by the finger, while the more healthy parts were firm and tough.

" The other Viscera were carefully examined, and found healthy in every respect. As there was no symptom of disease in the head before death, it was not opened; and we felt fully satisfied that the disease in the lungs was quite sufficient to occasion death.

(Signed) THOS. PERRY, District Surgeon,

(Signed) B. G. KIFT.

A. G. BAIN.

" Graaff-Reinet, May 14, 1824."

From this period to January, 1829, the disease did not occur in this District; but then it returned with a fatality equal to that of 1819; and I did not lose the opportunity of obtaining further experience of the disease. I paid particular attention to many sick animals, and prescribed for some of them; I also dissected many when dead. The result of this year's experience did not militate in any degree against that obtained on the former occasions, — the symptoms during life were the same, and also the principal features of the post-mortem examinations. In consequence of the greater number of deaths, the field of observation at this period was necessarily extended; and I observed some appearances which escaped my observation, or which did not occur on the former occasions.

The head of the animal was sometimes swelled in an extraordinary manner, but always from congestion; I have repeatedly examined the brain in such cases, but have never discovered the least indication of disease therein; and the appearance and conduct of the animal during his illness have never betrayed the slightest symptom of aberration of intellect.

Another appearance on dissection not noticed in the above account, is a yellow gelatinous matter attached to the Pleura, or internal lining of the Chest, which always indicates inflammation. I have observed the same substance in the human subject, arising from the same cause. I must also observe that the frothy matter which flows from the nose of the horse at the time of death, is generally considered as a symptom peculiar to this particular disease in this animal. But long experience has taught me that the same appearance, but in a lesser degree, is observed in the human subject in the same disease; that is, an inflammation of the substance of the lungs. No people of whom I have ever read, or of whose diseases I have had experience, are so liable to pulmonary complaints as the Hottentots. It has been my lot to open many of them after death; and it is from the experience thus obtained that I assert their liability to the disease in question; — in them I have very often observed the same frothy matter in the substance of the inflamed lungs, and the same exudation from the nose after death. It is occasioned by the increased secretion of mucus in the inflamed lung, and rupture of the minute air cells. Accelerated motion necessarily increases the impetus of the blood through the lungs, and consequently hastens the termination of the disease.

An unfortunate belief prevails in this District, that no remedy is useful in this disease, and even by some persons it is believed that all curative measures are prejudicial; this belief must be attributed to the fatal nature of the disease. Some decry bleeding, and others purging — for both remedies have been tried, but without any regular plan of cure. It must occur to every one on reading the account of the appearances on dissection, that the only method of cure which can be adopted, with any prospect of success, is to lower the circulation by bleeding largely from the neck immediately the disease is discovered; I also purge plentifully with a ball,

composed of 4 drams of aloes, 1 dram of soap, 30 grains of calomel, and 10 drops of oil of carraway, and occasionally quicken its operation with half a pint or more of castor oil — keeping up the action of the intestines through the whole course of the disease.

It is an object of primary importance, to irritate the skin of the chest by some acrid application, as blistering flies, scalding water, or what will, perhaps, prove more efficacious than either, with the bruised leaves of that species of Ranunculus, called in this country " Brand Boschjes." It grows plentifully in moist places, and will raise a blister on the skin in ten minutes, the effect of which continues much longer than that of a blister raised by Spanish flies, while it costs nothing. I shall take the liberty of troubling you shortly with an account of the medicinal properties of some of the indigenous plants of this District, when I shall be more particular in describing the effects of the Ranunculus, which deserves to be generally known. It is also important to occasion a determination to the skin, which may be effected by antimonials and warm clothing, paying particular attention to the atmosphere of the stable. If the animal will eat, I give him warm mash of wheat bran, with nitre in it. The great difficulty is to discover the disease in its first stages, for it is only then that these remedies can be useful; they never succeed when a large portion of the lung is inflamed, and the progress of the disease is so rapid that I have known horses fall down before they were thought to be sick, as in the case of the animal whose dissection I have given above. It is no uncommon thing here to find a horse dead in the stable in the morning, which was supposed healthy the night before; but a careful observer will discover some of the symptoms I have enumerated: the first is generally a slight cough, and then no time is to be lost, for if left to itself, the disease will invariably terminate in death.

The disease in question is evidently an Epidemic, produced like other Epidemics, by some unknown peculiarity in the atmosphere, recurring at uncertain periods; I have no reason to believe it infectious, but the contrary. Its remote cause appears to be the sudden application of cold to the surface of the body; hence well groomed and well stabled horses are seldom attacked by it; this is an observation of such constant occurrence, that I think it can admit of no doubt; not that I mean to assert that no stabled horses will die of it; unfortunately I know the contrary too well, but even the imperfect stables of this District, which are often without doors, and always without windows, their place being supplied by unclosed air-holes, are a great protection to horses, as common experience proves. But as by far the greater proportion of the horses of this Colony is necessarily left to run at large in the field, it becomes an object of primary importance to inquire what description of country is least liable to the disease, and there it is that my inquiries and experience may benefit the public.

It is a constant practice in this District to send horses to the highest

lands about Midsummer, at which time the disease generally makes its appearance, and on any rumour of its approach. This practice is founded on experience and reason, for it is known that there are places on the highest mountains of the Sneeuwberg, where horses seldom or ever die of the "Sickness," which is to be accounted for by the well-known fact that those places, although cold, are not liable to sudden changes of climate; on the other hand, the low country, or as it is here called, the "Thornland," for the Mimosa does not grow on high mountains, is notorious for its unhealthiness for horses; for although warm, it is liable to sudden changes to cold, when the mist descends from the hills, bringing death to horses. Summer is here the rainy season; the wetest summers have been the most fatal; rain is always followed by a change of temperature, and the appearance of young grass, thence the popular belief that horses die of eating it; but the change of temperature will account for that much better; the same may be said of the belief that dew is injurious to them, for it is known that horses die when turned out of a warm stable to graze before the dew is off the grass, and it is also known that when the atmosphere becomes warm the dew is evaporated.

Garlic given with food is constantly used here as a preventative, but on what grounds I do not know, neither have I ever seen any good effects from it; but popular experience is always worthy of attention; I believe the only preventatives for this fatal malady, are good grooming and stabling, or probably warm clothing of some description or other, and where these things cannot be obtained, sending the horses to the highest mountains, where the disease is known not to reach.

The only plan of cure when the disease appears, is to be vigilant, and use the remedies above-mentioned, or similar ones, in its earliest stages.

Jl. S.A.V.M.A.,
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CORRESPONDENCE.

Veterinarians and Dehorning of Cattle.

P.O. Box 2655,
Johannesburg.

The Editor,
Journal S.A.V.M.A.,
Pretoria.

Sir, — Several articles appeared lately in the lay press advocating dehorning, whereas the official veterinary opinion appears to be against this operation. As a veterinarian I regard dehorning as a necessary form of disarmament in the cattle world, and we have to tackle it in a practical way. It is no use being blinded by sentiment. A colleague contended in a discus-

sion that it was unspeakably inhumane, and that the bellowing of oxen in agony could be heard many miles away when dehorning was being done. In my experience the same happens when oxen have to be vaccinated against anthrax. We shall have to regard anthrax vaccination as inhumane and abolish it if we regard the bellowing as proof of unbearable pain. In my opinion it is not so much pain as fear and rage that cause this bellowing.

From our professional point of view it is a matter of choosing the lesser evil. It is a fact that many horned animals inflict unspeakably cruel treatment on other animals off and on all the year round; so much so that it may even be necessary to destroy the victims. Taking everything into consideration dehorning, even of full-grown animals, must be regarded as a blessing. To show that the cruelty is not extreme, I wish to mention the following case. I dehorned a herd of 100 milk cows and heifers, the average daily yield of which used to be 170 gallons. On the day the dehorning was done the yield was still 170 gallons, on the second day it was 167 gallons, on the third day 169 gallons and thereafter again 170 gallons. None of the milk cows suffered any ill-effects, except that one bled rather excessively.

What we veterinarians must do is to see that this operation is carried out properly, and that the proper method is taught at colleges, and we shall avoid far more unnecessary cruelty than we shall by merely condemning the whole operation. The following are, in my opinion, essential points of procedure:

1. — The operation must be carried out in winter.

2. — It must be done on the standing animal to avoid soiling of the wound and sinus infection.

3. — The operator must be strong enough to amputate a horn with one quick cut.

4. — A bucket of clean water, and another containing an efficient disinfectant must be handy. The dehorner must be cleaned after each animal, and allowed to stand in the disinfectant until the next animal is ready.

5. — No after-treatment is given and the animals are turned loose for grazing. Excessive bleeding may be stopped by bandaging the wounds with cotton-wool dusted with iodoform.

6. — A saturated solution of iodoform in ether is poured into the wound when a sinus infection is apprehended.

Summary: Dehorning full-grown cattle without an anæsthetic is not excessively cruel, when properly carried out. It would benefit our cattle industry if this operation were more freely advocated.

Yours faithfully,

G. G. KIND.

ABSTRACT.

Investigations of Bacteria.

THE ETIOLOGY OF ANTHRAX, BASED ON THE ONTOGENY OF THE ANTHRAX BACILLUS.

By ROBERT KOCH.*

At the time (1877) when Koch wrote this article, doubt on the etiology of anthrax existed in the minds of many scientific workers and some went the length of denying any significance to the anthrax bacillus. Davaine had demonstrated tiny rods in the blood of infected animals and, with such blood, had transmitted the disease. The spread of the disease he attributed to those bacilli which, in the dry state, retained their vitality for a considerable time. But other workers claimed to have reproduced the infection with bacteria-free blood and stated that the disease stood in some relation to soil and temperature conditions, *e.g.*, it occurred more frequently in river valleys, in swampy regions and in the warmer months of the year.

Koch passaged anthrax from mouse to mouse for 20 generations and in smears from the spleen observed an abundance of rods, some in the state of division, but in no instance did he note motility or spore formation. The blood never contained many bacilli and sometimes it appeared to be devoid of them. (This explains the view of those who said that anthrax could be transmitted with bacteria-free blood.) Bacilli were frequent in the blood of the infected guinea-pig, but sparse in that of the rabbit.

If a slide-coverslip preparation of a drop of artificially infected ox serum be incubated in a moist chamber, enormous elongation of the bacilli is observed after 15 to 20 hours; threads, 100 or more times the length of the original rod, are seen. Many of these threads contain spores, so arranged in the thread that they produce the appearance of a string of pearls. This phenomenon occurs chiefly in the threads lying near the edge of the coverslip, where aerobic conditions are maximum. However, the edges of the coverslip were not sealed down, so to be quite certain that the elongation of the rods and the spore formation occurred in the anthrax bacillus and not in aerial contaminants Koch infected a drop of serum or aqueous humour on a coverglass and sealed this over the concavity of a hollow-ground slide. This, placed on the warmed stage of a microscope, was observed every 10 to 20 minutes. After 2 hours, the bacilli showed

* Untersuchungen über Bacterien.
V. Die Aetiologie der Milzbrand-Krankheit, begründet auf die Entwicklungsgeschichte des Bacillus Anthracis. Beiträge zur Biologie der Pflanzen (1877) 2. 277-310. Reproduced in the original German and as an English translation in "Medical Classics" (1938) vol. 2 No. 8. The Williams and Wilkins Co., Baltimore, U.S.A.

little more than a slight thickening; after 3 to 4 hours they had increased enormously, and still later had formed a tangled network. After 10 to 15 hours spore formation with disintegration of the threads had occurred.

To observe the germination of the spore into a bacillus, some spore-containing fluid was allowed to dry on a coverslip, and this was inverted over a droplet of aqueous humour on a slide. The slip was luted on with oil, and the preparation incubated at 35°C in a moist chamber. Germination commenced in 3 to 4 hours. It was often possible to observe, in one preparation, the formation of spores and the germination of bacilli from these.

Spleen, lymph glands, and blood of infected mice, rabbits, and guinea-pigs were dried in a shady airy place. Larger and smaller (pea to millet seed size) pieces were used and also thin layers on cover slips. Thereafter, mice were inoculated and a culture experiment conducted, at first daily and later every two days, with the material emulsified in aqueous humour. Thin layers were not viable after 12 to 30 hours, thicker pieces were infective for 2 to 3 weeks, and still bigger pieces for 4 to 5 weeks; but in no instance was this period exceeded. At 35°C, spore formation was copious after 20 hours, at 30°C, 30 hours were required, and at 18°C to 20°C 2½ to 3 days were necessary. Below 18°C it was exceptional, and under 12°C no growth at all was obtained. At a temperature over 40°C bacillary development was poor and appeared to cease at 45°C.

If blood or aqueous humour, infected with anthrax, is incubated in a well corked, but not sterile bottle, putrefaction occurs, the bacilli disappear in 24 hours, and the culture no longer sets up anthrax on inoculation into a mouse. The death of the organisms is due to the lack of oxygen. If the same material is left in a watch-glass, covered with a loosely-fitting glass plate, at 18°C, the anthrax bacilli multiply and sporulate in spite of putrefaction occurring.

Anthrax can be produced, after 11 weeks, with infected vitreous humour in which spore formation and putrefaction have occurred, and such material retains its infectivity after drying.

These experiments clarify the position. When a small quantity of infected blood is dried quickly spore formation cannot occur and the period of viability is short, at most 5 weeks. When a larger amount of material is allowed to dry more slowly at a moderately warm temperature, spore formation can occur and anthrax infection may be set up with it, years later (4 years in Koch's experiments, but this was not the end point).

Workers claimed that the disease produced with anthrax-infected material was the same as septicæmia.* To rule out the possibility, Koch

* "Septicæmia" appears to be the name given by workers of the time of Koch and Pasteur to a disease caused by injecting decomposed material into an animal. Probably many different kinds of microbes were involved, but possibly the most common were those of the gas-gangrene type.

injected decomposed but anthrax-free blood or aqueous humour into mice; of twelve animals treated in this way, two only died, and although their spleens were enlarged at post-mortem examination, no bacilli were observed in them. No success attended attempts to produce anthrax by feeding mice and rabbits with infected material.

The maternal blood of anthrax-infected pregnant mice and guinea-pigs set up the disease on subinoculation, whereas the foetal blood failed to do so.

Nine mice were infected at the same time and one was killed after 2, 4, 6, 8, 10, 12, 14 and 16 hours respectively, and the spleen examined immediately. The bacilli were first seen in the animal killed at the 14th hour, in greater numbers in that of the 16th hour animal, and in enormous numbers in the last mouse, which died after 17 hours.

Although rabbits, guinea-pigs, two dogs, one partridge and one sparrow were repeatedly inoculated with fresh material, anthrax was not produced in any. (This statement is difficult to reconcile with the experiments on the bacillary content of rabbit's blood and on the virulence of maternal and foetal guinea-pig blood: perhaps Koch meant that he was unable to cause death in these animals). The frog was quite resistant even when large pieces of infected mouse spleen were inserted under the skin.

Koch admits that all his research was carried out on small rodents and although he feels that ruminants would behave in a similar manner yet he realizes that experiments on the larger animals would not necessarily have the same results. For example, feeding experiments on ruminants might give different results, in view of the difference in their digestive systems.

In the prevention of anthrax Koch says that the most certain way would be the destruction of all substances that contain *B. anthracis*. As the sterilization of carcases by chemicals, boiling heat or even burning is impossible, he recommends burial at a depth of 8 to 10 metres and the fencing of the grave. At this depth, the temperature is reduced to below 15°C and oxygen is excluded; thus spore formation is prevented.

Koch observes that sheep appear to be the true hosts of anthrax because the disease prevails amongst them, off and on, throughout the entire year; if sheep be removed from a farm, the disease is very seldom seen in the cattle.

J.H.M.

REVIEWS.

The third and latest edition of *Dollar's Veterintry Surgery*[1] is an improvement on the previous issue in certain respects. The same arrangement as in the second edition has been followed, but a number of illustrations have been omitted and the text has been amplified slightly.

Although this book is an invaluable aid to the practitioner and student, and probably is the best book on the subject in English, yet it cannot be described as a thoroughly comprehensive treatise on veterinary surgery.

As might be expected when reviewing a book dealing with such a wide field one cannot agree with all the author's recommendations and statements. For instance one is surprised to find that chloral hydrate is considered to be a dangerous narcotic, to be used mainly on subjects "about to be destroyed"; similarly blood transfusion is summarily disposed of as being too dangerous for practical application.

The methods of anæsthesia for dogs are well described, except that mention of barbiturates like Pernocton has been omitted.

Several improvements have been made and various methods of treatment brought up to date. Among the most important of these must be mentioned a well illustrated description of the treatment of fractures in the dog and cat by means of cellona casts, and a chapter on autoplastia.

H.P.S.

* * *

The fourth edition of Hutyra and Marek's *Special Pathology and Therapeutics of the Diseases of Domestic Animals*[2] comprises three volumes. Volume I deals with the specific diseases, and volumes II and III with the diseases of the various systems.

In this edition the improvement of the arrangement of material, considerable simplification, elimination of much repetition, and numerous additions are indicative of the large amount of painstaking work that has been expended by the various people concerned in the translation. Particularly is Dr. Russel Greig to be congratulated on his editing. This work now definitely can be regarded as the standard book of reference on veterinary diseases.

It would add considerably to the value of the next edition if the section dealing with Protozoology were to be more authoritative and more up-to-date. Statements such as *T. brucei* infection may destroy entire herds of cattle, the lack of reference to the importance of *T. congolense* infection in bovines,

[1] *Dollar's Veterinary Surgery.* J. J. O'Connor, M.R.C.V.S. 3rd Edition, 1938; pp. 990 + IX. Price £1-10-0. Ballière, Tindall & Cox, London.

[2] *Special Pathology and Therapeutics of the Diseases of Domestic Animals,* by Hutyra and Marek. Fourth English Edition (1938), edited by J. Russel Greig. Vol I, II and III; pp. 947, 687, and 731. Price £6-6-0. Baillière, Tindall & Cox, London.

the giving of a dose of acaprin, which is twenty times the recognised one, etc., would not occur if the section were compiled by an authority on the subject.

The next edition also calls for a considerable amount of attention to the correction of typographical and other errors. These are mainly in connection with the spelling of the South African names of diseases, but also of proper names (Thelier for Theiler, Koch for de Kock, Parker for Parkin, etc.). Some, however, are concerned with drugs (quinine for quinidine, leutin for lentin, etc.).

Many references to authors and research workers are given in the text. Such incomplete references are of little value to those who wish to consult the original works.

This edition, notwithstanding the minor errors and omissions, can be strongly recommended to practitioners, research workers and students.

B. S. P.

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The new edition of Kelser's *Manual of Veterinary Bacteriology*[3] differs but little from the previous one and republication has been carried out to meet a demand for further copies rather than to bring the material up to date. That the demand has been sufficient to call for a 3rd edition indicates that the book must play an important rôle in American veterinary education, for which purpose it was originally written. Nevertheless it is very far from being a desirable textbook for students, being uninspiring to read and containing incorrect statements, and omissions which render the book extremely misleading to beginners.

In order to justify these criticisms a few specific points may be cited. Thus in a book which covers the vast field of bacteriology (including viruses) and protozoology, five pages are wasted on a description of how to build a home-made Mackintosh & Fildes jar for anærobic cultivation, whilst no mention is made of the commonly used and easily purchased bronze anærobic jar. The recipes for numerous obscure varieties of culture media are detailed, whilst a commonly used medium such as McConkey agar is not even named.

After starting the chapter dealing with immunity in a promising fashion, the author then drifts off into an uncritical description of Ehrlich's side-chain theory and ultimately leaves the reader with the impression that this covers all that there is to be said upon the subject. In common with all other writers of textbooks of bacteriology the author has completely failed to reduce the chapter dealing with the streptococcus group to coherence. The

[3] *Manual of Veterinary Bacteriology,* by Raymond A. Kelser, D.V.M., A.M., Ph.D. Chief, Veterinary Division, Surgeon-General's Office, War Department, Washington. 1938. pp. 640. Price £1-7-6. Baillière, Tindall & Cox, London.

name *Corynebacterium pseudotuberculosis* is given as a synonym of *Pasteurella pseudotuberculosis*, and in describing the preparation of vaccine against haemorrhagic septicaemia no mention is made of the significance of the capsular material of *Pasteurella septica*. By retaining Bergey's and ignoring the Kauffmann-White classification of the genus Salmonella the writer has rendered his description of this group chaotic. For instance, a single organism is described thrice, firstly as *S. aertrycke*, then as *S. typhimurium* and finally as *S. psittacosis*. Then again by ignoring the antigenic method of classification, *S. gallinarum* and *S. pullorum* have been described in separate genera, although these organisms are antigenically identical and differ only in certain minor fermentative properties, and in the epidemiology of the diseases they produce. The reviewer would question strongly the statement that, if agglutinins for *Br. abortus* are present in the blood of children, these have arisen by absorption of antibodies from cow's milk which has been drunk. In dealing with immunisation against anthrax, spore vaccines are not even mentioned. The statement is made that artificial culture vaccines of *Cl. chauvoei* (i.e. dead organisms + toxoid) have little immunizing ability. No description is given of the different toxins produced by types of *Cl. welchii*. For some unexplained reason the writer insists upon differentiating the cause of Lamsiekte from *Cl. botulinum* and calling it *Cl. luciliae*. The relative parts played by *Actinomycosis bovis* and *Actinobacillus lignieresi* in the causation of "lumpy jaw" and "wooden tongue" are not made clear. The name *Pasteurella muriseptica* is given as a synonym for *Erysipelothrix rhusiopathae*, whilst in the section dealing with tuberculosis is to be seen a picture of a luxuriant growth of *Mycobacterium tuberculosis* (bovine type) on agar.

The foregoing examples justify, in the reviewer's opinion, the statement that the book is unsuitable for students. The examples cited have been picked at random from the section dealing with bacteriology, but the other sections of the book might just as easily have been criticised; thus when describing Nagana, *Trypanosoma brucei* is considered, but *T. congolense* and *T. vivax* are not even named.

E.J.P.

* * *

On opening Professor Panisset's *Traité des maladies infectieuses des animaux domestiques*[4] the first thing that strikes the reader is the peculiar arrangement of the contents. There is no index, but there is a table of contents at the end. The book commences abruptly with a description of anthrax, but there is no attempt at any classification of the diseases described. It is not easy to grasp for whom the book was written as there

[4] *Traité des maladies infectieuses des animaux domestiques* (Infectious diseases of domesticated animals), by Professor L. Panisset, National Veterinary School, Alfort, Paris. pp. 562. Price 120 fr. Vigot Frères, Paris.

is no introduction nor preface. The arrangement of the diseases is rather haphazard; the bacterial and virus diseases being in no special order, distemper of dogs, for instance, appearing between strangles and tuberculosis.

Certain very important diseases such as contagious abortion, botulism and actinomycosis are not even mentioned. The only protozoal disease discussed is dourine of horses, which comes at the end of the book. A large number of authors are quoted in the text, in the descriptions of the various diseases, but as only the name and date are given, one could not easily refer to the original papers. No bibliography is given.

Apart from the drawbacks mentioned, the individual diseases are quite well described and there is evidence of a real attempt to collect most of the available information. In some of the diseases the information has not been brought up to date. To quote one instance: in the chapter on horse-sickness there is no mention of the immunization method based on the use of the neurotropic virus obtained by passage through mice.

One is forced to conclude that the book was written to describe a few of the infectious diseases of domesticated animals in which the author was particularly interested, but the omissions and the haphazard arrangement of the material constitute serious defects in an otherwise readable book.

E. M. R.

NEWS.

Mr. G. P. Bishop, a fourth-year student, has been awarded the S.A.V.M.A. Book Fund Prize for 1939. Mr. Bishop is the first recipient of this prize, which is to be awarded annually.

* * *

The Arnold Theiler Memorial Medal donated by the South African Biological Society has been awarded to Mr. P. R. Mansvelt, who obtained his B.V.Sc. degree in December last.

* * *

Messrs. A. C. Kirkpatrick and C. J. van Heerden were elected to represent the Association on the South African Veterinary Board, for the next three years.

* * *

We are pleased to announce that Mr. C. J. van Heerden, our Vice-President, has completely recovered and resumed duty after a serious attack of malaria contracted while on foot-and-mouth disease duty.

* * *

Mr. J. W. A. Brooks has resigned from the Division of Veterinary Services in order to take up private practice in Johannesburg.

OBITUARY.

William Edward Footner, *M.C.*, M.R.C.V.S.

The death of Captain Footner, which occurred at the Berea Nursing Home, Durban, on March 25th, 1939, came as a great shock to his many friends and colleagues. Although he had been in indifferent health for some time good hopes for his complete recovery were entertained up till a day before he died.

Footner was born in London 58 years ago, qualified at the Royal Veterinary College, London, in July, 1909, and joined the R.A.V.C., attaining the rank of Captain. At the outbreak of the Great War he went to France and was attached first to the Cavalry Division and then to the Guards. He was awarded the Military Cross and was mentioned in despatches for gallant and conspicuous services.

After the war he came to South Africa as a settler on the Sundays River Irrigation Scheme, but abandoned farming and joined the Union Veterinary Division in January, 1922. After serving for brief periods as Government Veterinary Officer in Bloemfontein and Pretoria he was transferred to Durban.

He was an esteemed member of the Durban S.P.C.A. and always displayed a keen desire to alleviate suffering in both man and animals. He displayed particular interest in the humane slaughtering of animals and invented the Footner Electric Collar, which is considered to be a great step forward in humane slaughtering.

Footner was a man of kind and lovable nature and showed a deep thoughtfulness for others, and was always prepared to render assistance where this was required. The profound sympathy of all our members will go out to his wife in her irreparable loss.

* * *

Fred Hutchinson, M.R.C.V.S.

In the death of Mr. Fred Hutchinson the Association lost one of its oldest and most respected members.

Born on 17th January, 1869, at Foggathorpe, Yorkshire, he was one of three brothers all of whom became veterinary surgeons. He qualified in Edinburgh in May, 1889, and came to South Africa in 1895 to manage the stud farm at Beginsel, Standerton. In 1899 he was appointed Government Veterinary Officer in the Natal Civil Service and was stationed at Newcastle. On the outbreak of the Anglo-Boer war he was granted a commission in the Veterinary Corps with the rank of Captain and was placed in charge of remounts at the Point, Durban. On completion of hostilities

he returned to his civil post at Newcastle, where he remained until his headquarters were removed to Dundee, in 1909.

During the Great War he saw military service in the Veterinary Corps, with the rank of Major.

In 1921 he was transferred from Dundee to Pretoria as Senior Veterinary Officer, Transvaal, and on being retired on pension in 1925 he settled at Hilton Road, Natal, where he remained up till the time of his death on March 20th, 1939.

During his younger days he developed a keen interest in horsemanship which he maintained right up to his death, and his reminiscences of the old horseback and mule-cart days were packed with humorous and exciting incidents. He was a well-known figure at agricultural shows where he appeared as a competitor in jumping events, winning many trophies, and he also acted as a judge, especially of thoroughbred horses.

* * *

William Alexander Simson, M.R.C.V.S.

We deeply regret having to record the death on Good Friday, 7th April, of Mr. W. A. Simson, Government Veterinary Officer, Queenstown.

Mr. Simson was born in Kilkerran, Ayrshire, Scotland, qualified at the Royal Veterinary College, London, on 16th July, 1903, and joined the Veterinary Division of the Cape Government in October, 1908. After being stationed for short periods in Vryburg, Mafeking and Cradock he was transferred to Queenstown, where he served up till the time of his death.

He was of a quiet, retiring and almost shy disposition, and shunned publicity at all times, and was accordingly not well known to very many members of the profession. Those, however, who had the privilege of coming into contact with him came to regard him as a man of strong character, a good friend and one who had a very thorough knowledge of veterinary science. His opinion on veterinary problems, never voluntarily given, could always be accepted as sound and practical. He was therefore held in high esteem by the farming community and enjoyed a wide circle of friends, especially in the Queenstown area.

Mr. Simson, who died in his 60th year, is survived by a widow, a daughter, and three sons, the eldest of whom is studying medicine in Scotland. To them we extend our sincere sympathy in their sad bereavement.

THE ASSOCIATION.

Council Meeting held at Carlton Hotel, Johannesburg, 4th April, 1939.

Present: S. T. Amos (President), P. J. du Toit, V. Cooper, A. C. Kirkpatrick, A. D. Thomas, R. Alexander, D. Coles, M. Sterne, P. J. J. Fourie, H. H. Curson, J. H. Mason, H. O. Mönnig, G. v. d. Wath, and S. W. J. van Rensburg (Hon. Sec.-Treas.).

APOLOGIES FOR ABSENCE: C. J. van Heerden, A. S. Canham, and A. M. Diesel. The latter two were represented by Messrs. Cooper and Kirkpatrick.

(1) MINUTES OF MEETING HELD ON 24TH OCTOBER: These had been circulated and were taken as read and were confirmed.

(2) MATTERS ARISING FROM THESE MINUTES: (*a*) *Arrear* subscriptions of two members were considered. In the case of one it was decided to let the matter stand over in view of the member's serious illness, while the other is to be dealt with according to rule 7(b) of the Constitution.

(*b*) *Complaints* against a Stock Inspector who was alleged to have done veterinary work had been investigated, and in view of the report received it was decided that no further action be taken.

(*c*) *Dr. Schulz:* The Secretary reported that Mr. C. W. Clark, M.P., had consented to submit a petition to Parliament on behalf of Dr. Schulz, during the present Session.

(3) VETERINARY OFFICER: S.A. PERMANENT FORCE: The Secretary pointed out that the conditions pertaining to this post were not satisfactory and that no applications would be made unless the grievances were rectified.

Dr. du Toit stated that as the result of discussions which he had with certain Heads of Departments that afternoon he hoped the question would be settled satisfactorily in the near future.

The President thanked Dr. du Toit for the action he had taken in the matter and suggested that further action be entrusted to Dr. du Toit and the Secretary.

(4) MEAT INSPECTION: The Secretary stated that the Sub-Committee appointed by the general meeting had circularised a large number of abattoirs and obtained valuable information. Dr. du Toit said the contents of the two resolutions passed at the General Meeting had already been brought to the notice of the Secretary for Agriculture. The President pointed out that the question of measles was assuming serious proportions, and that proper supervision was apparently not always kept, as infected meat was sometimes taken out of cold storage before the necessary period of freezing had expired. It was agreed that the Sub-Committee be asked to correlate

all the information and to submit it in the proper form to the Secretary for Agriculture through the Director of Veterinary Services.

(5) TRANSLATION OF THE CONSTITUTION: The report submitted by the Sub-Committee indicated that there was a difference of opinion between the members of the Committee as to the most suitable translation of the name of the Association. After full discussion it was decided to adopt " Suid-Afrikaanse Veterinêr-Mediese Vereniging."

A motion of thanks to Dr. Mönnig and the other members of the Committee was passed unanimously.

(6) " VETERINARY ADVISER ": " SUNDAY TIMES ": The Secretary reported that he had to complain to the " Sunday Times " about a breach of the regulations framed under the Veterinary Act in certain replies to veterinary questions in the " Farmer's Supplement." He was subsequently informed that the Veterinary Adviser to this paper had resigned and the Association was requested to find a substitute. After discussion it was decided to advise that all veterinary queries be referred to the Director of Veterinary Services.

(7) BOOK FUND PRIZE: The meeting was informed that the Faculty of Veterinary Science had recommended Mr. G. P. Bishop for the S.A.V.M.A. Book Fund Prize for 1939. This recommendation was unanimously accepted.

(8) INTERNATIONAL VETERINARY CONFERENCE: Dr. du Toit explained the constitution of such conferences. The country in which the conference is held usually bears the expenses. There are, however, certain incidental items such as printing and the cost incurred by the permanent Commission which has to carry on the work in the interim between conferences. In the case of small countries this may involve a severe financial strain on the country concerned. Therefore a resolution was passed at the last session to the effect that these two items be met by contributions from all participating countries, this to be proportional to the membership of the Associations represented. He considered that our contribution would be approximately £50.

It was resolved that Council agrees in principle and that the question be further considered when the exact amount required is known.

On the suggestion of the President it was decided that just before the next Conference members of this Association be circularised and advised of the fact that they can become members of the International Conference and receive all the publications on payment of £1-10-0.

(9) IMPORTATION OF HORSES: The Secretary stated that a member had enquired as to the fate of the resolution passed at the October, 1936, General Meeting. It was decided to refer to the Department of Agriculture for a reply.

(10) ANNUAL GENERAL MEETING: Decided that this be arranged by the General Purposes Committee in collaboration with Drs. du Toit, de Kock and Mr. van Heerden.

(11) GENERAL:

(a) *Complaint:* An advertisement of a certain dog hospital and boarding kennel that appeared in a newspaper was considered, following a complaint from a member. Since such cases are not covered by the Act, as it is as present, no action can be taken.

(b) *Publication of Journal:* Dr. Sterne explained the negotiations which were proceeding whereby it was hoped to get the Journal published at a reduced rate. The Committee's action was approved.

(c) *Late W. E. Footner:* The Secretary reported that by special request no wreaths were sent for the funeral. It was decided that the President should investigate the desirability of making a contribution to the proposed memorial fund and that, if this would be acceptable, £5-5-0 be sent.

(d) *Veterinary Board:* The meeting was informed that the recent election resulted in the return of Messrs. A. C. Kirkpatrick and C. J. van Heerden.

The Secretary was instructed to write to Mr. Carless, expressing Council's appreciation for the services rendered by him while a member of the Board. It was further unanimously decided to recommend to the next General Meeting that Mr. Carless be elected an Honorary Life Vice-President in recognition of the services rendered by him to the Association and to the Veterinary Profession in South Africa.

(e) *Finance:* Dr. Alexander as member of the Finance Committee reported that this committee had authorised an advance of £7 to the widow of a late member. This was approved.

The meeting adjourned at 11 p.m.

S. W. J. van Rensburg,
Hon. Sec.-Treas., S.A.V.M.A.

Jl. S.A.V.M.A.
X(3): 91—102
1939.

The Differential Diagnosis of Chronic Arthritis in Bovines.*

By J. G. VAN DER WATH, Onderstepoort.

This short paper on the differential diagnosis of chronic arthritis in bovines is presented with the object of drawing attention to the existence in this country of at least three major diseases, which are responsible for enlarged joints, whether these are due to serous or serofibrinous polyarthritis, tendovaginitis, bursitis, periostitis, or periarthritis. The diseases concerned are contagious abortion, chronic arsenical poisoning, and phosphorus (P_2O_5) deficiency.

With the possible exception of aphosphorosis (stywesiekte), only a few members of our profession in South Africa have recognised these conditions in the field. Martinaglia (1929) investigated salmonellosis and streptococcosis in tendovaginitis of equines in South Africa. He found an organism indestinguishable from *Salmonella abortivo-equinus,* and sometimes a streptococcus in cases of bursitis and tendovaginitis associated with horse-sickness inoculations. Martinaglia noticed that the sera of horses which had passed through an attack of tendovaginitis agglutinated *S. abortivo-equinus* to a high titre.

McFadyean, Edwards, and others have studied organisms similar to *S. abortivo-equinus* and consider them to be of ætiological significance in joint-ill in foals.

Recently at a veterinary conference in Vienna, Ueberreiter reported cases of chronic polyarthritis in horses whose sera agglutinated *Brucella abortus.* He thought that these horses were infected by cattle with which they were grazing. Frost has actually isolated *Brucella abortus* from a case of gonitis in a horse. Joint lesions were also observed by Baumann and Kuscher in guinea pigs infected with *Br. abortus.*

It is therefore clear that *Br. abortus* may be associated with lesions of the joints, tendon sheaths, and bursæ, in horses and mules. The occurrence of a serous arthritis in man has been demonstrated by Hegler (1934), who isolated *Br. abortus* from the joint exudate. The shoulder, hip, knee and ankle are the most frequent seats of affection, and osteological changes have been demonstrated by means of X-rays. Similarly it has been known that cows suffering from contagious abortion may develop bursitis, but this has never been reported in South Africa, where it would appear this disease occurs to a considerable extent.

* Paper read at the 33rd General Meeting of the S.A.V.M.A. at Onderstepoort, October, 1938.

The South African veld is well known for its phosphorus deficiency which leaves its impression on cattle in degrees varying according to the availability of this element in pasture and soil. Seasonal deficiency is reflected by the disease known as *stywesiekte* and is seen in the various deficient areas. Annual deficiency allows development of stywesiekte and the further advanced form, viz., rickets.

Affections of joints in cases of phosphorus deficiency were noticed as early as 1884 in Griqualand West by Hutcheon, who reported that he had seen symptoms typical of rachitis.

Theiler (1912) noticed cases of stywesiekte with enlarged joints in the Western Transvaal, Bloemfontein district, Stellenbosch, Grahamstown, and Middelburg (Tvl.) and described the clinical appearance. In May, 1937, the writer saw many animals in the Transkei which showed lesions similar to those described by Theiler, only more accentuated, because animals with advanced natural rickets were actually found in large numbers.

In regard to *mineral poisoning* it is reported by Grimmett and others that chronic zinc poisoning was produced in pigs feeding from galvanized iron troughs and that these pigs were unthrifty, developed chronic ulcerative arthritis and some died. Experimentally rickets has been produced in rats by chronic strontium poisoning (Sobel, Cohen and Kramer, 1935). Petri, in her work on the pathological anatomy and histology of poisoning, merely mentions that chronic arsenical poisoning produces an ostitis and periostitis. Chronic arsenical ulcerative arthritis, periostitis and periarthritis on a large scale was noticed by Steyn in cattle on farms in the Northern Transvaal in 1934 and subsequently on various other farms.

Aphosphorosis.

Last year the writer investigated the occurrence of aphosphorosis in the Transkei as reflected in cattle suffering from " stywesiekte " and natural rickets. It was found that these conditions occurred along parts of the coastal belt of the Transkei and Tsitsikei and also more inland, *e.g.*, in the district of Umtata on the farm " Belmont " and elsewhere. On these farms the P_2O_5 content of the soil and grass is insufficient to prevent the insidious onset of stiff sickness and rickets. With heavy overstocking the condition developed also on some farms, on which otherwise it might not have made its appearance.

Large numbers of cattle were examined for clinical symptoms in different areas. Blood samples were drawn from clinically affected animals and ribs were resected from the same subjects for histological examination. Grass and soil samples were collected for chemical analyses of P_2O_5 and other constituents.

In this way it was found that in parts of the coastal belts of Flagstaff and East London districts, which are the worst areas, 60 to 70 per cent. of

the animals constantly grazing there were obviously affected and showing markedly enlarged joints.

There was usually a history of the occurrence of stiff sickness, frequent fractures of legs, and infertility. The more common clinical symptoms were cracking movements of the joints when in motion; the joints of the fore-legs, particularly the carpi and fetlocks, were prominently enlarged; the hocks and hind fetlocks were always less affected. The enlargements were bilateral, hard, and of bony nature due to increased size of the epiphyses of the long bones. On palpation they were sometimes painful and warm. There was no visible evidence of affected synovial structures. The animals rose with difficulty and had a stiff gait. These enlarged joints were seen mostly in young animals up to two and even three years old. The size of the enlarged joint seems to undergo a proportionate decrease with increasing age. Adult cows or oxen were seldom seen with large joints, although some of them were still markedly bandy-legged and knock-kneed due to weakened ligaments and constitution. There definitely seemed to be an atony of the musculature. Typical stiff sickness was seen, especially amongst lactating cows when the supply of P_2O_5 was low and drainage from the system high and continuous. Atrophy of the udder with a decreased milk yield always followed; sterility was prevalent; various degrees of pica were exhibited. Marked stunting, physical weakness, soft bones, fractured legs and ribs, and coarse bony skeletons were symptoms frequently seen in severe cases. (See figs. 10 to 13.) It was noticed, too, that in the parts where the disease was most severe, the grass was evergreen, though very coarse and unpalatable; the rainfall averaged from 40 to 60 inches per annum. If the grazing of affected cattle was changed from the "flats," where the disease was most severe, to valleys with alluvial soil and a "sweet" vegetation, recovery followed quickly as a rule.

Histological examination of the resected ribs was subsequently carried out at Onderstepoort and a diagnosis of various degrees of P_2O_5 deficiency established, including some cases of florid rickets. The blood, grass and soil analyses revealed a very low P_2O_5 content.

In Bechuanaland, where stiff sickness occurs, affections of the joints are seen, but are not so severe as in the coastal belts.

In experimentally produced calcium deficiency in bovines at this Institute, chronic arthritis also manifested itself, although not necessarily with bony enlargement. Natural occurrence has, however, not yet been encountered in cattle in South Africa, although such a disease has recently been reported in horses in the wheat districts of the Cape Province (Bredasdorp) and is known there as "*Beensiekte*," a condition which corresponds to the so-called "Big Head" or "Bran disease" of the Far East.

Contagious Abortion.

The widespread occurrence of contagious abortion in cattle in South Africa is well known. In urban dairies many cows with so-called "house-

maid's knees" are most probably suffering from nothing else than a bursitis or tendovaginitis due to a general infection of *Brucella abortus bovis*. On farms throughout the country the incidence of contagious abortion is high, even up to 70 per cent. on ranches in the Northern Transvaal.

The writer recently examined a herd of cattle consisting of Africanders and Aberdeen Angus-Africander crosses on a ranch in the bushveld area of Pietersburg district. It was noticed that a fair number of cows and several oxen showed enlarged joints due to large fluctuating swellings of the precarpal bursæ, joint capsules and tendon sheaths. In some instances the swellings extended up the forearm for 15 to 20 cm. (see fig. 4). Others again, on the dorsum of the knee, extended halfway down the metacarpus and had a diameter of anything up to 15 cm. (see fig. 6). In the hindlegs the distensions were usually less prominent.

In the earlier stages there is moderate lameness and the condition consists of a bursitis, tendovaginitis and arthritis of a serous nature; but in older, well-established cases it develops into a periostitis and periarthritis with marked proliferation around the joint (see figs. 8 and 9). Marked lameness, pain, and a stiff gait follow. The animals most affected were in the poorest condition, due mostly to inability to reach good grazing and water. The fact that these animals had to walk long distances to water and to graze was probably a predisposing cause to the local effects of the *Brucella abortus bovis* on the most vital parts of the organs of locomotion.

Fluid withdrawn aseptically from those swellings was submitted to Onderstepoort for bacteriological examination, as there was a history of contagious abortion in the herd. It gave a positive agglutination test and *Br. abortus bovis* was isolated from it. The herd, which consisted of 488 cows and heifers and 12 bulls was then tested for contagious abortion: 225 females proved to be positive, with 60 doubtful reactors, leaving only 203 as negative; of the positive reactors it was found that 27 per cent. had enlarged joints. Only one ox was tested and it was found to be positive. From its synovial fluid the organisms were isolated. Fig. 8 shows sections of the knees of this ox, which had to be slaughtered as the condition of its joints had become so bad and so painful that it could hardly walk. In addition to a polyarthritis, polybursitis and polytendovaginitis, the joints, particularly the knees, showed a well-established periostitis and periarthritis resulting in the formation of a thick band of fibrous tissue around the bony elements. In the Eastern Transvaal similarly enlarged joints have also been observed by me in Africaneers suffering from contagious abortion. At Onderstepoort a grade Africander cow showed tremendously enlarged precarpal bursæ and tendon sheaths and on testing was found to be positive for contagious abortion. Figs. 6, 7 and 9 show the legs of this animal. A sero-fibrinous exudate fills the precarpal bursæ, which have become enveloped in thick fibrous capsules.

Chronic Arsenical Poisoning.

Chronic arsenical poisoning is liable to occur wherever extensive and heavy and sometimes negligent spraying operations of arsenic have been conducted during the locust campaigns. The veld and water reservoirs are poisoned, although the toxicity diminishes with time and in many instances becomes negligible.

There are unfortunately, however, still farms on which no stock can thrive and for years to come will be dangerous to any animal kept long enough to allow of the slow but certainly deleterious effects of chronic arsenical poisoning to develop. Farms are known where up to 95 per cent. of cattle showed clinical symptoms of chronic arsenical poisoning, the majority of them having enlarged joints. (See figs. 1 and 2).

Steyn (1934) has observed that in cases of chronic arsenical poisoning in cattle enlargement of the joints is a symptom practically always seen. Even in young calves exposed to poisoning for periods of three to six months he has noticed prominently enlarged joints. The enlargements are much more prominent in the forelegs, but are not necessarily bilateral. Adult animals may show tremendously enlarged knees, which are hard and painful; lameness and stiffness supervene (ostitis). Synovial structures are not usually affected, but the articular cartilage may be ulcerated (arthritis). The enlargement is due to thickening of the ends of the long bones coupled with the development of thick fibrous tissue around the joint (periarthritis). Poor condition, according to the degree of poisoning, is usually seen in affected animals and some may die from unthriftiness.

Other conditions, similar to those described above, but of different ætiology, may be responsible for some degree of confusion and have to be considered:—

(1) Arthritis, tendovaginitis or bursitis due to *traumatic influences,* e.g., hygroma or "housemaid's knee" and swellings on any of the joints, particularly of the fetlock and pastern, due to sprains or luxations, frequently seen in hilly country; ankylosis and exostoses.

(2) Infectious diseases, amongst which are joint ill, chronic tubercular arthritis and post-partum arthritis in cows.

Differential Diagnosis.

In cases of traumatic origin the lesion is usually unilateral with a history of previous acute lameness and swelling. It occurs sporadically.

Hygroma or a serous distension of the precarpal subcutaneous bursa with an extension to the sheaths in advanced cases, is usually seen in stabled animals due to injury from hard floors. A definite diagnosis can only be made after the animal has been tested for contagious abortion. If the test is negative the diagnosis is safe, but if it is positive, fluid has to be withdrawn aseptically from the swelling and submitted to cultural and biological tests for *Br. abortus bovis.*

Cases of *tubercular arthritis, post-partum arthritis* in cows and arthritis in recovered cases of *joint-ill* are rare and usually occur singly. In joint-ill the history is important. Post-partum metastatic infections are usually of a purulent nature and, in the acute stages, are associated with hyperthermia, great pain, and lameness. Eventually in the chronic state exostoses form and ankylosis follows. If there is no history or outward evidence of a previously open joint, a clinical diagnosis is difficult and one has to resort to tuberculin, agglutination, cultural, or histological tests. *Chronic tubercular arthritis* may be suspected in animals which are known to be or appear tuberculous; application of the tuberculin test may be necessary, otherwise the position is the same as in chronic post-partum arthritis.

Contagious Abortion. — If several animals with enlarged joints are present in a herd the history would be one of the first points to be enquired into. If evidence of contagious abortion and a slow development of the enlargements is obtained, the herd should immediately be tested for contagious abortion. In addition to this, fluid should be withdrawn aseptically from the swellings and submitted for bacteriological examination.

Should there be no history of contagious abortion, the possibility of *chronic arsenical poisoning* or P_2O_5 *deficiency* should be kept in mind. With arsenical poisoning, a history of spraying for locusts or other arsenic polution should be investigated. If no spraying has been done there may be a dipping tank constituting a source of arsenic. Material like bones of dead animals, pieces of skin, hoofs, horns or hair should be collected for chemical analysis, likewise samples of grass, shrubs and water. Blood samples of affected animals should be submitted for agglutination tests to exclude contagious abortion. In any case, to establish a diagnosis of chronic arsenical poisoning there must be, in addition to history and other evidence, a definite proof by chemical determination of the presence of arsenic in the bones.

When there is no history of either contagious abortion or chronic arsenical poisoning one has to think of mineral deficiencies, the most likely being *aphosphorosis.* Clinical symptoms and the progress of the disease, which is of an insidious nature, are important. Failure to feed bonemeal or if bonemeal or its equivalent is fed to a section of the animals, e.g. the milch cows, with good results would definitely point to P_2O_5 deficiency. Bones, preferably pieces of ribs with the costo-chondral junction intact, should be obtained either by biopsy or at autopsy, for histopathological examination. To place the diagnosis beyond doubt, samples of blood, soil and pasture should be submitted for chemical determination of P_2O_5.

One could conceive of all or a number of the conditions mentioned above occurring in a single herd of cattle, when the question of a diagnosis will not be easy and will resolve itself into a process of elimination, based, not a superficial observation, but on thorough investigation and application of the laboratory methods at our disposal.

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Discussion.

Dr. A. D. Thomas: Mr. v. d. Wath has done the profession a great service in focussing attention on the significance of swollen joints in cattle. Most of us have seen at some time or other unusual manifestations of Brucellosis such as for instance necrotic orchitis in bulls, but I must confess that I would not have suspected the association of extensive serous bursitis and tendovaginitis with contagious abortion.

I know that Mr. v. d. Wath was at first mainly interested in recording the occurrence of Brucellosis as an affection of synovial membranes. His action in bringing other types of swollen joints into consideration from the differential point of view is to be commended as it presents the difficulties as well as the importance of these changes clinically.

Although swollen joints may thus mean that one of half-a-dozen different diseases may be present, they nevertheless constitute symptoms of great value in the field as pointers to likely tests that should be carried out for diagnosis. In fact, such unmistakable clinical changes may well be the means of spotting the existence of one of these conditions whose presence may not otherwise have been suspected, since mortality is not usually present.

Little can be found in the literature regarding the pathology of these conditions and no systematic work has yet been carried out here. In the cases before you the following changes have been noted: great enlargement of precarpal bursa with a wrinkled, fibrous membrane (organised fibrinous pseudo-membrane), lined with a necrotic layer, on which clumps of Gram-negative bacilli are to be found, and thrombosis of small blood vessels is frequent. There are of course a number of bacterial diseases in which the organisms affect the synovial membrane either during or after the main attack.

I have not yet seen a suitable explanation advanced for these so-called secondary invasions. In some diseases like strangles and joint-ill the invasion is described merely as metastatic. To me there is something more to it than that. Assuming that the organisms during the primary disease do become distributed throughout the body tissues by means of the blood and lymph streams, then it seems fairly obvious that if the joints and sheaths become infected in preference to other tissues then there must be some predisposing factor in the joint to account for it.

In the herd under consideration, swollen knees were not in evidence while the cattle were still on the high veld, although contagious abortion must have

been present. It was only when they were transferred to the bush veld, where much travelling over rough country to and from grazing, water and kraals had to be done that the swollen knees became apparent.

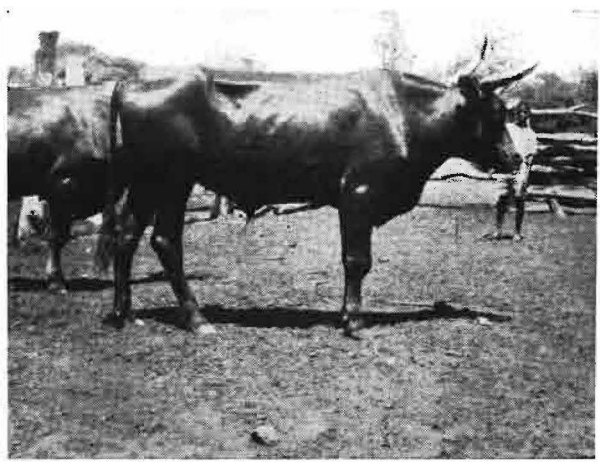
It would appear, therefore, that functional stress of the sheaths and bursæ, i.e. excessive pressure and rubbing of tendons and joints, constitutes one of the predisposing factors to such secondary infections of synovial membranes.

In arsenical poisoning the process is entirely different. As you know, sublethal doses of the poison are gradually accumulated and fixed in the bone, where it produces a chronic inflammatory process. The articular cartilage may be pitted or ulcerated, but the synovial envelopes are not affected. The thickening is due to fibrous periostitis and periarthritis.

Of course, in aphosphorosis or *stywesiekte* the changes are typical of rickets and affect the whole skeleton. Clinically, however, the most noticeable symptoms are the stiff gait and the enlargement of bony extremities, especially of the limbs at the knee and the fetlock joints. The enlargement here is due partly to formation of exostoses and partly to an over-production of soft bony tissue (osteoid), which tends to bend or expand mushroom-like under the weight of the animal.

CHRONIC ARSENICAL POISONING.

1. — *Note enlarged right knee.*

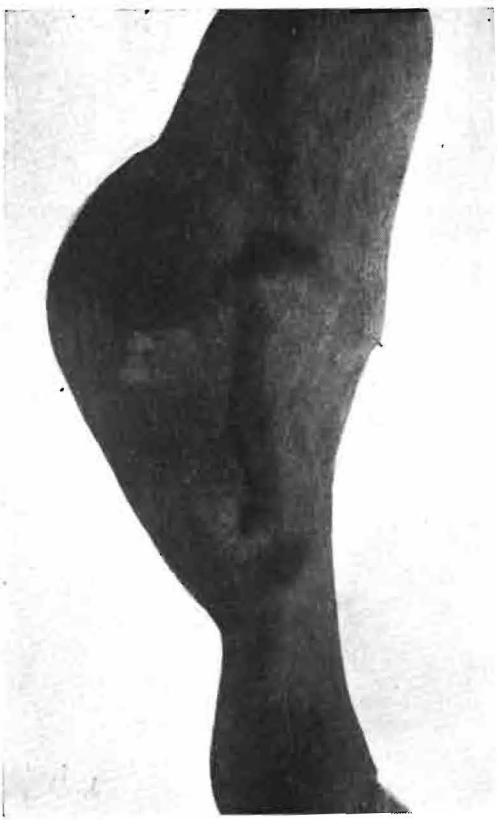


2. — *Note enlarged right knee.*

3. — *Cow: Note distended tendon sheath above right knee.*

4. — *Cow: Note distended tendon sheaths and precarpal bursæ on both forelegs.*

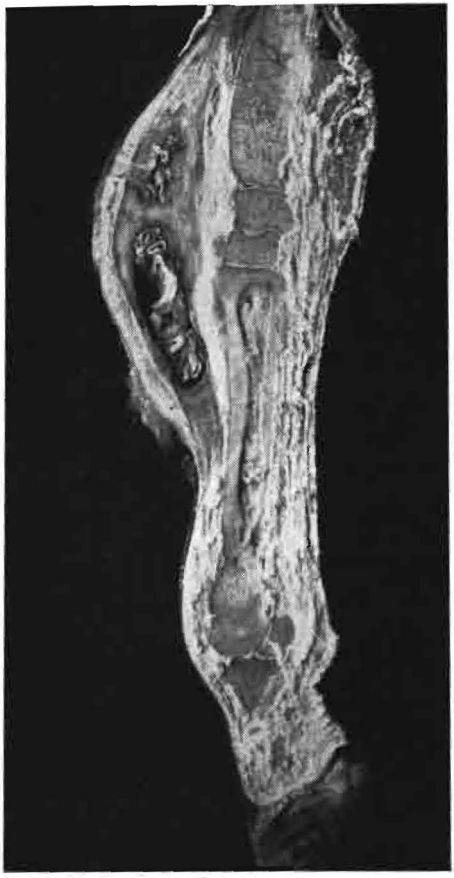
5. — *Cow with large hygroma of left knee.*



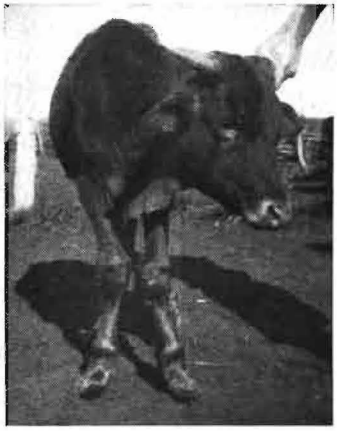
6. — *Cow with large hygroma left knee.*

7. — *Same cow showing distension of lateral tendon sheaths of hock.*

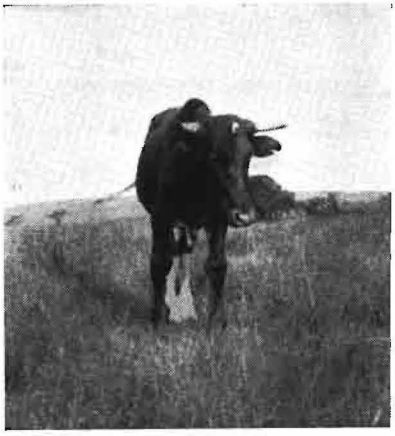
8. — *Cross and sagittal sections of carpus of ox. Note thick fibrous band around joint.*



9. — *Sagittal section of No. 6 showing serofibrinous exudate in precarpal bursæ and thick fibrous bands surrounding joint.*



10. — *Eighteen month old bull calf showing marked knock-knees and enlarged joints.*



11. — *Two year old heifer with bowlegs and enlarged joints.*

12. — *Three year old ox with markedly enlarged fetlocks.*

13. — *Ox showing site where rib was resected.*

Jl. S.A.V.M.A.,
X(3) : 103—105
1939.

The Appearance of *Bartonella canis* in a Dog Suffering from *Rickettsia canis*.

By W. O. NEITZ, Onderstepoort.

Introduction.

In the course of a series of *Piroplasma canis* experiments, Kikuth (1928) found round and rod-shaped parasites in and upon the erythrocytes of some of his splenectomized dogs. These organisms were described and named by him *Bartonella canis*. In splenectomized dogs this infection produces an anæmia which may become so severe as to cause the death of the animal. These observations have been confirmed by several workers mentioned in the sub-joined table.

Bartonella canis has also been recorded in non-splenectomized dogs. Kikuth (1932) demonstrated these organisms in dogs reacting to *P. canis*. MacNaught & Woods (1935) found them in a dog during the course of a plasmapheresis experiment and Ray & Idnana (1938) in animals suffering from *Babesia gibsoni* as well as in dogs in which no other blood parasites could be demonstrated.

Table showing the appearance of *Bartonella Canis* in different countries.

Host.	*Country.*	*Author.*	*Year.*	*Remarks.*
Dog.	Germany.	Kikuth.	1928.	Parasites appeared after splenectomy. *B. canis* was observed in non-splenectomized dogs which were suffering from a severe *P. canis* infection.
Dog.	France.	Perard.	1929.	Parasites appeared after splenectomy.
Dog.	Russia.	Yakimoff & Rastegaieff.	1931.	Parasites appeared after splenectomy.
Dog. Cat.	Germany.	Regendanz & Reichenow.	1932.	Parasites appeared in the dog after splenectomy. Splenectomized cats found to be susceptible to *B. canis*.
Dog.	U.S.A.	Knutti & Hawkins.	1935.	Parasites appeared in splenectomized bile-fistula dogs.
Dog	U.S.A.	Rhoads & Miller.	1935.	Parasites appeared in splenectomized dogs.

Table showing the appearance of *Bartonella canis* in different countries—(continued.)

Host.	*Country.*	*Author.*	*Year.*	*Remarks.*
Dog.	U.S.A.	MacNaught & Woods.	1935.	Parasites appeared in a non-splenectomized dog in the course of plasmapheresis experiments.
Dog.	Spain.	Goyanes.	1936.	Parasites appeared after splenectomy.
Dog.	Peru.	Weiss & Muzzo.*	1936.	Parasites appeared after splenectomy.
Dog.	India.	Ray & Idnani.	1938.	Parasites produced febrile reactions and anæmic changes in non-splenectomized dogs.
Dog.	Union of South Africa.	Neitz.	1939.	*B. canis* found in a dog suffering from *Rickettsia canis* infection.

* Quoted by Weinman & Pinkerton.

OBSERVATIONS.

A strain of *Rickettsia canis* which parasitizes the monocytes as well as the neutrophiles was isolated from a dog in the Kruger National Park. This infection was maintained in dogs by passage. Up to the present, forty dogs have been used for this purpose and all have died. Although in many cases *R. canis* was responsible for the deaths, *P. canis*, which appeared in most of the animals either as the result of the injection of infected blood or due to the activation of a latent infection, aggravated the disease. In addition to the blood parasites mentioned it was noticed that about 40 per cent. of the dogs harboured *Hepatozoon canis* in the neutrophiles. This parasite apparently did not adversely affect the health of the animals, but it was observed that some of these leucocytes were parasitized by both *Hep. canis* and *R. canis*. The proportion of the neutrophiles showing a mixed infection of these parasites was much higher in dogs harbouring many *Hep. canis* gametocytes than in animals with a relatively light infection. From this observation it would appear that *Hep. canis* modifies the cytoplasm in such a way as to make it more attractive to and suitable for the development of *R. canis*.

In one of the dogs suffering from rickettsiosis and harbouring both *Hep .canis* and *P. canis* (latent infection), blood parasites identical with those described as *Bartonella canis* were observed. A febrile reaction due to *R. canis* was noticed from the 8th to the 16th day after infection. On the 28th day *B. canis* appeared in small numbers and approximately 0·5 per cent. of the erythrocytes were parasitized. There was a gradual increase

of these parasites and on the 31st day about 1 per cent. of the red blood cells were infected. Their numbers decreased and on the 36th day no organisms could be demonstrated. The anæmia that was already present before the appearance of *B. canis* became very marked after their disappearance. On the 46th day the dog died and, in lung smears, numerous *R. canis* were present.

Conclusions.

1. *Bartonella canis* is recorded for the first time in South Africa. It occurred in a non-splenectomized dog suffering from *Rickettsia canis* infection.

2. A latent infection of *Bartonella canis*, in non-splenectomized dogs, may be activated by *Piroplasma canis, Babesia gibsoni* or *Rickettsia canis.*

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Jl. S.A.V.M.A.,
X(3) : 106—110
1939.

Two Cases of Congenital Porphyrinuria ("Pink Tooth") in Friesland Cattle.

By CYRIL FLIGHT, Oudtshoorn.

Introduction.

Fourie (1936) described the occurrence of congenital porphyrinuria or "pink tooth" in South Africa, and dealt with a number of such cases which occurred in Swaziland, where the available evidence strongly suggested the hereditary transmission of the condition through a certain pure-bred Shorthorn bull. Rimington (1936) undertook chemical studies of the living animals and post-mortem material.

In October, 1938, at Onderstepoort I had the opportunity of examining a number of the Swaziland cases, as well as other cases, but none constituted such excellent clinical cases as did two animals which I had encountered in September, 1938, in the Cape Province. Especially as the effects of photosensitization were more marked in these than in Fourie's cases, a description may be of interest to veterinarians in the field.

History.

Mr. C. R. Human, of the farm Weltevrede, Ladismith district, C.P., reported that two of his young heifers had been suffering from an affection of the skin for approximately 9 months, during which time the urine from both animals had been reddish in colour. Numerous remedial agents had been applied to the skin lesions, with no improvement. As he owned valuable Friesland cattle, he feared that the condition might prove to be of a contagious nature. I accordingly visited the farm on the 19th September, 1938, and examined the animals in question.

Symptoms.

The animals, both of the same age, about one year, presented a miserable appearance, being very stunted and of the size of normal six-month-old calves, No. 2, called Lelieblom, being the more badly affected.

The symptoms were similar in both cases, viz., dejected appearance, dull staring coat, poor condition, infestation with lice, numerous and extensive lesions of the skin of the unpigmented parts of the head, body, limbs, etc., consisting of dry scabs and crusts, and a peculiar fan-shaped deformity of the hoofs, with a correspondingly increased interdigital space. This deformity of the hoofs was not seen by the writer in the Swaziland and other cases. The animals stood with both fore and hind feet spread well apart, and tired easily when driven; Lelieblom showed signs of distress

after being driven a short distance, lay down, and refused to move. When allowed to graze in the sunlight, both animals sought shade after a time.

The conjunctival, nasal, and buccal mucous membranes were slightly pale in colour. There was a slight mucous discharge from the eyes and nose, but no scabs or crusts were present.

The incisors and molars showed a very characteristic brownish-pink discoloration, identical with that seen in the cases described by Fourie.

The temperature at 11.15 a.m. in the case of No. 1, viz., Lemoen, was 103·2° F. and in the case of No. 2, viz., Lelieblom, 105° F.

It must be mentioned that it was a very warm day and both animals, particularly Lelieblom, were very restive and struggled a lot when brought from the paddock to the homestead, and while being examined. This no doubt had an effect on the temperature.

Left: No. 1 (Lemoen); Right: No. 2 (Lelieblom).

The pulse and respiration were accelerated, no doubt also partly due to the heat and unaccustomed handling.

Both animals were somewhat pot-bellied, but ate and drank normally, their food consisting mostly of green lucerne, grass, weeds, etc.

The fæces of No. 1 were very loose, probably due to the succulent food.

The urine had a characteristic appearance; that of No. 1 had a clear reddish-brown colour, and that of No. 2 was slightly turbid and amber-coloured.

What first struck the eye was the very extensive affection of the skin, confined, however, only to the unpigmented parts; those parts of the body covered by black hair were entirely unaffected. Practically all unpigmented parts of the body were affected, these areas being for the most part devoid of hair and the skin covered with dry, soft scabs and crusts, which bled easily when scraped with a knife. Down the limbs, and particularly round

the joints, a certain amount of dermatitis was present, the lesions presenting a raw appearance with numerous crusts and cicatrices. This condition was no doubt aggravated by flexion and extension of the limbs during movement, the absence of the normal protective coat of hair, and the action of the rays of the sun on the exposed surfaces. Animal No. 2, in particular, showed numerous injuries to the limbs as a result of walking through fences and bushes during attempts to find shade.

The unpigmented parts of the body not exposed to the direct rays of the sun, such as the ventral aspect of the thorax and abdomen, were unaffected and presented normal white hair. The lesions were clearly defined, and in no instance did they extend from the white to the adjoining

No. 1 (Lemoen): Crust on rump.

black parts of the skin. No lesions were present along the middle of the back where the hair parted, or on any other parts not covered by hair, as was seen in some of the Swaziland cases.

Lemoen showed a circular raised crust in the frontal region, entirely devoid of hair, about 8 cm. in diameter, and surrounded by coarse black hair; also a very extensive and irregular crust on the rump, approximately 30 x 20 cm. All four limbs of Lemoen were affected from the body downwards.

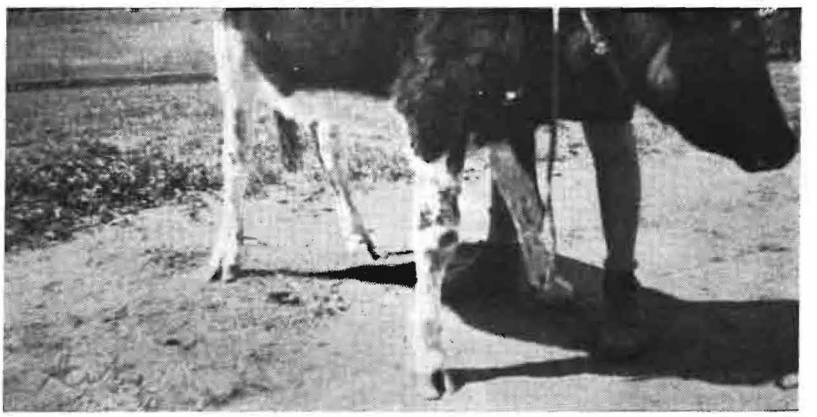
Lelieblom showed lesions mainly confined to the limbs.

Differential Diagnosis.

The history of the cases, the general debility and inappetence, the extensive lesions confined to the unpigmented and exposed parts of the skin, the hypersensitiveness to sunlight, the characteristic discoloration of the teeth and urine, and the negative skin scrapings and blood smears clearly differentiated this condition from other more commonly encountered affections of young cattle, such as ringworm, bovine mange, hornfly infestation, scalding due to dipping, keratosis of Friesland cattle, piroplasmosis, hæmaturia due to injuries to the bladder, etc.

The diagnosis was confirmed by the spectroscopic examination of the urine at Onderstepoort, when typical porphyrin bands were identified. The blood smears showed anæmic changes.

The first impression gained when seeing such cases is that one is dealing



No. 2 (Lelieblom): Lesions on limbs and hoof deformity.

with a primary affection of the skin. Closer examination at once reveals that the skin lesions and also the deformity of the hoofs are purely secondary, the former being due to hypersensitization, and the latter to general weakness with loss of tone and consequent relaxation of the digital ligaments, resulting in the fan-shaped growth of the hoofs and an increase in the interdigital space.

From the date of examination, viz., 19.9.38 until 2.11.38, on the advice of the writer, the animals were confined during the day to a shed and allowed to graze only at night. Lemoen showed considerable improvement, the white hair reappearing on the bare parts, but Lelieblom showed little improvement. On 2.11.38 they were despatched to Onderstepoort for breeding experiments.

Discussion.

These are the first cases of congenital porphyrinuria to be described among cattle in the Cape Province.

The writer feels convinced that the condition must occur in other parts of the Union, some cases no doubt being overlooked owing to the mild nature of the symptoms shown; the disease is probably sometimes mistaken by the owners for one or other of the affections mentioned under "differential diagnosis."

The question of the hereditary nature of the disease is being investigated by Fourie, so that this will not be dealt with. A few remarks, however, may be of interest. The sire of both of the affected animals was a pure-bred, registered Friesland bull, "Kamnatie Charles," who was used, apparently very successfully, on Weltevrede and the adjoining farms for a number of years, during which time he served some of his own daughters. The affected heifers were out of well-bred Friesland cows, which were themselves daughters of "Kamnatie Charles." A number of other calves bred from this bull, and kept under similar conditions, were perfectly normal.

Up to the time of writing this article, investigations had not yet been completed, particularly as regards the examination of the living ancestors of the affected heifers, and the study of the ancestry of the bull in question, who may possibly possess a recessive character for this condition, and thus be responsible for the hereditary transmission of the disease.

Summary.

(1) Two cases of congenital porphyrinuria in Friesland heifers in the Ladismith district, C.P., are described.

(2) The clinical symptoms in these cases are compared with those seen in the Swaziland and other cases.

(3) The evidence suggests hereditary transmission of the condition through a pure-bred Friesland bull.

(4) These are the first cases to be described among cattle in the Cape Province.

Retirement of Col. Irvine-Smith, C.B.E., V D., M.R.C.V.S., F.R.S.I., J.P.

Introduction. — Col. James Irvine-Smith, the Director of the Abattoir, Live Stock Markets and of Veterinary Services of the City of Johannesburg, retired on 4th August ,1939, after a distinguished career. As a pioneer of veterinary science in this country he succeeded in advancing the status of the profession by his administrative ability and profound knowledge of economics.

Irvine-Smith qualified with first-class honours at the Glasgow Veterinary College in June, 1898, after a most successful course, being awarded a gold medal for the best professional examination, several silver medals and various first-class certificates.

Military Experience. — After serving as assistant to Sir Henry Simpson, Veterinary Surgeon to Her late Majesty the Queen at Windsor, he proceeded to South Africa early in 1900 as a civil veterinary surgeon in the Imperial Army. It was not long before his unusual ability became apparent and he was selected first as O.C. Advanced Base Veterinary Hospital, De Aar, and then as O.C. A2 Indian Field Veterinary Hospital, both of which posts were field officers' commands and had been held by majors.

At the beginning of 1902 he handed over to Major Richardson and transferred to the South African Constabulary as Assistant Principal Veterinary Officer with rank of captain and frequently acted as P.V.O. In point of fact he was responsible for the organisation of the first civil veterinary department in the Transvaal.

At the close of the Second Anglo-Boer War but little was known regarding the nature and extent of communicable cattle diseases and since the Repatriation Department was engaged in restocking farms, there were many cases of mortality, the causes of which were obscure. Such an outbreak occurred at Komatipoort in August, 1902, and was shown upon investigation by Capt. Irvine-Smith to be East Coast Fever.[1]

At the end of June, 1906, Irvine-Smith left the S.A.C. and was attached to the Civil Veterinary Department for a few months and in May, 1907, was appointed Municipal Veterinary Surgeon of Johannesburg.

During this period he had interested himself in the volunteer movement and in January, 1906, was appointed P.V.O. Transvaal Volunteers. He organised the Transvaal Veterinary Corps [see History of Transvaal Volunteers (1902 – 1913), *Jl. S.A.V.M.A.*, IV (1), 1933], was promoted to Lieut.-

[1] Soon after this Transvaal Ordinance No. 17 of 1902, the first Diseases of Stock law under Crown Colony government, was promulgated.

Colonel in May, 1909, and transferred to the Reserve of Officers in June, 1914.

On the outbreak of the Great War in August, 1914, nothing had been done towards organising a Veterinary Service for the Union Defence Forces. There was indeed one veterinary officer, Major Collyer, M.R.C.V.S., attached to the Permanent Force (consisting of five S.A. Mounted Riflemen regiments), but he was already doing the work of several men.

The Defence authorities therefore recalled Irvine-Smith and entrusted him with the creation of the S.A. Veterinary Corps. The Director of Veterinary Services, Union Defence Forces, made speedy arrangements for veterinary organisation during the Rebellion and German South-West Africa Campaign and soon established an efficient service. In March, 1916, he was compelled to return to his civil duties as Director of the Abattoir and Live Stock Markets at Johannesburg.

For his services Irvine-Smith was mentioned in despatches and in 1918 was awarded the military C.B.E. Subsequently he received the V.D. (Colonial Auxiliary Forces Officers' Decoration).

Professional Interest. — Irvine-Smith is characterised by his enthusiasm for the profession which has enabled him to succeed in life. At the end of 1902 the organisation of a professional society was suggested and on 16th February, 1903, the inaugural meeting was held at Long's Hotel, Johannesburg, Capt. Irvine-Smith (then Acting P.V.O., Civil Veterinary Department) being elected the first President [see History of Transvaal Veterinary Medical Association (1903 – 1920), *Jl. S.A.V.M.A.*, II (1), 1931]. He at once interested himself in the question of legal protection for the profession and a bill acceptable to the Administration was drafted. Unfortunately opposition arose within the Association with the result that legal recognition was never attained.[2]

In 1920 the S.A. Veterinary Medical Association was formed and from 1921 to 1924 Irvine-Smith was President. In 1924 he was elected Honorary Life Vice-President in recognition of his labours for the profession.

Municipal Veterinary Organisation. — As mentioned above, Irvine-Smith entered Municipal service in May, 1907, when arrangements for stock sales and slaughter were exceedingly primitive. At this time the post of veterinarian was subordinate to the M.O.H.

The subject of our sketch soon realised that there were great possibilities for an energetic man and that if business methods were introduced, he would receive much support from the Council.

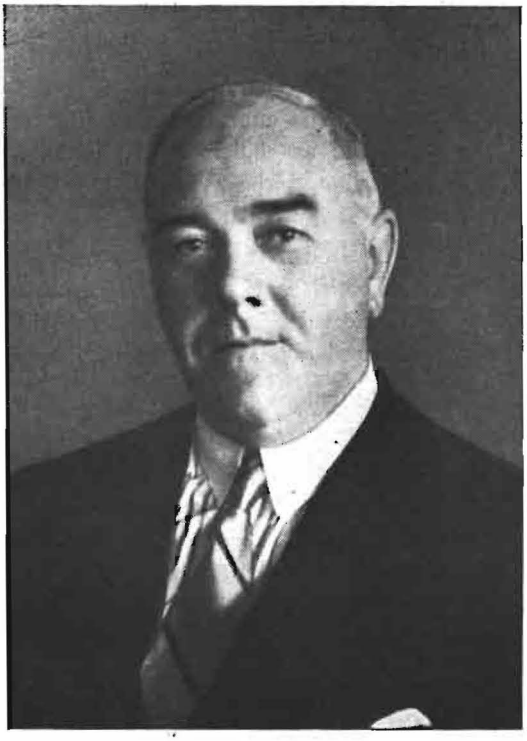
He commenced by establishing a horse and mule shoe factory, encouraged an all-white labour policy and indentured apprentices to the farriery trade.

Progress was then made with the erection of the Abattoir and Live

[2] Until the passage of Act No. 16 of 1933, Natal was the sole Province where the Profession was legally protected.

Stock Markets (recommended as early as 1902) which, along with a quarantine market, were opened in 1910.[3] In 1911 the By-Product Plant was installed and in 1919 the Refrigeration Plant was completed. In 1930 laboratories were opened for the biological examination of milk and systematic dairy inspection undertaken. Numerous other routine services are also performed.

By his vision and tenacity Irvine-Smith succeeded in gaining special powers for veterinarians employed in abattoirs. These powers were specially defined in the Transvaal Local Government Ordinance of 1912 and later



COLONEL JAS. IRVINE-SMITH.
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were incorporated in the Union Public Health Act of 1936. Irvine-Smith is an approved veterinary surgeon under both these measures.

On account of the expansion of the public health activities for which the Director of the Abattoir became responsible, additions to the professional staff were made as follows:– Assistant Director (Mr. A. C. Kirkpatrick, M.R.C.V.S.) in 1920; Dr. G. Martinaglia in 1930; Mr. M. C. Robinson, B.V.Sc., in 1935.

[3] All slaughter "poles" were thereupon closed.

Abundant information in the form of statistics is available in the excellent annual reports compiled in respect of the Department for which Colonel Irvine-Smith is responsible. It is sufficient to state that more animals are slaughtered per annum at the Johannesburg City Abattoir than at any city in Great Britain.

Membership of Public Bodies. — On account of his experience in handling food supplies and in public health generally Irvine-Smith has served on three Government Railways and Harbours Live Stock Commissions, namely in 1909, 1916 and 1923. As a result of his minority report in 1923, transit cooling stores have been erected at Cape Town and Durban. He was chairman of the Egg Export Commission of 1925 – 1926 and was a member of the Agricultural Advisory Board to the Minister of Agriculture in 1920.

Irvine-Smith has served on the executive committee of the Witwatersrand Agricultural Society, acted as chairman of the finance committee of the Transvaal Agricultural Union, and served on the executive of the S.A. Agricultural Union.

Educationally he is recognised as an authority on public health for he has acted as an examiner to both the University of the Witwatersrand and to the Royal Sanitary Institute. In 1924 the latter body awarded him a Fellowship " for noteworthy and outstanding sanitary work."

Appreciation. — It is not surprising that Colonel Irvine-Smith's Department receives special mention in the book *City Government: The Johannesburg Experiment,* by John P. R. Maud, who considers Irvine-Smith's policy, i.e. to provide essential public services at cost or as near cost as possible, to be the right one.

In view of the striking developments Irvine-Smith has brought about it can be well understood that on six occasions he has received votes of appreciation from the Council of the City of Johannesburg. On three occasions he has obtained the highest mark of esteem, namely an appreciation inscribed in vellum and under the seal of the Council.

In 1935 Colonel Irvine-Smith was awarded the King's Jubilee Medal for meritorious service to the City of Johannesburg.

Arriving in South Africa thirty-nine years ago as an unknown civil veterinary surgeon, Irvine-Smith has by sheer grit and determination created the highest paid veterinary post in Africa. Possessing the qualities of courage, confidence, and creative ability he has left his mark on every phase of professional activity in this country and his life story may well serve as an inspiration to young colleagues for many years to come.

Still at the height of his mental powers and as energetic as ever, it is the sincere wish of the members of the profession that he be spared for many more years of usefulness.

H. H. C.

Suggestions for the Eradication of *Cysticercosis-taeniasis* *

By N. F. VILJOEN, Bloemfontein.

Before proceeding to suggestions regarding the eradication of the cysticercosis-tæniasis, common to two of our most generally consumed meat animals and to man, it may be advisable to indicate the extent of this infection, the serious import of the disease in the field of hygiene, and the economic losses this country sustains annually through its toll.

The incidence of *Cysticercus cellulosæ* in pigs, as evidenced by statistics obtained from some 60 abattoirs in South Africa, varies from 0·5 per cent. to as high as 25 per cent. From no less than 24 abattoirs were percentages in excess of 5 returned. In conducting research into this question a couple of years ago the writer was enabled to draw up a map of the Union showing the incidence of pig measles at approximately 60 abattoirs. A definite "black" zone could be demarcated, ranging from Vryburg and Mafeking in the North-Western Cape, through the whole of the Central Transvaal, namely Lichtenburg, Potchefstroom, Rustenburg, Pretoria, Witbank to Middelburg. A similar distinct "black" zone was traceable on the map of the Eastern Free State, viz., those districts bordering on, or close to, Basutoland. In this area the highest percentages of all were obtained, namely from Wepener, Clocolan, Senekal, Ficksburg and Bethlehem. These facts, of course, are significant when we consider that pigs slaughtered at the above-named abattoirs are drawn mainly from areas adjacent to or within Native Territories. Some of the official figures are really quite startling when we compare, for example, the incidence of infection at Senekal (25·07%), Ficksburg (25·0%), Lichtenburg (19·48%), Potchefstroom (15·3%) and Bethlehem (11·49%) with official statistics supplied by nearly all other civilised countries, the highest of which came from Madagascar, where, according to Poisson (1926, 1929) and Buck, Lamberton and Randriambeloma (1935) the incidence of *Cysticercus cellulosæ* in pigs varied between about 5% at Diego-Suarez and 12 to 20% at Antsirabe.

In no other country in the world is the incidence of *C. cellulosæ* in pigs so high as in South Africa. The official statistics for Kenya, for example, give the incidence of *C. cellulosæ* as being negligible, but that of *C. bovis* as very high. The incidence of *Cysticercus bovis* in South Africa is considerably lower than that of *C. cellulosæ*. It occurs in from a fraction of 1% to about 7 or 8% of slaughtered carcases at some 60 Union abattoirs. Here again it is interesting to note that definite "black" zones occur in, or close

* Read at the 33rd General Meeting of the S.A.V.M.A. at Onderstepoort, October, 1938.

to, Native areas. For example, at Durban Mr. Dykins has found that the great bulk of infected carcases come from Native areas such as Swaziland, North Zululand, Richmond and Ixopo. In Natal the highest official percentages were obtained from the abattoirs at Dundee, Greytown, Ladysmith, Pietermaritzburg and Vryheid — all places which draw a considerable amount of slaughter stock from Zulu areas. In the Cape the highest percentages came from the eastern abattoirs at Port Elizabeth, Fort Beaufort, King-williamstown and East London, close to the Transkei Territories. In the Transvaal those abattoirs (e.g. Barberton) close to the Swaziland border gave the highest percentages.

The incidence of *C. bovis* in this country compares favourably with the official returns from some of our neighbouring territories, such as Kenya (about 17%), Tanganyika (from 5 to 18%), Uganda (from 15 to 25%), Abyssinia (approximately 100%), Senegal (10%), French Guinea (50%) and Belgian Congo (about 9%). It must, however, be pointed out that most of the above-mentioned are Native territories and that none exports beef abroad. Compared with all European countries, Australia, New Zealand, the United States, Canada and the Argentine, the incidence of *C. bovis* in South Africa is a sad business. In a few contries in Europe, e.g. Germany, Holland, Denmark, Bulgaria, Switzerland and Czechoslovakia, the incidence of beef measles may reach up to about 1%. In Australia it occurs very sporadically, in New Zealand it is quite unknown, and in the three named American countries it is very rare.

The actual incidence of tapeworm infection in South Africa is not known, but estimates of the frequency of its occurrence may be gauged from the extremely limited literature on the subject and from the few records of statistics which have been compiled by medical authorities from time to time. Compared, however, with figures from certain African Native and Oriental territories, we derive a small amount of insecure comfort from the fact that in South Africa the proportion of tænia carriers among Natives is not 100%, as was, until recently, the case in Abyssinia. It has been written that the native Abyssinian regards his tapeworm as one of his most cherished possessions, and as long as he carries his guest, he believes that he will remain free from most of the internal disorders! That *Tænia saginata* is quite a common parasite in Syria and Lebanon is gleaned from the statistics supplied by Yenikomshian and Berberian (1934), who state that in places such as Beirût the incidence of infection was found to be as high as 10 to 15%. Daubney and Carman (1928) examined the stools of the inmates of a Government reformatory in the Kenya Highlands and found that 50% of these were infested with *T. saginata*. In Tanganyika, in 1933, it was found that infestation with tapeworm was even higher at some places than in Kenya. For instance, at Moshi Dr. R. C. Spiers found 313 infested stools among 552 examinations from prisoners, sanitary porters, school boys, and other native children. It is a great pity that it has not been found possible to undertake regular surveys of the incidence of tæniasis in South

Africa. Annie Porter (1918) detected the ova of tapeworms in the excreta of 37 out of 375 native patients and in one out of 60 Europeans in the Johannesburg General Hospital. Porter emphasised that none of these cases had been admitted to hospital for worms and several were surgical cases. Of this number, 26 natives harboured *T. saginata* and 11 *T. solium.* Furthermore, in 104 autopsies on native mine workers, Porter found tapeworm in the intestines in 20 instances — 12 *T. saginata* and 8 *T. solium.* Watkins-Pitchford (1923) estimated the incidence of tapeworm infestation among South African natives to be from 10 to 19%. Dr. W. O. Fischer found at the City Deep Central Native Hospital between the years 1928-33, inclusive, that in autopsies 75% of the Swazis, 25% of the Bacas, 22·2% of the Hlubis and 5·9% of the Zulus carried *T. saginata,* and in addition 11·1% of the Hlubis carried *T. solium.* Dr. Viviers, District Surgeon, Vereeniging, informed the author that he was of opinion that at least 25% of the natives in that district carried *T. solium.* Cawston (1935) related that in about 1928, prior to the erection of a proper abattoir at Potchefstroom, it was found that some 30% of European school children attending the clinic of the Potchefstroom Health Committee harboured tapeworm. The Chief Medical Inspector of Schools in the Transvaal informed the writer that in rural schools in the Transvaal, and especially in the so-called Bushveld areas (e.g., Marico, Zwartruggens, Rustenburg, Waterberg, Lydenburg, etc.) infestation with tapeworm was fairly high. Roughly stated, the incidence of tapeworm infestation in European school children in the Transvaal ranges from a fraction of 1% to as high as 20 to 25%.

Necessity for Eradication of Cysticercosis-Tæniasis.

The two factors which demand the eradication of cysticercosis and its correlative tæniasis are economy and hygiene. When we consider both these important factors, it should be agreed that a strong plea for greater recognition from the scientific and official points of view is not unreasonable. Cysticercosis is a costly scourge to the stock-raising community in South Africa, and its correlative tæniasis is a serious and disgusting affliction of the population, European as well as Native. In this country it is essential that we should take the economic factor into serious consideration. The fact that we are trying to compete with rivals on overseas markets, where the incidence of *C. bovis* is considerably lower than in South Africa, has repeatedly been pointed out by abattoir veterinarians, especially by Colonel Irvine-Smith. It has been pointed out that our chilled beef industry would receive a nasty jar if measles were found on re-inspection of the carcases in England. It must also be remembered that chilling of beef and of pork, even for 40 days, does not kill the measle.

Roughly estimated, the toll of measles at our abattoirs costs the South African stock producer or the butcher some £70,000 per annum. Annually about 7,000 pigs and the same number of bovines are found to be measly at our abattoirs.

It must, however, always be remembered that the object of a plan for the eradication of measles is, of course, to eradicate tapeworm and safeguard the meat consumer from infection. From the veterinary and medical point of view, therefore, the hygienic necessity for the eradication is obvious and perhaps an important reason why the State might be called upon to expend a sum in excess of the £70,000 loss to the producers. Although the fear of infestation may be obvious to all, the effect of tæniasis on the human host is not so well known — even to medical men and to many veterinarians. In the healthy adult an ordinary single infection with either *Tænia solium* or *T. saginata* may not have very severe clinical effects on the patient. Both species of human tapeworm, with which we are concerned, may occasionally show amazing tenacity of life. They may grow to a length of 20 to 30 feet and may live in the host from 12 to 20 years or even longer. Franke (1931) recorded a case of *T. saginata* in which the patient required six vermicides before the tapeworm was eventually expelled. In general the following derangements in man may be caused by tænia infestation:– loss of nourishment is usually compensated by the eating of larger amounts of food, owing to the abnormal appetite the patient develops. Frequently diarrhœa, followed by constipation may be observed; flatulence, tympanites, spasmodic colicky pains and sometimes a sensation of "pressure" in the abdominal region occur. Nervous derangements may also follow and in weak subjects anæmia. Among the sequelæ which frequently follow tapeworm infestation, appendicitis is the most common. Cases of acute appendicitis, in which on operation the cause was found to be the presence of proglottides of tænia, or portions of the strobilæ in the appendix, were recorded by Braun-Seifert (1923), Altenkamp (1935), Pytel (1935) and many others. Farzane and Ibragimov (1935) found that ileus of the intestine had been caused by a conglomeration of *T. solium* segments. Burnet (1919) placed on record three cases of chorea which had their origin in the presence of tapeworm infestation and were cured when the worm was expelled. In South Africa very interesting work on the subject of psychosis due to tapeworm infection has been performed by Dr. A. S. van Coller, who found that in 450 cases of psychosis the presence of tapeworm infestation acted as a precipitating factor and most of those cases were cured in from 3 to 12 months after expulsion of the worm. Similarly Dr. Egerton Brown of Pietermaritzburg found that in 207 positive cases of tapeworm infestation in native patients, the absorption of a toxin secreted by the living tænia was the cause of acute mania, and of this number 139 recovered after expulsion of the worm. Tæniæ have been known to migrate from the intestinal tract upwards into the pharynx and thence downwards into the trachea, causing distressing dyspnoea and death. Such a case was recorded by Shahan (1932). Finally, it should be remembered that, especially in the case of *Tænia solium* infection, man may infect himself or someone else with cysticerci. Thus it may be possible for man to play the rôle usually reserved for the pig. The carrier of

T. solium is thus a particularly dangerous individual. He may, himself, swallow the ova excreted by him, or he may infect an entire household with which he comes in contact, by conveying the ova on his hands to the food of those unfortunate individuals. In South Africa, where in almost 100% of households our food is prepared by native servants, we are particularly liable to infestation with pig measles, a large percentage of our natives being potential tapeworm carriers. The habit of using human excreta as fertilizer for vegetable gardens is not only disgusting, but dangerous. Such vegetables as lettuce, parsley, celery, watercress, etc., which are generally eaten in a raw state are positively dangerous. Infestation of the human subject with cysticerci follows very much in the same way as in the pig. Eggs are ingested and young larvæ are conveyed in the blood stream and lodge between muscle fibres where they develop into cysticerci. In man the brain is said to be a predilection site of these parasites and also the eyeball and its annexes. MacArthur (1934), however, has pointed out that it may be possible that, as in the pig, the general musculature may harbour the measles as frequently as the brain, but their presence may not be felt by the patient and they may thus not be detected. Cysticerci in the brain are of greater importance, since, after they die off and degeneration sets in, they act as foreign bodies, with resultant pathological changes and effects. The commonest sequel of this infection in man is epilepsy, which generally arises some years after initial infection. Many such cases of epilepsy due to cerebral cysticercosis have been recorded in British troops who have served in India, and South African records mention quite a number of cases in natives and not a few in Europeans as well. Treatment is hopeless and death invariably follows, even if only after a number of years. It will, therefore be agreed that tapeworm or cysticercosis disease is a serious, disgusting and even pitiable infestation and certainly warrants a campaign of drastic eradication by the united efforts of the Departments of Public Health and Agriculture. A successful campaign against the disease could be embarked upon on the following lines:–

(a) *Co-operation between the Veterinary and Medical Professions.*

Such co-operation need not lead to encroachment by the one profession on the province of the other. In other words, the eradication of the cysticercus in our food animals is the work of the veterinarian, and the eradication of the tænia is that of the physician.

The medical profession should most certainly embark, or assist in a campaign against the tænia. They could seek our aid in this campaign and advise us, for example, of patients treated by them, where they come from, and the species of tapeworm expelled. That would be a guide to us, for, as has been said, eradication of the cysticercus should be in the hands of the veterinarian, which brings us to our second point.

(b) *Veterinary Control at all Abattoirs serving Towns with Populations of 7,500 or more Europeans.*

From the point of view of making propaganda for the profession, this point should meet with universal approval from all veterinarians. However, that is not the motive of this paper. We veterinarians are certainly the only scientists, other than zoologists, who are competent to diagnose cysticercosis. But, since cysticercosis is not the only pathological condition we have to deal with in meat inspection, although in South Africa it may be considered the most important, the zoologist is not qualified, nor does he pretend to be qualified, to examine meat for disease. At the present time there are only six municipalities in the Union which employ full-time veterinary officers. At all other abattoirs, including those of some of our larger cities, the superintendent in charge is a trained inspector — in most cases a splendid practical man — but, according to law he is not allowed to condemn meat and must merely detain the carcase until his Medical Officer of Health confirms his diagnosis.

Our meat regulations lay down that any *medical practitioner* or an *approved* veterinary surgeon may condemn diseased meat. What a paradoxical state of affairs this is. It would rather appear that the wording of the clause should be exactly reversed and that any qualified veterinary surgeon or an *approved* medical practitioner should have the right to condemn diseased meat. In most cases the general medical practitioner knows considerably less about diseased meat than the qualified meat inspector who has had to call him in, and yet, in towns where no full-time veterinary officer or medical officer of health is employed medical men actually visit abattoirs when called upon, and condemn meat. It is the Government's bounden duty to protect the urban meat consumer, and, therefore, it should be compulsory for all urban areas with European populations of 7,500 to employ municipal veterinary officers, who could, at the same time, take control of the dairies and town animal transport departments as well. At present there may not be sufficient veterinarians in the country to carry out this work in all these towns, without depleting the Department of Agriculture's personnel, but if attractive salaries were offered, it is almost certain that many more young men would take to the profession. This country is very far behind nearly all countries in Europe and the Americas in regard to veterinary control of abattoirs. Ours is a just claim, which, unfortunately, we allowed our medical confrères to usurp at the time when we, as a profession, were too busily engaged on other State duties, and we simply did not have the veterinarians who could be spared for, or showed the natural inclination for, abattoir duty. We should now ask that this position be remedied.

(c) *As regards the smaller urban areas,* namely those municipalities with European populations of less than 7,500, it should be made compulsory that all animals intended for human consumption be slaughtered at an

approved abattoir, and that meat be inspected by a qualified meat inspector. These inspectors should hold the Meat and Other Foods Certificate of the Royal Sanitary Institute or other approved body. Very unfair competition exists throughout the country between buyers of stock in small urban areas or villages, where no meat inspection whatever exists, and the larger and more enlightened townships close by, where thorough modern inspection is practised. An example of such an anomaly recently came to the writer's notice. At Barberton thorough inspection exists, but in the surrounding rural centres such as Sabie, Noordkaap, Sheba, Eureka, Louwscreek, Hectorspruit, Komatipoort, Kaapsche Hoop and Nelshoogte, animals are slaughtered at slaughter poles, where no inspections exist. The result is that unless the unfortunate Barberton butchers pay the full prices to farmers for condemned cattle, they are excluded from supplies, and the farmers supply the butchers of those mentioned smaller surrounding settlements, who slaughter and sell measly meat without restraint.

A difficult but nevertheless practicable solution of this anomalous state of affairs would be for the Department of Public Health to appoint qualified meat inspectors, who could be stationed at the central urban area and from there could follow daily itineraries and inspect meat at the slaughter poles of the surrounding villages. At no village in South Africa are stock slaughtered daily, and the inspector could, with very little inconvenience, arrange the various slaughter days and times of inspection in his area. The salaries of such inspectors could be subsidised by Government and the small townships in the area could each pay a *pro rata* share to make up the rest; or the salaries could be entirely paid by the Department of Public Health. The main idea behind such a scheme should be to protect all buyers of meat from contracting tapeworm and to ensure that no unfair competition should exist between slaughterers of uninspected carcases and those butchers who have to agree to a careful inspection of their meat carcases.

(d) *A More Thorough System of Meat Inspection.*

Unfortunately, time and space do not permit a recapitulation in this paper of the so-called predilection sites, where measles are most commonly found on meat inspection.

It is doubtful whether the technique followed in South Africa can be improved in respect of the inspection of pig carcases for *C. cellulosæ*, but for the inspection of beef carcases for *C. bovis* the writer recommends the following technique:–

1. Two long and parallel incisions into the masseters on both sides of the face, in an upward direction, to completely sever the parotid gland below the ear.

2. Two long incisions into the pterygoids on each side.

3. Numerous longitudinal incisions into the muscles of attachment of the tongue.

4. Careful manual palpation of the whole of the heart; complete halving of the left centricle; careful inspection of the myocardium.

5. Careful manual examination of the oesophagus.

6. A complete incision into the triceps brachii and the deltoideus on each side.

7. One incision into the psoas muscles on each side.

8. Usual routine inspection of the viscera, without further incisions.

9. A transverse incision into the hump after the carcase has been cleft.

10. A deep incision into the adductor about an inch below and parallel to the pelvic symphysis.

11. In the event of measles being found in any of the above-named incisions, then the secondary incisions laid down by the Public Health Act must be made.

It must now be explained that South African meat inspection regulations do not provide for incisions into the hump and into the hind limbs of bovine carcases, as a routine practice. Our research at Bloemfontein has shown that next to the masseters and behind the elbow, the hind limbs must very definitely be considered as a most important predilection site for measles, and in the hump we also very frequently found many cysts in those carcases which were totally condemned and which we were enabled to dissect minutely, in order to study carefully the commonest predilection sites. The hindquarters of beef cannot be incised at random, and experimentation at Bloemfontein showed that a deep transverse incision into the adductor muscle on the medial aspect of the thigh, just below and parallel to the pelvic symphysis had very little damaging effect on the appearance of the carcase and in this muscle we found many measles, which would have escaped detection on ordinary inspection. Meat inspectors should remember that permitted incisions should be made deeply and as long as possible. In fact, the present writer has observed at many of our larger abattoirs and nearly all smaller ones he has visited, that inspectors appear to make their incisions far too ineffectively.

(e) *The Abolition of all Insurance Schemes which include Indemnification against Measles.*

At a number of the larger abattoirs in the Union bovine cysticercosis is included in insurance schemes. Premiums are imposed on all animals to be slaughtered and the farmer or butcher is quite indifferent as to whether or not his ox is condemned. The direct result of these insurance schemes is that the farmer does nothing to safeguard his cattle from infection. What the farmer does not realize, however, is that it is he who after all pays the insurance premium, and that abattoirs are not philanthropic institutions which willingly, out of sympathy for the unfortunate farmer, refund the price of the ox lost to him, without making him pay extra for the many

uninfected oxen which have passed inspection. It is questionable whether such insurance schemes serve any useful purpose, and at several of our larger Union abattoirs (Bloemfontein, Port Elizabeth and Cape Town) they are totally discouraged. At others, e.g. Durban, special premiums are imposed for stock from "clean" areas and heavier premiums on stock from "black" areas.

In considering this question from all points of view, the conclusion come to is that insurance schemes which include indemnification against measles are definitely not in the interest of the country, and we should press for legislation forbidding the inclusion of measles disease in abattoir insurance schemes. Measles disease is, and should in every case be considered by buyers themselves, a latent defect, if found in stock slaughtered within a reasonable time after purchase. Measles are generally observed and visible to the naked eye after six weeks of infestation, and thus if found in cattle slaughtered within six weeks of purchase, give the butcher clear proof that the ox or the pig was infested prior to purchase. Butchers could assist in the eradication of measles if they would all decline to pay farmers for such infested purchases.

(f) *Education and Enlightenment of the Farming Community, Rural Dwellers and Natives.*

It is astonishing how little the general public knows of the life-history of the parasite. Our campaign must, therefore, be directed at the rural source of the disease. All three Departments interested, namely, Agriculture, Native Affairs and Public Health, can collectively assist in the eradication of the parasite. The Department of Agriculture can stimulate interest in the scourge by encouraging or instructing its veterinary officers, stock inspectors and extension officers to lecture to groups of farmers on elementary farm hygiene and the life-history of *Tænia-Cysticercus*. Similarly the officers of the Public Health and Native Affairs Departments can disseminate knowledge among the natives. Pigs should be kept in sties and on no account be allowed to wander about the farm-yard. Suitable latrines or privies should be constructed on all farms for the use of Europeans and separate latrines for natives. What would appear to be quite a practical suggestion is the fencing off of strips of veld, within which natives may defæcate. Such narrow fenced strips, with narrow inlets so that cattle cannot enter them, can be provided on various parts of the farm, especially close to such parts where the farm labour is most frequently required, and also in grazing camps for the herd-boys, etc. Such fenced surface latrines may not, however, overcome the possible spread of tænia eggs by such agencies as water, insects, dungbeetles, blowflies, etc. The provision, therefore, of similarly fenced off trench latrines and the enforcement of the immediate covering up of the deposited excreta by the native may thus be more effective. Covered bucket latrines are, undoubtedly, the most effective. There is little doubt that good work, through enlightenment, may be

expected from native chiefs, who are ever ready to co-operate in campaigns concerning the health of their livestock. This may sound idealistic, but many of our present native chiefs are quite intelligent and if instructed to do so, they may be expected to enforce strict hygienic sanitary laws among their people. The trouble is that, up to the present, they have not been requested to embark on such a campaign, nor has the necessity for the enforcement of tribal latrines been brought home to them. This is an experiment well worthy of a trial.

On no account should slaughter bovines be grazed on lands fertilized by human extreta, or on sewage farms, and the use of fodder grown on such lands must be entirely discouraged.

(g) *The Prohibition of Sale of Pig Carcases slaughtered on Farms and brought to Abattoirs for Inspection.*

Some municipalities, e.g. Worcester, Mossel Bay, Burghersdorp, Clocoland, and, I am sorry to say, also Bloemfontein, permit the slaughter of pigs on farms and the sale of the carcases on the local market. The carcases must be brought to the abattoir for inspection, with the pluck attached — stomach, intestines and other viscera are not produced. In fairness to the Bloemfontein City Fathers, however, I must mention that this arrangement was authorized by the Free State Administration many years ago, and despite our efforts to have it rescinded, it is still in force. The results of this practice are clearly reflected in our observations at Bloemfontein. The average for the last few years has been an infestation with measles of between 2 and 3 per cent. of pigs slaughtered at the abattoir, and yet, out of many hundreds of pigs slaughtered on the farms and brought to this abattoir for inspection, in four years we have only found two pigs measly. Pigs are more commonly very heavily infested with measles, and obviously farmers do not bring to the abattoirs pigs they notice measly on inspection. They have a very ready sale for this measly pork to their unfortunate natives, and thus they disseminate the disease. Here is an anomaly which can be immediately rectified by legislation. If the serious hygienic and economic importance of the disease were to be brought home to them, even the most ardent, and at times almost fanatical legislators, who vigilantly safeguard the interests of the farmer, would vote in favour of the compulsory slaughter of all pigs intended for urban consumption, at urban abattoirs.

(h) *The Offer of Rewards for Production of Tænia Heads, after the Carriers have subjected themselves to Free Medical Treatment.*

The enlightenment of our farming and native populations should be followed by the provision of free medical treatment. Liquid extract of male fern, or whatever vermifuge othe Public Health Department may recommend, should be available for all tænia carriers, whether on farms, native reserves, or in towns. Magistrates, District Surgeons, Justices of the Peace in rural areas, and native chiefs should be provided with quantities

of the drugs required, and careful directions for use given to those who, whilst having to dispense the drugs, may have no knowledge of therapeutics. Carriers should then, by the offer of rewards for the production of portions of evacuated tæniæ with heads, be encouraged to undergo this free treatment. It is possible that such a campaign may cost a considerable amount of money, but in consideration of public health and the loss of our local meat industry, as well as to our potential chilled beef overseas market, it may well warrant the offer of small rewards to all those, European or Native, who, after having been treated, produce either the complete tænia or portions including the head. Perhaps free treatment, plus 2/6d. for each head might be a big incentive to our poorer Europeans and Natives to rid themselves of their health-destroying guests. Rewards have been offered by the Government for the production of evidence of destruction of other vermin, e.g. the brushes of jackals, and it is quite possible that the economic loss from these marauders has been less than that caused by the tapeworm carrier. This experiment has been followed with great success both in Australia and in Germany.

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Jl. S.A.V.M.A.,
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1939.

Observations on Torsio Uteri in Cattle.

By G. G. KIND, Johannesburg.

The ideas obtained by me from teachers and textbooks on this condition gradually underwent a change through years of practice and it appears to me worth while to put these changed ideas on record.

Diagnosis.

The description in the textbooks of the symptoms is, in my opinion, not quite correct and this may make it difficult for an inexperienced beginner to make a diagnosis except in extreme cases. Examining through the vagina one may erroneously expect to find an even ring of folds converging into something like a funnel. Actually the folds formed by the wall are not pronounced, especially if the seat of torsion is oral to the cervix, as is usually the case. The narrowing of the passage is rather abrupt and into this narrow passage one feels one or two thick cords stretching transversely and obliquely towards the uterus. In slight cases one feels only one cord situated dorsally, but in more severe cases there is another situated ventrally. These cords are formed by the stretched *ligamenta lata*. Regarding rectal examination, the textbooks give the spiral cord of the twisted uterus as typical for diagnosis. To me the spirals never appeared to be so striking. But the stretched *ligamenta lata* are a most striking symptom as they appear to be wound round a thick tough cord which is the twisted part of the uterus or the vagina.

Therapy.

Some textbooks suggest that in most cases the fœtus dies very shortly after the occurrence of torsion and that the prognosis is unfavourable if the condition has existed for more than 24 hours. In my opinion this is not necessarily the case in torsions of 90° to 270°. Fatal results may often be due to an unsuitable technique.

It is my intention to criticize the various methods described in textbooks from the experienced practitioner's point of view and to describe a method which I have not come across in the literature accessible to me and which I have found more successful than the others.

In cases where it is possible to introduce the hand into the uterus it has been suggested:

1. To remove the calf without rectifying the position of the uterus. This may be possible, but it should never be attempted as the parts of the uterus weakened by mechanical strain and by impaired blood circulation are easily damaged.

2. To turn the calf and with this the uterus on the standing cow or on the cow suspended by the hind legs by swinging the calf with the

hand which is introduced into the uterus. Judging by my own efforts I doubt whether the average veterinarian has sufficient strength to accomplish this. Besides this, the forcible manipulations are again liable to damage the twisted parts and I cannot recommend this procedure.

3. To pull two of the calf's legs out of the vagina, tie them together, insert a stick between them and then turn the foetus. By this procedure the legs of the calf, which may still be alive, will surely be damaged. The twisted parts of the uterus or the vagina are also liable to suffer damage, and again my advice is not to try this method.

The following methods are recommended when it is impossible to introduce the hand into the uterus.

4. To push the fœtus and with it the uterus into the proper position from the outside of the abdominal wall. I never succeeded with this method.

5. To turn the uterus after laparotomy. This is in my opinion a waste of time and energy as cases which cannot be rectified by simpler methods are in any case hopeless.

6. To pull the cow down and then roll her over in the same direction in which the uterus is twisted, always keeping the hind quarters higher than the rest of the body. This treatment is based on the theory that the uterus is stationary, while one turns the body round this stationary organ. This method is assisted by fixing the calf and the uterus, either by holding the calf or by pressing the hand against the twisted region. It is also recommended to do the turning in jerks. This technique is the one generally practiced. Often one succeeds with this method, especially if the condition has only existed for a few hours. These cases are very rare and in my opinion the condition has usually existed for a much longer time than that given in the textbooks. Then one may have to keep on turning over and over for hours and in the end one may or may not succeed. There are obvious difficulties in this method:–

(a) By forcing the hand through or into the twisted region this may be damaged.

(b) It is rather difficult for a few men to give sufficiently strong jerks to such a heavy object as a cow to undo an old torsion.

(c) It is hardly conducive to good health to turn an animal over and over for hours.

I have practiced the following method for many years:– The cow is pulled to the ground. One attendant holds the head. Two attendants hold the tied-up fore-legs and two others the tied-up hind-legs. The four legs must never be tied together as pressure would be put on the abdomen and the uterus would be fixed instead of being allowed to swing freely. It is advisable to have the cow lying on a slope with the hindquarters higher. The cow is then pulled over in the opposite direction to that in which the torsion runs, in the following manner: The attendants stand dorsal to the cow, holding the legs with ropes, and they pull the cow over with as much

acceleration as possible so that she comes to rest with a sudden jerk when the legs strike the ground. The effect is that the body comes to a sudden stop while the loose internal organs swing further. The cow is then examined and, if the torsion has not completely disappeared, is rolled back to its original position. This rolling is done in a more jerky manner, but in such a way that the body does not come to a sudden stop. In this way one combines the new with the old method. The operation is repeated until all signs of torsion have disappeared. About this one has to make absolutely sure.

The advantages I have found in this method are the following:–

(a) It is easy to produce the jerk necessary to swing the uterus round.

(b) It does not require any effort on the part of the veterinarian.

(c) There is no damage done to the twisted part by forcing the hand through or into it.

I have had the experience that the reduced torsion appeared again as soon as the animal got up. I therefore do not now allow the animal to rise and I proceed immediately with the extraction of the calf, unless the cow is not ready to calve. (I had one case in which the torsion was rectified three days and another 2½ months before a normal calving; the latter torsion had occurred in an accident.) Even in animals which are ready to calve I have often found the cervix not properly dilated. I have left such cases to allow natural dilatation to occur, but have always found the cervix is the same condition even 12 hours afterwards. I therefore now immediately dilate the cervix. This is occasionally difficult in cases that are of old standing. Embryotomy should be resorted to if there is the slightest difficulty in removing the calf, to prevent damage to the portion weakened by torsion.

Lithopædion and Torsio Uteri.

Williams in his textbook on Obstetrics emphasises that sepsis usually sets in rapidly after torsion and that emphysematous calves frequently complicate this condition. Only occasionally have I found an emphysematous fœtus. Williams does not believe that an aseptic lithopædion may form and he says that "there is no evidence that such desiccation of the fœtus has occurred in uterine torsion." I have on two occasions found a partially dried-up fœtus in a uterus which was sealed by a torsion. In the first case a dealer sold to a dairyman a cow supposed to calve within about a month. The cow did not calve and nearly a year afterwards the dealer sued the dairyman for the purchase price and the dairyman sued the dealer for the cost of a year's feeding. By the time the case came before the court, over a year had elapsed. Two veterinarians gave evidence on behalf of the dairyman, that the cow was not in calf. I appeared for the dealer and diagnosed a torsion of the uterus containing a lithopædion. To settle the argument the cow was slaughtered in the presence of four veterinarians, and a fully grown sterile fœtus was found. At the torsion, which was

found immediately adjoining the orificium internum, where in my experience most torsions take place, the uterus formed a firm cord which was about 1½ inches thick and about 4 inches long. There were no signs of inflammation or adhesions. The dry fœtal membranes adhered to the calf. Cotyledons were not visible; the wall of the uterus was not thickened. Evidently the circulation from the ovarian vessels was functioning. According to the history the torsion must have existed for at least a year.

In the other case a farmer asked me to examine a cow which according to his dates should have calved about 6 months before. Rectal examination showed a torsion of the uterus and an apparently fully grown mummified calf. I recommended slaughter. I was unable to make a post-mortem examination.

These two cases show that even in severe torsion the ovarian blood-vessels can take over the circulation. I therefore maintain that in less severe cases this is still more likely. I have good reason to believe that slight torsions do take place, possibly weeks before calving, but would at the time cause only a slight colic which might scarcely be noticed. At the time of calving the fœtus cannot pass and then the veterinarian is called in. A slight torsion presents itself which, if it is recent, should very easily be corrected but which in old cases needs several efforts to rectify it. The calf may still be alive.

SUMMARY.

Typical symptoms in torsion of the uterus are not caused so much by the folds of the vaginal and uterine walls as by the stretched *ligamenta lata.*

The treatment recommended is rolling in the opposite direction to the torsion.

Two cases of formation of a lithopædion after torsion of the uterus are described.

It is maintained that a torsion of the uterus found at the time of calving may have existed for several weeks. Such a torsion may still be rectified and the cow delivered of its fœtus.

The 34th General Meeting of the S.A.V.M.A. will be held at Onderstepoort on the 25th, 26th and 27th October, 1939. The principal subject of discussion will be " The Production of Pure Milk in South Africa."

The Sir Arnold Theiler Memorial will be unveiled by General the Rt. Hon. J. C. Smuts at Onderstepoort on Thursday, 26th October.

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On the Question of the Intra-uterine Transmission of *Anaplasma Centrale.*

By KARL ENIGK, Berlin.*

The possibility of an intra-uterine transmission of blood parasites is of importance for the study of the epizootology and the control of diseases caused by these organisms. In the literature published in recent years many instances are quoted where fœtuses became infected with Piroplasma, Theileria and Trypanosoma. The transmission of anaplasmosis in this way has also been observed in some instances. Lignières (1914) concludes from an experiment on one calf that the Anaplasmæ do not enter the blood of the fœtus. Boynton (1929) observed acute *Anaplasma marginale* infection in two pregnant cows. In the calves that were born he failed to demonstrate *Anaplasma marginale* either by microscopical examination or by subinoculation of their blood into susceptible calves; these reacted later on testing their immunity. The same results were obtained in another calf born from a cow which reacted some time before parturition to *Anaplasma marginale.* He concludes from these observations that an intra-uterine transmission of *Anaplasma marginale* does not take place. Donatien and Lestoquard (1930) on the other hand are of the opinion that the transmission of Anaplasma in this way is possible. Donatien, Lestoquard and Kilcher-Maucourt (1934) carried out hysterectomy in two pregnant ewes which were heavily infected with *Anaplasma ovis* and *Babesiella ovis.* In the smears prepared from the heart blood of these fœtuses the mentioned parasites could be demonstrated microscopically. Dikmans (1931) states that in newly born animals, structures which resemble Anaplasma closely, have been observed frequently, but on closer examination were found to be breakdown products of the nucleus.

The writer had the opportunity to study this question in a calf born from a splenectomized cow which was a carrier of *Anaplasma centrale* and *Theileria mutans.* In the blood smears which were examined twice weekly these mentioned parasites could be demonstrated microscopically before and during pregnancy and also after parturition. The following is a brief summary of the observations made:–

The cow was born 27.7.1934 and splectomized by Dr. Quinlan of this Institute on the 15.10.1934. On the 30.1.1935 she received a subcutaneous injection of blood that contained *Anaplasma centrale, Theileria mutans* and *Piroplasma bigeminum.* The last infection died out after a few months and a second infective dose of blood containing the same parasites was injected

* Submitted while Dr. Enigk was visiting Onderstepoort.

into the animal on the 7.1.1936. During pregnancy *Anaplasma centrale* and *Theileria mutans* could be demonstrated microscopically, but the *Piroplasma bigeminum* infection had died out again. The calf was born on the 28.5.1938. In order to ascertain whether this calf harboured any blood parasites 50 cc. of blood was injected on the 21.9.1938 into two susceptible calves, and the calf in question in its turn received 50 cc. of blood from its mother on the same day. The two susceptible calves did not show any blood parasites in the blood smears which were examined daily up to the 12.11.1938, whereas in the experimental calf *Anaplasma centrale* appeared on the 19th day and *Theileria mutans* on the 23rd day. It must be concluded from this experiment that an intra-uterine infection did not occur.

Another observation was made on a practically full-term calf fœtus which was aborted by a cow that harboured a latent infection of *Anaplasma centrale* and *Theileria mutans.* In several blood smears prepared from the peripheral blood no blood parasites could be found.

The two described cases confirm the observations made over several years at Onderstepoort. In about 100 calves, which were reared under tick-free conditions, and which were born from cows which serve as vaccine reservoirs for *Piroplasma bigeminum, Anaplasma centrale* and *Theileria mutans,* an intra-uterine infection has not yet been encountered. All these calves were found to be susceptible to the three mentioned parasites.

Conclusion: In view of the above-mentioned observations on a calf, it is concluded that an intra-uterine infection of *Anaplasma* and *Theileria mutans* does not take place.

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Members will be interested to learn that Mr. D. T. Mitchell, M.R.C.V.S., having retired from the position of Director of Veterinary Services, Burma, has returned to this country and has settled at Maritzburg. In recognition of his services in Burma he had the O.B.E. conferred upon him in the last Birthday Honours List.

Botulism in Equines due to Contaminated Drinking Water and Hay.

By J. M. FOURIE, Hoopstad, and P. S. SNYMAN, Bloemfontein.

Introduction.

Botulism in equines is well known to-day, and may be regarded as a condition which occurs in accidental circumstances only, and which is usually associated with contaminated hay fed to stabled animals.

Theiler and Robinson (1928) described nine outbreaks that occurred in South Africa between the years 1913 and 1924. The ninth outbreak led to the discovery of the ætiology, when a partly decomposed rat was found in the hay rack. The organism which was isolated from the rat carcase was named *Cl. parabotulinum equi.*

Robinson (1930) subsequently proved that his *Cl. parabotulinum equi* must be considered as a variant of *Cl. botulinum C.*

Outbreak 1: Botulism in Horses and Mules.

During May of this year an outbreak of disease amongst equines was reported from a farm in the Boshof district. When the farm was visited five horses and mules were already dead and a horse and a mule still sick.

The sick mule was lying on its sternum with the head and neck flexed laterally against the thorax. When stimulated it was only able to raise the forequarters. The temperature of the animal after it had struggled vainly to rise was 101° F. The conjunctiva was red and congested.

The sick horse, apparently only in the initial stage of the disease, showed signs of weakness and marked stiffness. The animal walked with a gait somewhat similar to that of a horse with laminitis of all four feet.

Botulism was diagnosed and the source of the toxin traced to the presence of the carcase of a sheep in the water supply. The carcase was lying in a small compartment containing a ball-valve, which regulates the supply of water to the trough to which the horses and mules had access. The disease did not occur amongst other horses on the farm, which had no access to the water concerned. The animals concerned were stabled, but no carcase material could be found in the manger or hay. According to the owner the carcase could have been in the water for a fortnight.

Outbreak 2: Botulism in Donkeys.

On the farm Sydenham in the Bloemfontein district between the 4th and 11th June seven donkeys out of a span of sixteen succumbed to this condition. Briefly the symptoms revealed were as follows: the onset of the condition was noticed by inability to walk properly, and by twitching

of the muscles of the shoulder. The animals would then go down and be unable to rise.

In one case the animal was almost completely paralysed, assuming a lateral stretched out position. It could only remain in the normal lying position when assisted and supported by stones. When the head was brought to the natural position it would sway immediately to one side like the lid of a chest on its hinges.

In another case the donkey could support itself in the natural lying position when assisted to it, and could keep its head up and even turn it. In both cases the jaws could be pulled open by stretching the lips, when the tongue would protrude.

The donkeys had to subsist on the veld only, so that accidental poisoning did not occur from infected hay. Carcase material could not be found on the veld and may be ruled out, since no cases occurred in the cattle confined to the same camp. The only probable source of the toxin was put down to a small pool of stagnant water to which the donkeys only had access and were seen to drink from. The water in the pool was very muddy and had a very offensive smell. It is thought that dead frogs or some other aquatic animals may have been the source of the botulinus toxin.

Outbreak 3: Botulism in Horses.

Recently a farmer in the Brandfort district reported that his horses were dying from a disease unknown to him. When the farm was visited two horses were sick and three had died during the previous ten days. One of the affected horses was lying flat on the ground almost completely paralysed, being only able to raise the head and neck. The animal was fully conscious, fairly lively and reacted readily to external stimuli. Its temperature was 100° F. The other animal, apart from obvious stiffness in all four limbs, was grazing and revealed no other signs of disease. Although it was a hot day the temperature of this animal was only 96° F. Blood smears from the paralysed horse were negative.

From the owner it was ascertained that all the affected horses had been stabled at some time or other during the past ten days and not previously. The first horse which died was found dead in the stable in the morning entangled in the headstall riem. The other two horses which died showed signs of paralysis, mainly of the neck, and disinclination to feed; they rapidly lost condition and finally went down. Amongst about thirty horses, which were always on the veld, no cases occurred.

On further investigation and enquiry it was ascertained that the owner prepares his own bonemeal and that the unsterilised bones were partly broken up on the same cement floor on which he chaffed the oat-hay fed to the horses. Pieces of evil-smelling bone and a fair amount of bone dust were still lying on the floor when the place was inspected. No material could, however, be found in the manger.

From the symptoms, history, and circumstances, a diagnosis of botulism due to contamination of the hay with carcase material was made. The case is reported to draw attention to the circumstances under which it occurred.

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Photographs of Donkeys suffering from Botulism.

1. — *A donkey in the prostrate position.*

2. — The same donkey as in fig. 1, showing complete paralysis of the neck. Note the open mouth.

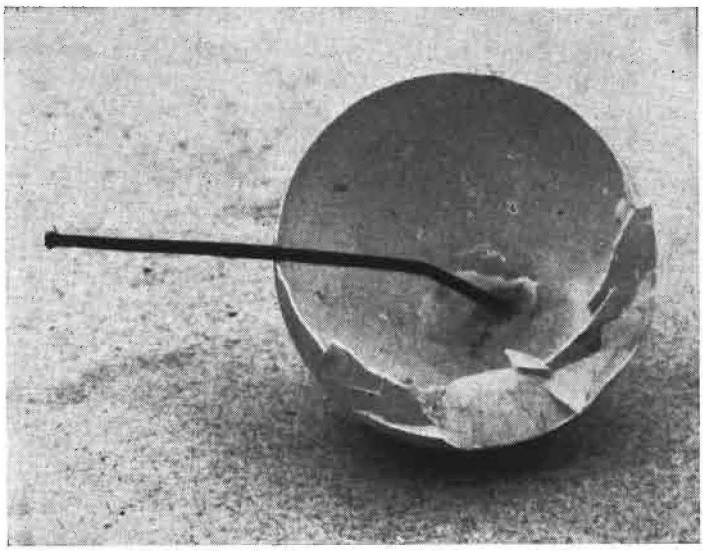
3. — *A donkey that could remain in the normal lying position when assisted to it. When coaxed to rise it would make feeble attempts only with the hind legs.*

4. — *The case in which the jaws could be pulled open by pulling the lips apart.*

Grass as a Foreign Body in Eggs.

By A. S. CANHAM, Allerton Laboratory, Pietermaritzburg.

Recently a Director of one of the Union Egg Circles brought to the Laboratory two dried up pieces of thin grass, each about 4 to 6 inches long, and stated that these had been found inside eggs received from a certain member of the Circle. At the time of candling, it was suspected that a worm of the Ascaris group was present in the egg, but on breaking it, grass was found. It was suggested by the writer that this was a mistake, and that someone was indulging in practical joking. However, within a few days, an intact egg was received from the same farm and, on its being candled at the Laboratory, a green body that moved slightly when the egg was rotated was seen.



The egg was broken and a piece of broad green grass was found passing through the membrane enclosing the albumen; it was fused at the end to the inside of the shell. This piece of grass was roughly 4 inches long. Worms and fæcal matter are at times present in eggs, and more frequently small clots of blood are found also. Grass, however, is not a common foreign body. A questionnaire was kindly sent out by the Egg Circle to the farmer who forwarded the eggs. The questions and answers are as follows:–

1. Do your hens lay in nest boxes, or out on the grass? — The hens lay in boxes lined with grass.

2. If eggs are collected from nest boxes, are you using chaffed grass for nesting material? — The eggs are collected from ordinary boxes which I use for nests, and the grass I am using for nesting material is not chaffed.

3. What kind of grass are you using? — Sometimes I use old hay. If none to hand, I cut fresh green grass and dry it before putting into the nest. The last grass I used was Umtshigi grass.

4. Has this material recently been renewed, because two of the three pieces were still green? — Yes, the material was renewed a few weeks ago.

It is surmised that the grass found in the egg at the Laboratory was Umtshigi grass. The only probable explanation for the presence of the grass in the egg is that during laying, suction or a form of reversed peristalsis took place in the oviduct, drawing the grass far up into this organ, where it became included in the egg. A photograph was taken, showing this uncommon egg inclusion.

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Pronounced Allotrophagia in Bovines.

By O. T. DE VILLIERS, Stellenbosch-Elsenburg College of Agriculture of the University of Stellenbosch, Stellenboch.

The article "Gastritis Traumatica and Rumenotomy in Bovines" by Dr. G. Kind in *Jl. S.A.V.M.A.*, March, 1939, has recalled to my mind the following case of pronounced allotrophagia in cattle.

Subjects. — Hereford oxen aged about two years.

History. — While I was stationed at Grootfontein School of Agriculture, Middelburg, Cape, a farmer reported that one of his oxen had died and that a few others appeared sick from a cause unknown.

Diagnosis. — Post-mortem examination revealed the rumen and reticulum almost completely filled with rounded river stones, the largest of which were about the size of a golf ball. On palpation of the abdomens of the sick oxen, which were all pot-bellied, it was evident they also had been ingesting stones freely.

Aetiology. — Aphosphorosis possibly caused the craving, because the animals subsisted entirely on the veld.

Treatment. — I performed rumenotomy on one of the animals and removed sixty-two pounds of stones (see photograph). First-intention healing took place, but, although the animal fed fairly well, it was still in poor condition when about two months later it was accidentally killed.

Old Motor-Car Batteries as a Source of Acute Lead Poisoning in Cattle.

By J. M. FOURIE, Hoopstad.

That parts of old motor-car batteries may be a source of acute lead poisoning in cattle is indicated by the following cases encountered recently in the Brandfort and Hoopstad Districts.

During March of this year in the Brandfort District one farmer lost eight head of cattle within a week. The cases occurred about three weeks after the animals had been turned into a camp where they had access to the plates of old batteries left behind by workmen of the National Roads Board.

Most of the animals died rather suddenly, but one cow was sick for four days. The symptoms noted were signs of abdominal pain and a short spell of bloody diarrhœa in the case of the cow which lived for four days. Most characteristic, however, were the symptoms of pronounced cerebral excitement, e.g. turning in one direction, rolling of the eyes, champing the jaws, convulsions, and crashing into objects. Profuse salivation, signs of blindness, and semi-unconsciousness were noted. On post-mortem examination, fatty degeneration of the liver, catarrhal to hæmorrhagic enteritis, and hæmorrhages in the myocardium were observed. Specimens of liver and stomach contents, submitted to Onderstepoort for analysis, contained respectively 34·2 and 159 mg. lead per 100 gm. of material. These figures are so high that there is no doubt that the animals died of lead poisoning.

More recently, in the Hoopstad District, a farmer lost three head of cattle on consecutive days. The first animal, which was not seen to be ill, was found dead in the water in the morning. The other two animals, which were both sick for a day only, showed such pronounced cerebral excitement that the owner reported the cases as suspected rabies (later excluded by histological and biological examination of brain material). Unfortunately a post-mortem examination could not be carried out at the time, but a few days later, when the occurrence was investigated, parts of an old battery which a fortnight previously had been broken up on the homestead, were still lying about. Most of the plates, however, had apparently been consumed by the cattle. In view of the previous experience, a diagnosis of lead poisoning appeared justified.

Obviously this danger is greater in areas where cattle are more inclined to develop pica. The acid or salty taste of recently broken-up batteries probably constitutes an additional attraction for the animals. Some of these old plates contain fairly big accumulations of lead salts, which are probably more easily assimilated and thus render the poisoning more acute. In view of the fact that old batteries are not always properly disposed of, similar cases may not be uncommon.

BOOK REVIEWS.

The second edition of Gaiger & Daves' now well-known *Veterinary Pathology and Bacteriology*[1] contains many improvements, errors having been eliminated, and numerous sections having been brought up to date and amplified. Nevertheless the general criticism still holds good, that in attempting to cover so immense a field, fundamental subjects have been treated too summarily. In fact, the sections dealing with general pathology and general bacteriology consist of little more than a series of elaborated definitions which constitute a completely inadequate foundation of knowledge for the subsequent study of epidemiology and preventive medicine. A glaring example in this connection is the sketchy description of Ehrlich's side-chain theory, which is all that the authors have to say about the origin and nature of antibodies and the possible explanations of the phenomena of immunity. Then again the question of optimal proportions is never mentioned in connection with the precipitin test.

It is of interest to note that the strange order of arrangement of the material is still adhered to. Thus general and special pathology are separated by 300 odd pages of bacteriology, but the pathology of infectious diseases is described under the chapters dealing with the causal organisms. Consequently the reader is faced with the histo-pathology of tuberculosis before he knows the definition of pneumonia. Then again, why not describe heartwater under a general heading of "Rickettsia," instead of discussing heartwater first and the general heading "Rickettsia" afterwards?

Generally speaking, the teaching in this book is orthodox and reliable and should be useful to students cramming to pass examinations, but it will only serve to replace rather than to supplement a well-balanced course of lectures. The treatment of tropical and subtropical conditions is too brief for this work to be popular in the Southern Hemisphere.

E. J. PULLINGER.

[1] "Veterinary Pathology and Bacteriology," by S. H. Gaiger, F.R.C.V.S., Prof. of Veterinary Pathology, Univ. of Liverpool, and Gwilym O. Davies, M.V.Sc., M.R.C.V.S., D.V.H., Lecturer in Veterinary Pathology, Univ. of Liverpool. 2nd Edition by Gwilym O. Davies, 1938, pp. 712. London: Baillière, Tindall & Cox. 25/-.

* * *

Professor Wright has done those members of the veterinary profession who cannot read German a great service by making Professor Benesch's little book on *Veterinary Obstetrics*[2] available in English. It is pre-eminently a practical book which cannot be too strongly recommended.

Professor Benesch reveals a very thorough knowledge of the mechanics of obstetrics. The book is clearly written, very well illustrated and descriptions and instructions are so lucidly given that the reader can grasp them without effort.

Little or nothing about the physiology of pregnancy, parturition and the post-natal care of the mother and offspring are given, but this cannot be described as a defect. The author's object was obviously to assist practitioners and students to perfect their technique in obstetrical practice and in this he has succeeded exceptionally well. Several instruments are described and illustrated which must be invaluable and will probably be new to many South African veterinarians.

H. P. S.

[2] "Veterinary Obstetrics (Bovine and Equine)," by Dr. Franz Benesch; English translation. Edited by John G. Wright, F.R.C.V.S., 1938, pp. ix + 258, illustrations 135, London: Baillière, Tindall & Cox. Price 12/6.

It is a remarkable fact that English veterinary literature is poor in really good textbooks dealing with diseases of dogs and cats. *Hobday's Surgical Diseases of the Dog and Cat*[3] is probably the best one available dealing with the surgical diseases of these animals.

It is eminently suited to the requirements of the general practitioner and student, mainly because it is well and clearly written and concise. It deals with all the conditions which are commonly encountered.

The main defects noted are the very incomplete description of the treatment of fractures and an absence of a detailed description of the technique of plating, pegging and wiring bone.

H. P. S.

[3] "Hobday's Surgical Diseases of the Dog and Cat," by J. McCunn, pp. viii 389. 311 Illustrations. London: Baillière, Tindall & Cox. Price 21/- + 1/1.

* * *

Not only veterinary science, but human medicine owes a debt of gratitude to Feldman for his excellent monograph on avian tuberculosis.[4] The four hundred odd pages may look formidable, but the book has been written in such an attractive style that it is a pleasure to sit and read through the mass of information presented.

Those who know nothing of tuberculosis will profit by studying the book from cover to cover. Those who already know a lot, will profit still more. These remarks apply to students of all three types of the disease — human, bovine and avian. Comprehensive and at the same time valuable works on avian diseases are woefully scarce, and the publishers are to be congratulated on bringing to the light of day this monograph by Feldman.

J. D. W. A. C.

[4] "Avian Tuberculosis Infections," by William H. Feldman, D.V.M., M.S.; pp. ix + 483. Illustrated. London: Baillière, Tindall & Cox. 31/6.

OBITUARY.

Major W. G Barnes.

The death occurred in Durban somewhat unexpectedly on August 1st of William Gordon Barnes, M.R.C.V.S., M.C., (Major, late R.A.V.C.), barely a week after the death of his son, Mr. Harry Barnes. It is believed that the shock of his son's death hastened his demise.

Born in the Highlands of Scotland some seventy-three years ago, the late Major Barnes was widely travelled and had had a varied career. At one time he was attached to the Indian Army; he saw service in the Anglo-Boer War with the Rough Riders, and during the Great War he was attached to the Lahore Division of the Indian Army. At the time he left South Africa to join up he was entrusted with the important work of carrying dispatches from General Botha to Lord Kitchener.

Prior to his arrival in South Africa, the late Major Barnes held the appointment of Superintendent of the City of London Abattoirs and Cattle Markets at Islington, and many of the South African students no doubt will remember with gratitude the guidance and assistance so freely given by him,

when attending at that abattoir for practical demonstrations. For his services in London he was made a Freeman of the City in 1910.

It is noteworthy that evermy member of his family saw active service in the Great War, and one of his sons fell in action.

The late Major Barnes was appointed Director of the Municipal Abattoir and Transport Department of the Durban City Council in 1913, and he served the Corporation faithfully and well.

He had a very sound knowledge of all matters appertaining to abattoir control. Members of the profession in South Africa should be very grateful to him and other pioneers for the eminently satisfactory status which has been accorded to professional officers. It is as well to record points of this nature, since we are living in an age when so much is taken for granted.

The funeral took place at Stellawood Cemetery on the 2nd instant, and was conducted by the Rev. H. F. Yule, full military honours being accorded.

A wreath was sent by His Worship the Mayor and Councillors of Durban, and the City Council was represented by Councillor A. Youngleson and Mr. W. A. Dykins, who succeeded the late Major Barnes in office in 1932.

W. A. D.

THE ASSOCIATION.

Secretary's Report for the year ending 31st March, 1939.

Membership. — At the close of the year under review the membership was 173 (168 ordinary members, 4 life members and 1 Honorary Life Vice-President). It is with sincere regret that we record the death during the year of five of our oldest members, namely, S. H. Ewing, W. A. Footner, F. Hutchinson, J. A. Maybin and B. Young. Two members resigned and seven new members were elected.

Council. — Three meetings of Council were held. One of these was a special open meeting held in Johannesburg mainly for a discussion of complaints raised by private practitioners regarding the admission of animals to Onderstepoort for treatment.

International Veterinary Conference. — The Association was ably represented by Drs. P. J. du Toit and J. Quin at the Thirteenth International Veterinary Conference in Switzerland in September, 1938. It has already been suggested that the 1946 Conference be held in South Africa and every endeavour should be made by this Association to bring that about. Most of our members do not appear to be aware of the fact that they can become members of such conferences and receive all the publications on payment of £1.10.0 subscription.

S.A.V.M.A. Book Fund Prize. — This prize of £10 annually for students taking the Veterinary Course was founded last year, the nucleus being furnished by the transfer of £100 derived from the profits of the Book Fund. The first award was made this year, the recipient being Mr. G. P. Bishop, who is a fourth year student.

Dr. K. Schulz. — For over four years Council has been pressing the claims of this member for proper compensation for injuries sustained by him when involved in a shooting affair in the course of his duties. A petition was finally submitted to the last Session of Parliament with the result that substantial compensation was awarded. It is gratifying to record that throughout this

period all representations made by the Association received very sympathetic consideration from the Department of Agriculture and that the Department also accorded its full support to the final petition to Parliament. In appreciation of the assistance received from the Association Dr. Schulz has made a generous donation to the Benevolent Fund.

Finance. — Reference to the report of the Finance Committee will show an all round improvement in the funds of the Association. The credit balance of £1,276-19-11 shown in the balance sheet is an increase of £126-3-3 over that of last year. It should, however, be pointed out that of this sum £100 represents a transfer from the Book Fund.

The Benevolent Fund shows a credit balance of £379-5-6 which is an increase of £49-4-2. A net profit of £15-14-9 was made on the Group Endowment Insurance scheme. This will be transferred to the Benevolent Fund.

In conclusion it is a pleasure to record thanks to all those members who contributed their fair share in promoting the welfare of the Association. In this connection one would particularly mention the President, Vice-President and Assistant Secretary.

S. W. J. van Rensburg,
HON. SEC.-TREAS., S.A.V.M.A.

Report of Finance Committee for 1938-39.

Members of Committee: R. Alexander, B. S. Parkin, C. J. van Heerden, G. J. van der Wath.

The books of the Association were duly audited for the year ending 31st March, 1939, and the appended statements reflect the position of the funds on that date.

A. CASH STATEMENT OF INCOME AND EXPENDITURE.

Receipts.				*Expenditure.*			
Credit Balance on 1.4.38	£91	2	10	Printing of Journal	£232	13	0
Members' subscriptions	340	6	6	Stationery	6	7	6
Donations: Benevolent Fund	2	4	0	Placed on Fixed Deposit	250	0	0
Union Loan Certificates	49	12	0	Benevolent Fund	41	9	0
Interest	41	0	0	Clerical Assistance	30	0	0
Advertisements	30	6	0	To No. 2 Account	3	4	10
Subscriptions to Journal	28	1	11	Natal Branch	5	0	0
Sale of Reprints	34	15	0	Annual Dinner and Lunches	43	14	0
Annual Dinner and Lunches	46	3	3	Sundry Expenses: Annual Meeting	3	14	6
Book Fund	471	1	2	Book Fund	375	6	2
Refund	2	10	0	Petty Cash	30	0	0
				Auditing	2	2	0
				Advance	7	0	0
				Wreaths	2	2	6
				Council Meeting Expenses	1	1	0
				Bank Charges	5	5	7
				Credit Balance on 31.3.39	98	2	7
	£1,137	2	8		£1,137	2	8

B. BALANCE SHEET, 1938-39.

Assets.				*Liabilities.*			
Investments:				Caxton Printing Works	£40	5	0
Union Loan Certificates	£750 8 0			Natal Branch	15	2	0
United Building Society	400 0 0			Subscriptions paid in advance	46	15	0
		£1,150	8 0	Credit Balance on 31.3.39	1,276	19	11
Arrear Subscriptions		125	15 0				
Advance		4	10 0				
Cash in Bank 31.3.39		98	2 7				
Cash in Hand		0	6 4				
		£1,379	1 11		£1,379	1	11

C. BENEVOLENT FUND, 1938-39.

Credit Balance on 1.4.38 ..	£330	1	4	To Widow "C"	£20	10	0
From Cash Account	41	9	0	Credit Balance on 31.3.39	379	5	6
From Insurance Account	14	17	8				
Interest	13	7	6				
	£399	15	6		£399	15	6

D. GROUP ENDOWMENT INSURANCE ACCOUNT, 1938-39.

Credit Balance on 1.4.38	£14	17	8	Premiums: S.A. Mutual	£571	0	8
Premiums Collected	588	0	5	Benevolent Fund	14	17	8
				Bank Charges	1	5	0
				Credit Balance on 31.3.39	15	14	9
	£602	18	1		£602	18	1

S. W. J. van Rensburg,
HON. SEC.-TREAS., S.A.V.M.A.

Minutes ot fhe Annual General Meeting of the Natal Branch of the South African Veterinary Medical Association

held at Allerton Veterinary Research Laboratory, Pietermaritzburg, on 28th April, 1939.

Present. — Dr. P. J. du Toit, Messrs. R. Paine, A. M. Diesel, J. Nicol, R. H. T. P. Harris, C. M. Sharpe, S. T. Amos, L. Morford, D. T. Mitchell, F. J. Carless, A. S. Canham, Dr. P. R. B. Smith, N. C. Starke, S. T. Jackson, D. A. Haig, L. W. Rossiter, W. G. Barnard, J. F. Fick, J. H. Schoeman, J. H. B. Viljoen, F. B. Wright, A. Tarr, H. G. J. Franz, P. R. Mansvelt, C. W. A. Belonje, J. L .Doré, L. C. Blomefield.

The President, Mr. Paine, welcomed Dr. du Toit, Mr. Harris and members.

The Secretary then read the Financial Statement, which was accepted, and it was unanimously agreed that the minutes of the last Annual General Meeting be taken as read. The Secretary referred to subscriptions which were in arrears, and asked if the meeting was agreeable to his sending out accounts to members who were unable to come to the meetings.

The Secretary read a telegram received from Mr. van Heerden regretting his inability to attend the meeting and wishing it success. Messrs. Zwarenstein and Hellberg were unable to attend through ill-health.

The following *new members* were proposed, seconded and welcomed:— J. F. Fick, P. R. Mansvelt and A. Matthew.

Mr. R. Paine referred to the death of four colleagues during the year, Messrs. Ewing, Footner, Hutchinson and Simpson, and the meeting signified their sympathy in the usual way. It was proposed that a letter of sympathy be sent to each of the widows.

The President then addressed the meeting as follows:—

Gentlemen —

I do not intend to detain you long, but there are one or two topics of professional interest which it would be wrong to neglect.

Public Health. — I have never been satisfied with the progress we have made in South Africa in establishing our legitimate position in public health administration. One can hardly believe that for nearly forty years the profession has drawn attention to the fact that the veterinarian and he alone is qualified to estimate the extent and importance of a pathological lesion in an animal from a public health aspect; further, that he alone is qualified to determine the correct way in which animals should be kept in order to maintain them in a healthy condition.

I trust the profession will wake up and demand its position in such administration, as is the case of most progressive European countries.

Central abattoirs at various points in South Africa have been suggested. Such a movement would assist in establishing a competent veterinary staff at such centres, but we would still have the present glaring lack of efficient control of our dairies.

Department of Defence. — It is satisfactory to note that the Minister of Defence is not satisfied with the complete mechanisation of transport in his department. It should stimulate horse breeding in South Africa, and one feels that there is plenty of scope in this department for veterinary guidance in the control and general management of its equine transport.

Interest in public civil life. — Many members of our profession are handicapped in South Africa by their official positions, which prevent them from taking an active interest in civil public life. However, especially in Natal, we are to be congratulated on the position of some of our colleagues, and the esteem in which they are held by the public.

I also wish to draw attention to the S.P.C.A. and kindred societies. In Great Britain our colleagues take a very active part in the control and guidance of these societies to the advantage of the profession as well as of the actual societies concerned. As the authorities upon the control and management of animals we should establish our connection with these societies. We can guide them in the necessary and correct steps, and also keep them from becoming the tools of absurd and harmful movements, which would in many cases hinder necessary research work.

Proprietary medicines for stock. — We have a most excellent legislation in connection with proprietary veterinary preparations, and only the other day a member in the House of Commons drew the Minister's attention to it and suggested its adoption in Great Britain — another professional honour to South Africa.

I feel, however, that our members do not make full use of this machinery. A list of remedies registered for 1939 was published in the Government Gazette Vol. CXV No. 2611 of 28.2.39, and our members might keep this publication on their desks for their own information, and for the edification of the public who might make use of such preparations.

Before closing I feel that although the President of our parent Association has already made reference to the matter, we should as a Natal branch congratulate our colleagues in the field upon the most excellent and efficient way in which they dealt with the recent Foot and Mouth Disease outbreak in Natal. The success of the work must have been largely due to their tact and patience in handling the European and Native owners.

I also wish to express our thanks to our very capable and energetic Secretary and Treasurer, to those who have contributed papers, etc., to our meetings, and to the Officer-in-Charge, Allerton Laboratory, for the use of this building for our meeting.

The following *office bearers* were elected:—

President: W. A. Dykins.
Vice-Presidents: A. M. Diesel, R. Paine, A. S. Canham.
Secretary: L. C. Blomefield.
Committee: Messrs. C. M. Sharpe, S. T. Jackson, D. A. Haig, H. G. J. Franz and F. B. Wright.

General business. — Mr. Diesel referred to the death of Mr. Footner and the valuable work he had done. He said that a fund had been started in commemoration of Mr. Footner, and suggested that the Natal branch of the S.A.V.M.A. make a donation. Mr. Sharpe proposed £5-5-0, this was seconded by Mr. Diesel and all were in agreement.

Mr. Amos expressed the hope that this Fund would not be limited only to State Veterinarians, but kept open to all in the profession. He suggested that the form this commemoration should take should be a headstone, and also put forward the suggestion that a small committee be selected as Trustees to this Fund, as he pointed out that there would be a matter of about 30/- a year to be found for the upkeep of this grave. Messrs. Amos, Diesel and Paine were elected.

Mr. Diesel proposed and it was unanimously agreed that a telegram be sent to Mr. van Heerden expressing the hope that he would soon be restored to normal health.

Mr. A. S. Canham, F.R.C.V.S., then gave a paper on "*Ophthalmia in Cattle and Sheep.*"

The Chairman thanked Mr. Canham for the very comprehensive paper he had prepared, and for bringing to light so many points about which they all knew very little. He then declared the discussion open, and various questions were asked by the members to which Mr. Canham replied as follows:—

He was of opinion that ophthalmia is more frequent in wet seasons than in dry. Ophthalmia does not appear to occur with greater frequency among cattle pastured on damp places than among cattle grazing on dry lands, but cattle feeding in bushy country seem to develop the disease more frequently than cattle pastured on grass veld; also that stock brought in during winter for feeding in kraals often contract the disease. He showed graphs which bore out his theory.

With reference to the question of susceptibility of certain breeds of cattle, Mr. Canham is definitely of the opinion that imported breeds are more prone to this disease than cattle of the Afrikander type and its crosses. From his investigations, he has found that Hereford cattle are more susceptible than any other breed of cattle.

Mr. Barnard brought forward the question of ticks being the cause of ophthalmia, and Mr. Canham said that it was his opinion that ticks did cause this disease, the animal, in many cases, scratching its eye-ball in an attempt to scratch its ear.

Mr. Canham said that he had always been of the opinion that worms were the cause of ophthalmia, but he had once taken 45 worms out of the eye of a Hereford cow which had never had ophthalmia, and which, during the following five years, never developed it. Also he had experimented with calves two hours old (at this stage they are most susceptible), and put worms, male and female, into their eyes. They were closely watched, but in no case did ophthalmia result. Another interesting point was that, during all his investigations, he had never found worms in the eye at the commencement of ophthalmia.

To Mr. Amos' suggestion that flies played a great part in the causation of ophthalmia, Mr. Canham replied that he had carried out experiments in which saline emulsions of flies were put into the eyes of both adult and young cattle without ophthalmia resulting.

In reply to Mr. Diesel's query as to whether dipping affected the incidence of ophthalmia, Mr. Canham said that at the School of Agriculture, Potchefstroom, the animals were dipped every seven days throughout the warm weather, and that there was no reduction in the average monthly number of cases.

Mr. Canham agreed that it is a possibility that game may take a part in increasing the incidence of ophthalmia, but he had had no experience of it.

He also suggested that the main reason why calves appear to contract the disease easily is because they are in most cases kept in confinement. It also appeared that ophthalmia in calves is rather different from that disease in adult cattle, as it appears to spread more quickly among calves, but is more easily cleared up, leaving fewer blemishes on the cornea.

Mr. Canham was of the opinion that ophthalmia is not contagious, because during his investigations he had experimented by tying infected cattle with normal healthy cattle, letting them feed from the same trough, but had not been able to transmit the disease. He had also attempted to transmit the disease by means of applying the lachrymal secretion of infected animals to the eyes of healthy adult, young and baby cattle, on cotton-wool swabs, but failed again to transmit the disease.

As far as treatment of ophthalmia was concerned, Mr. Canham pointed out that it was very difficult to lay down any hard and fast rule, but said that from his own experience he had found the following to be most effective:—

1. In a new case, i.e. as soon as the eye starts watering, he had found by keeping the animal away from the sunlight and by washing the eye frequently with normal saline or even cold tea, good results are obtained.

2. In a case two or three days old, the most effective treatment is a lotion containing formalin. Formalin is a preservative and probably acts by hardening the cornea, thus limiting the spread of the process. The animals should be kept in the dark.

An interesting feature in the treatment of ophthalmia is the fact that one year the results from a certain treatment will be excellent, but the next year there may be no results at all from the same treatment. His advice, therefore, is to prepare several lotions, e.g. a saline lotion, a formalin lotion, and perhaps a boracic lotion, and give alternate treatments with these. Towards the close of the disease, yellow mercury oxide ointment is effective.

Ophthalmia in Sheep. — Mr. Canham said that he had only one thing to add to his paper on sheep ophthalmia, and that was that he had obtained excellent results from the use of methylene blue in the treatment of this disease in sheep.

This year a successful experiment had been made by which all members were circularised with a copy of Mr. Canham's paper prior to the meeting.

The following is a short resumé of Mr. R. H. T. P. Harris' talk on the *Electric Fence.*

I had heard good reports of this "one-wire" electric fence, so I went to Onderstepoort and suggested that we should try it out. They agreed, and an agent came out and set up a battery. Attractive foodstuffs were put outside the fence, and the animals simply walked right out. It failed hopelessly.

On my way through Durban, however, I managed to get a machine on 60 days' trial, and set this up at Hluhluwe. It had one strand of wire, three feet from the ground, and the animals broke this down daily, so my assistant, Capt. Shenton, had the idea of raising the wire from three to six feet off the ground and of hanging wires therefrom to within six inches of the ground in order that the main wire might no longer be broken.

The cattle have to be taught to avoid this fence, so, for this purpose, some well-moistened, attractive foodstuff is attached to the wire so that the animals receive a shock on their wet noses. In the case of wild animals, their habit of smelling any unfamiliar object leads them to attempt to smell the fence, resulting in a severe shock on their noses (probably their most sensitive organ). Animals soon learn to associate the pain of the electrical shock with the hanging wires, and will make no second attempt to pass through the fence.

The tsetse fly will not travel long distances unaided, therefore if we can stop the encroachment of game into cattle areas, we would have no more Nagana.

Mr. Franz then pointed out the advantage of the electric fence in connection with the trapping of flies in Nagana areas. He explained that traps were very expensive (about £1 each), and if a whole farm had to be trapped out the expense would be considerable, but if the cattle could be concentrated within a small portion of the farm — this would automatically bring the flies to the same spot — then it would only be a question of trapping a small area, and the expense would be considerably reduced.

Mr. Tarr said that the electric fence was being used quite extensively on the coast, and that he believed it was working very well.

Dr. du Toit paid special tribute to Mr. Harris' work and the President thanked him for his most interesting and instructive talk.

Mr. S. T. Amos opened the discussion on *Clinical Work.*

He said that he had brought up the subject of Strangles particularly, because horses were becoming more and more valuable economically to the country, and the consequences of an outbreak of Strangles in any of the racing centres would indeed be very serious.

Mr. Amos then described a very unusual outbreak which had occurred in Durban about last August. It was peculiar in this way: in no instance was the animal's temperature over 102·5°; even in the initial stages, laryngitis, which is one of the most common first symptoms, was practically absent; there was increased salivation; the discharge from the nostril, instead of being of a thick creamy nature, was of a diluted nature, and left a peculiar marking on the nostril; the gland was slow to ripen, and did so incompletely. The element of contagion seemed to be just as marked as in an ordinary acute outbreak, and he had to keep the horses in strict quarantine for five or six weeks. He treated them with a serum made by Bruno of France, which usually gave excellent results, but in these cases he did not get the usual reaction.

A general discussion followed, during which Mr. Franz said that he had encountered a similar case of Strangles, except that the discharge from the nostril was slightly oily.

The President invited Mr. Morford to give his experience of Biliary Fever in dogs and his opinion on Acaprin as a treatment. Mr. Morford said that he has found Acaprin very good indeed, and doses the dogs with 1 cc. per 24 lbs. body weight. (He always weighs the dogs, and usually makes the initial dose a little less.) Two days later he repeats the dose in full. In his opinion the main thing is to keep the dogs absolutely quiet, and he advises giving them a stimulant. He gave an example of a case which he treated for Biliary Fever, but which died, and on post-mortem examination it was found that the

dog was simply riddled with hookworm. He found it practicable, in Durban at any rate, to give a dog about a week's rest after Biliary Fever and then treat it for hookworm. He also mentioned a condition he had encountered rather a lot during the last six months — Canine Typhus. These diseases were thoroughly discussed, as was the debatable point whether dogs should be fed on bones or not.

The President thanked Mr. Amos and Mr. Morford for their most instructive discussions.

Mr. W. G. Barnard then gave an interesting paper on *Onchocerciasis in East Griqualand.*

During the short discussion following this paper, Mr. Diesel said that a few years ago, the Superintendent of the Pietermaritzburg Abattoir was greatly concerned as certain cattle from the midlands of Natal seemed to be badly affected with lesions on the top of the back. Representations were made to the Director of Veterinary Services at Onderstepoort, and an officer came down to investigate the condition. It was traced back and ascertained that the animals had come from the Weenen District, and farms in that district were inspected, and it was found that this condition was fairly widespread, but since then nothing further has been heard about this matter.

Mr. Rossiter said that he had seen a tremendous amount of this condition in the Ladysmith and Winterton Districts, and had encountered a lot in Northern Zululand. He was surprised to find, in view of the widespread nature of this condition in Zululand, that the beef was not condemned at the abattoirs, and suggested that perhaps the nodules were only superficial and did not affect the beef for human consumption.

Mr. J. H. Schoeman then gave a demonstration of a most ingenious way of tying a knot with forceps.

The meeting commenced at 9.30 a.m. and concluded at 4 p.m.

Jl. S.A.V.M.A.
X(4): 151—156
1939.

The Diagnosis of Arsenical Poisoning of Stock.

By DOUW G. STEYN, Section of Pharmacology and Toxicology, Onderstepoort.

Introduction.

A paper on arsenical poisoning in man and animal, in which the forensic aspect will be discussed, and in which detailed reference will be made to experiments conducted at Onderstepoort, will be published in due course in the *Onderstepoort Journal of Veterinary Science and Animal Industry.* Hence, in the present paper only the most important points concerning the diagnosis of arsenical poisoning will be considered. Special reference will be made to experience gained from the examination of routine specimens and from experiments conducted at Onderstepoort. In the last fourteen years approximately twenty-five thousand specimens were analysed at Onderstepoort for the presence of arsenic. The extensive use of arsenical preparations by stock-owners, as remedial agents, as dipping-fluids, insecticides, and worm remedies, and the use of arsenic for the extermination of locusts has been the cause of much arsenical poisoning of stock. Careless handling of these preparations has been responsible for a very high percentage of these cases of poisoning. Malicious poisoning also takes toll of a large number of stock in South Africa.

Submission of Specimens for the Detection of Arsenic.

The method of analysing specimens for the presence of arsenic which has been found of most practical value as a routine is that described by Green (1918). The limit of sensitivity of this test as used by us is approximately 0·02 mg. arsenious oxide (As_2O_3). This is a point of the utmost importance as the quantity of arsenic present in specimens of organs submitted and its distribution in the different organs are a most important consideration in the diagnosis of arsenical poisoning. They depend both upon the quantity of arsenic taken in and its point of entrance into the body (oral or parenteral). The size of a fatal dose of arsenic determines the length of the period which the animal will survive and consequently the amount and distribution of arsenic in the different organs, as is evident from the following discussion.

A. — *Peracute arsenical poisoning.*

At Onderstepoort, sheep died within twenty minutes after drenching with approximately two hundred minimum lethal doses of sodium arsenite in a ten per cent. aqueous solution. We found large quantities of arsenic in the gastro-intestinal contents (150-200 mg. As_2O_3 per 100 gm.), and liver

(4·0 - 10·0 mg. As_2O_3 per 100 gm.), as there was hardly any time for excretion of the poison from the body.

In cases of peracute and acute arsenical poisoning there may be a marked difference in the arsenic content of the ruminal and abomasal ingesta depending on whether the animals drank solutions of arsenic or licked powdered arsenical preparations. In the first case the solutions usually find their way into the rumen, whilst the powder may find its way directly into the abomasum.

A point of the utmost importance, which was proved by experiments conducted at Onderstepoort and amply corroborated by the analysis of specimens collected by the author in outbreaks of peracute arsenical poisoning in the field, is that the bones of animals dying from peracute arsenical poisoning not infrequently contain small quantities of arsenic not detectable by the routine method of analysis used by us and by workers in many other laboratories. That is, such specimens of bone may contain less than 0·02 mg. of As_2O_3 per 100 gm. It is obvious, therefore, that the quantity of any specimen, and especially of bone, taken for analysis is of the utmost imporance. If we were, for example, to take for analysis 25·0 gm. of a specimen of bone containing 0·06 mg. As_2O_3 per 100 gm. we should fail to detect arsenic, whilst the test would have been positive had we taken 50 gm. of bone. Therefore, in our routine analysis of specimens 100 gm. of bone is taken. Witth regard to other organs we have found it best to take 50 gm. of liver, 50 gm. of stomach contents, and 50 gm. or 100 gm. of skin or hair for each test. Where we suspect that the specimen contains only very small quantities of arsenic larger quantities are taken for analysis.

B. — *Acute and subacute arsenical poisoning.*

We have noticed, on a number of occasions, that animals, which succumbed to acute or subacute arsenical poisoning, but which lived for five or more days after ingesting the arsenic, may not have enough of this poison in the liver and gastro-intestinal contents for detection by Green's (1918) method of analysis. This means that such specimens contain less than 0·04 mg. As_2O_3 per 100 gm., since we take 50 gm. of these specimens for analysis and the limit of the test is 0·02 mg. In most cases of acute arsenical poisoning arsenic is still detectable in the liver and gastro-intestinal contents of the victims up to five days, and in some cases even up to fourteen days or longer, after ingestion. It is important that in protracted cases of arsenical poisoning arsenic is usually detectable, in appreciable quantities, in the bones, skin, and hair. This point is further discussed under (C) chronic arsenical poisoning.

C. — *Chronic arsenical poisoning.*

It is evident from the foregoing discussion that, as a rule, we shall not be able to detect arsenic by Green's method, in specimens of liver and gastro-intestinal contents in chronic cases of arsenical poisoning in which the last

quantity of arsenic was ingested a number of days before death. In chronic cases of arsenical poisoning following on a single toxic dose of arsenic, detectable quantities of arsenic will be found in the liver and gastro-intestinal contents only in most exceptional cases. In these, detectable quantities of arsenic are found in the bone, skin (especially epidermis) and hair, except in extreme cases in which the animals live for months and even years after having ingested the last quantity of arsenic. In the latter cases it may be impossible to detect arsenic in any part of the body as there has been sufficient time for excretion. Cases are known where progressive irreparable damage has been done by the repeated ingestion of small doses of arsenic over long periods. Such animals may linger for months and even years, although all the arsenic has been excreted from the body. These cases are analogous to delayed *Senecio,* atophan (cinchophen) and chloroform poisoning.

On the other hand it is evident that in peracute cases large quantities of arsenic will be detectable in the stomach contents and liver. In such cases it might seem unnecessary to submit specimens of bone and skin as well as specimens of liver and stomach contents.

However, it is essential that specimens of bone, skin, liver and stomach contents (500 to 1,000 gm. of each) be submitted for analysis in *all cases of suspected poisoning.* It is of the utmost importance that *each specimen be submitted in a separate container and that no preservatives be added.* Preservatives alter the weight of the sample and make a calculation of the amount of poison difficult and may in some cases re-act chemically with organic poisons. Specimens of suspected materials [licks (500 gm.), water (500 c.c.), feed (500 gm.), soil (500 gm.), etc.] should also be submitted. If specimens are taken from places where animals are suspected of having licked the soil, only about a quarter of an inch, or even less, of the surface should be scraped off and submitted for analysis. If the soil is dug up deeper the concentration of the arsenic is decreased, with the result that it becomes difficult to draw conclusions as to the dangerousness of the soil.

A point of importance is that the presence of appreciable quantities of arsenic in the gastro-intestinal contents is no definite proof that death was due to arsenical poisoning. This fact should be realised where it is intended to institute legal proceedings. To prove beyond doubt that an animal died from arsenical poisoning (or for that matter from any poison) it must be shown that the poison had circulated in its blood; that is, its presence should be proved in the liver, kidney and (or) bone. Hence the necessity for submitting specimens of liver, bone, stomach contents and skin in all cases of suspected poisoning.

It is of the utmost importance that all specimens submitted for toxicological analysis should be accompanied by *detailed information* on the following points:—

(i) History of the disease.

(ii) Symptoms (vomiting, diarrhoea, nervous symptoms, etc.), and post-mortem appearancees.

(iii) Length of the period which the animals lived after developing symptoms. *This information is essential* in order to allow of a correct interpretation of the quantities of the poison detected in the organs.

(iv) Treatment — administration of sodium thiosulphate, purgatives, emetics, diuretics, or any other treatment applied.

(v) Whether the animals are grazed, or stall-fed; kind of licks or feed fed; drinking from troughs, pans, dams or running water, and any other information.

All this is essential to assist the analyst in tracing the poison. The more information he has at his disposal the better and the more reliable will be the information he is able to supply, and the easier his task will be. There are many poisons and it is by no means an easy task to detect the rarer ones.

D. — *Living cases of arsenical poisoning.*

When sick animals are thought to be suffering from arsenical poisoning, specimens of faeces, urine, and hair, (± 100 gm. or more) should be submitted for analysis. In protracted cases of arsenical poisoning no or very little arsenic may be detectable in the fæces and urine, and in such cases it is of great value to administer sodium thiosulphate (sodium hyposulphite, commonly known as "hypo") to stimulate the excretion of arsenic in the faeces and urine. A full-grown beast or horse should be given 8-10 gm. of sodium thiosulphate intravenously in a 10 or 20 per cent. aqueous solution and 20-30 gm. orally in about 300 c.c. of water. Sheep and goats should receive one quarter of these doses. Collection of specimens of faeces and urine should commence within an hour of administration and should be taken on at least two following occasions. The total specimens collected should amount to approximately 500 gm. of faeces and 600 cc. of urine. It should be remembered that, even in acute arsenical poisoning, the excretion of arsenic in the faeces and urine is intermittent, and that it is inadvisable to collect specimens of faeces and urine once only. The sooner after the appearance of symptoms the urine and faeces are collected the greater is the chance of detecting arsenic.

Discussion.

The distribution of arsenic in the different organs is of great importance, *especially in border-line cases.* Hence the importance of submitting the different specimens in separate containers must again be stressed. As a rule we find the following: (a) In peracute arsenical poisoning: large quantities of arsenic in practically all organs, including the stomach contents and liver. In some cases the bones may be negative, or show only traces of arsenic. (b) In acute and subacute arsenical poisoning: fair quantities

of arsenic in the stomach contents, liver, bone, skin, and other organs. In cases which have survived for longer than five days no arsenic, or only traces, may be detectable in the stomach contents and liver. The longer the animal survives the less arsenic is detectable in the gastro-intestinal contents, liver, and in the other organs. (c) In chronic arsenical poisoning: here arsenic is generally detectable in the bone and hair (skin), but not in the gastro-intestinal contents and liver.

It should be kept in mind that many stock-owners use arsenical preparations (especially locust poison, sodium arsenite) in the anti-blowfly campaign. When carcases are exhumed for the purpose of collecting specimens it is advisable also to collect samples of the soil surrounding the carcase to see whether the arsenic present in the specimens of organs has found its way into the carcase from the surrounding soil. It is best to take three specimens of soil (approximately 500 gm.); one from just below the surface of the soil directly above, and the others on a level with and on each side of the carcase. It is unnecessary to go into great detail about the significance of different quantities of arsenic present in the soil. In acute and subacute cases of arsenical poisoning there will not be enough arsenic in the carcase to contaminate the surrounding soil to such an extent as to enable us to detect arsenic therein. However, in peracute cases arsenic may be detected in the surrounding soil in amounts depending upon the quantity of arsenic ingested, the degree of decomposition of the carcase, the nature of the soil, and the rainfall. It is obvious that in these cases analysis of specimens of skin and abdominal contents will tell us whether arsenic was poured or dusted over the carcase or introduced into the abdominal cavity, as is frequently done in the anti-blowfly campaign.

In the interpretation of the quantities of arsenic present in specimens of bone, skin, liver, and stomach contents collected some time after death, it should be realised that the distribution of arsenic in the different organs does not reflect the state of affairs at the time of death, before decomposition set in. It is, for example, obvious that in an advanced state of decomposition the liver will contain much more arsenic than when fresh, if a large quantity of arsenic was present in the stomach contents at the time of death (peracute and acute arsenical poisoning): for the arsenic can diffuse through the stomach wall and contaminate not only the liver but practically all the organs.

It is essential also to submit specimens of skin to determine whether the animals have been recently dipped or whether the skins have been immersed in arsenical solutions as is frequently done to keep them moth-free. It is of importance to note that in dipping experiments conducted at Onderstepoort we failed to detect arsenic in the bones of cattle which had been dipped at weekly intervals for three years. The animals were dipped in a sodium arsenite solution (0·16 per cent. As_2O_3). From time to time one of the animals was killed within a few hours of dipping and

specimens of skin, bone, gastro-intestinal contents, liver, and kidney analysed. Arsenic was never detectable in the bone or gastro-intestinal contents. On the other hand up to 0·1 mg. of As_2O_3 per 100 gm. was detectable in the liver and kidney. All the animals are still in very good health.

Summary.

The diagnosis of peracute, acute, subacute and chronic arsenical poisoning is discussed. The fact that no arsenic, or only a trace, is detectable in the bones in some cases of peracute arsenical poisoning is stressed.

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The author is indebted to his assistants, Mr. N. Reinach, M.Sc., and Mr. D. Malherbe, M.Sc., for the large number of analyses done by them.

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Some Remarks on Whistling and Roaring in Thoroughbred Horses and on the Results of the Ventricle Stripping Operation for Roaring in South Africa.

By JOHN QUINLAN and H. P. STEYN, Onderstepoort.

The object of this publication is to record the results of the ventricle stripping operation for roaring in race-horses in this country. Up to the present, so far as we are aware, no South African veterinarian has recorded such data.

The incidence of recurrent nerve paralysis has been a controversial matter amongst the veterinarians in South Africa. The older practitioners (Runciman, 1936) state that the disease has increased in this country, since the introduction of affected sires, and their unrestricted use for breeding thoroughbred horses. The reason for the importation of whistling and roaring horses may not be apparent to those who are not acquainted with South African conditions. It is difficult to obtain well-performed and well-bred thoroughbred horses for stud purposes from overseas, if they are sound. This has forced South African stud-breeders to take a chance with well-bred and well-performed roaring stallions which can be purchased at a relatively small price in England and Ireland. On the whole this method of introducing the best thoroughbred blood must be considered successful, since it has afforded breeders an opportunity of obtaining blood which would otherwise have been procurable only at a price prohibitive to most stud-masters.

It is a matter of opinion whether the incidence of whistling and roaring has increased in South Africa within the last quarter of a century. There are no data available to support or contradict the statement, made by the older veterinarians, that the introduction of stallions affected with recurrent nerve paralysis has become a menace to thoroughbred breeding in this country. After close association with thoroughbred breeding and racing in South Africa for twelve years, one of us (J.Q.) is unable to state that the incidence of laryngeal paralysis has increased, and he believes that heredity is a very questionable factor in the ætiology of the disease in South Africa.

In this connection it is interesting to note that climate has been inculpated as a factor associated with the incidence of roaring in horses. For instance, Weiss (1937), who has made a careful study of the literature, states that the disease is more frequent in the harsh, cloudy, cold climate of Northern Germany and England than in a more temperate environment. If this is so the dry climate of Southern Africa may act as a preventive

factor. Further according to Weiss (1937) the English Thoroughbred is affected with recurrent paralysis to the extent of about 5 per cent. There are no data available in South Africa to indicate to what extent the thoroughbred horse is affected. We suggest that the increasing incidence of the disease noted by some South African veterinarians may be more apparent than real; that is more cases come under the observation of the veterinarians because there has been a great increase in the number of thoroughbreds bred in this country during the past decade.

It has been impossible to collect comparative data of any real value as to the incidence of the disease in the progeny of affected and unaffected parents. There is only one stud in this country that gives one the impression that the condition may have a hereditary ætiology. In this stud three affected sires have been used covering a period of twenty years. No data are available on the respiratory soundness of the progeny of the first sire used. The second sire gave an indication that he transmitted the disease to a small percentage of his progeny. The third sire, now in use, does not appear to give an abnormally high percentage of affected horses. There are a few cases where it has been possible to follow up the female progeny of the second sire when mated to sound males; these have been sound horses. Again the female progeny of this same roaring stallion have been mated to an imported roarer and the offspring have been sound and good sprinters. It is rather early to make a definite statement, however, as to how they will turn out, as the oldest is now only three years, and the progeny are yet too few.

When discussing the possibility of heredity playing a part as an ætiological factor in laryngeal paralysis one must realize that certain diseases of the respiratory tract, such as strangles, influenza, pneumonia, may leave recurrent paralysis as a sequel. This statement would hardly be contradicted by the veterinarians in any country. *Babesiasis* or " biliary fever " in horses in this country is frequently followed by the same sequel. From our observation we can quite definitely state that many of the imported and locally bred thoroughbreds that suffer from an acute attack of babesiasis are subsequently affected with laryngeal paralysis or paresis. In many cases the affection remains permanent, in others under careful treatment it disappears. There was no question about the respiratory soundness of these horses prior to the attack of babesiasis.

It is difficult, if not impossible, to collect reliable data in this country on the rôle played by hereditary factors in the ætiology of recurrent laryngeal paralysis. Since certain respiratory affections and babesiasis are known to have whistling and roaring as a sequel, an acute attack of one of these diseases must be excluded. This is most difficult as the diagnosis is not always to be relied upon. The stud farms are, as a rule, far from veterinary assistance, so that the breeder frequently makes a diagnosis and treats horses without professional confirmation.

The part played by heredity would be far easier to clear up in England and Eire than in this country, because the previous veterinary history of whistlers and roarers would be reliable. In fact it is surprising that serious attempts have not yet been made to assemble data on the breeding and veterinary history of the numerous roarers that are met with in England and Eire. In this connection the work of Weiss (1937) is praiseworthy. With such data available one is confident that a statistician would be able to make a critical analysis which would help to decide the controversy that exists amongst the members of the profession, some of whom accept the hereditary factor without question, while others see the necessity for careful experimentation before accepting it. There is little to be gained from theorising. Conclusions can be arrived at only by conducting a carefully arranged long range experiment, carried out under environmental conditions which would exclude the diseases which frequently have recurrent paralysis as a sequel. To those veterinarians who accept the hereditary theory without sufficient consideration of reliable data the works of Vermeulen (1914), Mettam (1899), Haslam (1895), Weiss (1937), Wester (1937) and Argyle (1933, 1934) should prove interesting reading.

We hope to study the question of heredity as a factor in laryngeal paralysis at this laboratory in the near future. Investigations into the affection will be undertaken as soon as a suitable stallion becomes available. Such a stallion is not easy to find in South Africa owing to the necessity for knowing most accurately his clinical history, in addition to the history of his parents.

There has not been much opportunity for testing the results of the ventricle stripping operation on account of the relative infrequency of whistling and roaring in horses the value of which would warrant such surgical interference. Still the results have been so very encouraging that no apology is made for recording them. The numbers of horses used for hunting and hacking in this country are very limited, so that no wide field for observation is presented. Polo-ponies in this country are almost invariably sound winded. We have rarely heard an unsound-winded pony in ten years close association with the game. Consequently the majority of patients are thoroughbred race-horses.

A careful examination is made of each patient when it arrives in hospital. Its suitability for the operation is decided by its previous record. The usual history is that the horse at one time showed promise of developing into a useful race-horse, but that later on he failed to stay, subsequent to developing respiratory trouble. As a rule only horses likely to win races if the operation is successful are accepted. The results obtained are judged on subsequent racing records rather than upon the improvement in the inspiratory sound. Consequently only race-horses are considered as suitable subjects upon which to obtain reliable data.

The technique of the operation is that usually conducted [O'Connor

(1938), Merrilat (1921), Röder and Berge (1939), Williams (1907), Frick (1921), Wooldridge (1923), Hobday (1910, 1911, 1935)], with one slight modification. The horse is narcotized by a 10 per cent. solution of chloral hydrate injected intrajugularly while standing. About 35 cc. is injected per 100 lb. body-weight. The dose is slightly decreased or increased according to the condition and character of the animal. Horses in hard condition and those with an excitable nervous temperament get a slightly larger dose. It is always necessary to complete the anaesthesia during recumbency by chloroform inhalation given by the open method.

After the injection of chloral hydrate is completed the horse usually staggers and is controlled so that he falls without danger. An anti-back-breaking harness is always used. When the animal is on the mattress the hobbles are put on. As soon as anaesthesia is sufficiently deep the animal is placed in the dorsal positon by a rope passed through a ring in the ceiling of the theatre. The head is then fixed in a head-holder and extended. The site and also the cranial third of the ventral aspect of the neck is shaved and the skin is thoroughly cleaned with ether and tincture of iodine. Sterile cloths are then placed around the site of operation. Tracheotomy is performed and a tube placed in position before commencing the main operation. After this, chloroform is administered through the tracheotomy opening. Laryngotomy is performed through the cricothyroid ligament, and the cricoid ring divided. All bleeding points are picked up with Spencer-Wells forceps, ligaturing seldom being found necessary. A laryngeal dilator is then placed in the wound. Both ventricles are now operated upon (as the results appear to indicate that bilateral surgical interference is preferable). A burr is passed into the depth of the ventricle. With the horse in the dorsal position the burr is pressed with gentle pressure into the ventricle and rotated anti-clockwise on the left side and clockwise on the right side. As soon as the burr has caught the mucous membrane it is gently withdrawn until the lining of the ventricle can be caught in a forceps below the burr, which is then discarded. Rough use of the burr must be avoided so as to prevent injury to the *M. ventricularis* and *M. vocalis* between which the ventricle lies. The mucosa only must be withdrawn. By manipulation of the forceps the mucous membrane is stripped completely from the ventricle. Without releasing the forceps an incision is made through the mucous membrane all aroung the ventricular opening by snipping small pieces at a time with the blunt pointed scissors. The everted ventricular mucosa as well as a small circle of mucosa around the opening is removed by means of a curved scissors. In this way the mucosa is stripped over the cranial border of the vocal cord. Actual severance of the vocal cord as carried out by Reynolds (1934) and Coquot (1917), has not been done.

In cases where the stripping of the mucosa is not considered sufficient, that is where the border of the ventricular opening is not completely stripped,

the edge of the cut surface is picked up in an artery forceps and a further strip cut off with a curved scissors. This part of the operation is facilitated by passing an artery forceps wrapped around with gauze into the ventricle and everting the cut edge of the mucosa by slight withdrawal.

It would appear that the success of the operation depends upon sufficient stripping of the mucosa, and as little injury to the underlying ventricular wall as possible. At least since this technique has been adopted the results have been more uniformly good.

The tracheotomy tube is left in position until the danger of dyspnoea from laryngeal oedema has disappeared. This is usually about 4 or 5 days. The tube is removed and cleaned daily. The laryngotomy wound is cleaned twice daily with dry swabs and then dusted with iodoform powder. It is protected from flies and contamination by bedding with gauze for several days. The wound is completely healed in a month. The horse is taken out for walking exercise daily, and is not allowed to have anything but walking exercise for two months. After this period he can be tried for his wind but the trial must not be searching. Gradually increasing exercise can be given, but the searching trial should be postponed until three months have elapsed.

Oedema of the larynx has occurred in two cases, but otherwise there have been no untoward sequelæ. The results of the operation have been uniformly good, as the attached list of cases shows. In fact the slight modification of removing the mucosa around the border of the ventricular opening has considerably improved the results in our hands.

As indicated above, suitable cases to judge the improvement in a horse's stamina following operation do not often present themselves in South Africa. Consequently we confine the list to operations on thoroughbred horses that had become quite useless from a racing viewpoint. A careful record has been kept of these horses' performances. The success of the operation on hacks has been no less noteworthy, but since it is difficult to estimate the improvement these cases are not recorded here.

In all, seventeen thoroughbred horses have been operated upon by the modified technique described. Of these all were improved in stamina; eleven were quite sound; six were left with a somewhat harsh inspiratory sound, not the sound associated with roaring; nine won races subsequent to operation; four recently operated upon are still in training; four did not win races.

Fig. 1 shows the larynx of a horse operated upon for experimental purposes by the technique described. It will be observed that there could not possibly be any obstruction to inspiration in a larynx such as this.

Table I. shows a number of horses operated upon. The details given include notes on previous history and the history subsequent to operation, also some explanatory remarks.

TABLE I.

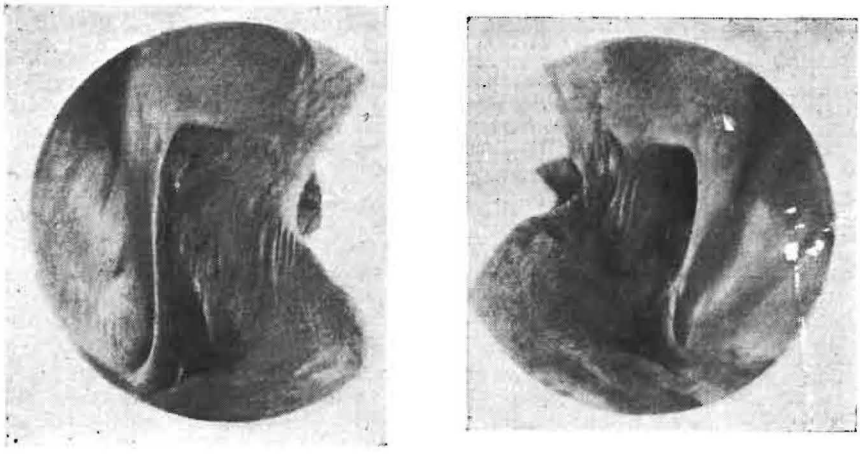
Name.	Previous History.	Subsequent History.	Remarks.
Eye Witness	Pronounced Roarer and abdominal cryptorchid. Sold as useless for racing.	Quite sound of his wind. Won several races and was subsequently killed in a railway accident while still in his prime.	The cryptorchid operation was first performed and ventriculectomy a month later.
Soopies Hoogte	A galloway of little or no promise; a pronounced roarer.	Inspiratory sound harsh. Never won or ran prominently. Died within a year following operation.	This horse showed no improvement although his inspiratory sound was much improved.
Death Ray	Good horse, won several races and then became useless as a result of pronounced roaring.	Inspiratory sound harsh. Won races and ran second in a Merchants' handicap, close up to one of the leading horses in the country. Died of horse sickness after being retired to stud.	The operation caused a wonderful improvement, and brought the horse back to his previous best form.
Ponell	A very good horse, won several races. He became useless as a result of pronounced roaring.	Quite sound of his wind. Won several races, over middle distances. Continued to race for 4 years following operation.	This horse was fired on his knee (N.F.), castrated and ventriculectomised on the same day. He was a robust horse and was not seriously affected by concurrent surgical operations.
Reveller	A bad horse. It is not known when he developed roaring, but he was a very pronounced roarer when examined. He could not gallop a furlong without great distress.	Inspiratory sound harsh. Only ran twice following operation, did not win a race or run prominently.	This horse showed great improvement in that he could finish a race, but he never won.
Masterful	A bad horse, never won a race. Later became a pronounced roarer and useless for racing.	Quite sound of his wind. Won two races and finished second in another, but his form although improved was not good.	This horse was greatly improved by the operation.
Bystander	A useful 2-year-old. Later developed roaring and became useless.	Quite sound of his wind. Won races subsequently.	The horse was greatly improved, but did not fulfil his 2-year-old promise.
Camelon	A bad horse, never showed promise. Later developed roaring and became useless.	Inspiratory sound slightly coarse. Never won a race although he ran in several middle distance races and finished with the horses.	This horse was greatly improved by the operation.
Portadown	Not a good horse, won one race. Got a lumbar injury as a three-year-old.	Quite sound of his wind. Won several races in modest company.	This horse was greatly improved by the operation.

TABLE I. (Cont.)

Name.	Previous History.	Subsequent History.	Remarks.
Faultless	A good horse, won a race as a 2-year-old and then developed roaring. He became a rogue as a result of being pushed in races when he was distressed.	Quite sound of his wind. Never won a race because he would not try. He was not genuine and put no effort into his work.	This horse was quite sound following operation, but his previous experience on the race course had soured him and he would not gallop. He showed good form in trials, but would not exert himself in races.
Denrose	Well-bred colt, but was quite useless on account of pronounced roaring. He could not stay a furlong.	Quite sound in his wind. Won two races, the last over 10 furlongs. Still in his prime.	This horse was greatly improved by the operation.
Roulette III	A well-bred horse. Never won a race. It is unknown when he developed roaring.	Quite sound of his wind. Won two races and ran prominently in others. Put to stud.	This horse was greatly improved following operation.
Roy Cherry	A well-bred horse, never raced. A very pronounced roarer and quite useless.	Quite sound in his wind. Suffered an injury and has not yet run. Still in his prime.	Young horse still in training. Owner states he shows great promise.
Stormount	A good horse, won two races. Developed whistling as a 2-year-old and could not stay.	Inspiratory sound slightly coarse. Has run once, but has not won.	Young horse. Still in training. Has not yet regained his original form. Trainer says his stamina is much improved.
Yankee Pro	A useful horse, won three races and then became a pronounced roarer and could not stay. He was raced several times while unsound.	Inspiratory sound slightly harsh. This was an old horse when operated upon. He has run twice but has not won.	Aged horse still in training. Has not yet gained his original form. His trainer says his stamina has improved.
Rameau	A useful horse, won one race as a 2-year-old. Later developed roaring and could not stay.	Quite sound of his wind. Won two races subsequent to operation. Still in his prime.	This horse was greatly improved following operation.
Waterproof	Well-bred horse, never showed any form. A highly excitable temperament.	Quite sound in his wind.	Young horse and still in training.

Fig. 1.

(a) (b)



1. — *Shows the larynx of a horse which had been experimentally ventriculectomised:*

(a) *Lateral ventricle completely closed and vocal cord atrophied.*

(b) *Lateral ventricle and vocal cord of the unoperated side.*

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NOTE.

Members who send papers for publication in the Journal are asked to read the instructions on the last page of the cover of the Journal.

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Can Hares and Rabbits Act as Hosts of Sheep and Goat Bankrupt (*Trichostrongylus spp.*) Worms in South Africa?

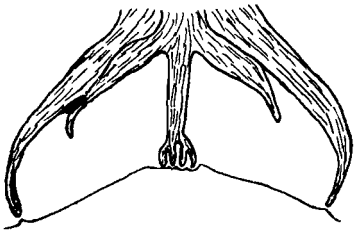
By R. J. ORTLEPP, Onderstepoort.

In connection with the zoological survey of South Africa, the Department of Zoology of the University of Stellenbosch recently consigned to this Institute the duodenal scrapings of two typical Cape Grey Hares. An examination of this material revealed the presence of about 20 mature trichostrongyles from the one hare, and over a hundred similar parasites from the other hare. These specimens the writer considers to be co-specific with *Trichostrongylus pietersei* le Roux, 1932, originally obtained from sheep and goats. This finding is the first South African record of a member of the *Leporidæ* acting as a natural host for the same *Trichostrongylus* sp. normally parasitic in sheep and goats. On consulting the literature a few records were found wherein sheep trichostrongyles are recorded from rabbits. Thus Skidmore (1932) records the occurrence of *T. colubriformis* in American rabbits and Mönnig (1938) lists the rabbit as a host for *T. vitrinus*. Hall (1916) found *T. colubriformis* to parasitise American squirrels, and Baylis (1934) also found this species in English squirrels. Leiper (1937) recovered the typical rabbit trichostrongyle — *T. retortæformis* — from goats in England. From these records it becomes clear that the occurrence of a sheep trichostrongyle in South African hares is not such a great rarity as one would at first think, and the question arises in how far these animals can act as natural reservoirs for these sheep parasites.

In order to throw some light on this question the writer decided to infect rabbits at weekly intervals with trichostrongyle larvæ from sheep. Three rabbits, which had been reared in captivity in wire cages and whose fæces showed no helminth eggs, were used, and each received orally a small amount of a rich culture of trichostrongyle larvæ from incubated fæces of a sheep. As the sheep was not slaughtered it was not possible to determine with what species the sheep was infested. At the commencement of the second and third weeks each rabbit again received about the same amount of larvæ from the same culture. The day after the third feeding, that is, 16 days after the initial feed, one of the rabbits died, and on post mortem numerous young adult trichostrongyles were recovered from the duodenum; these all proved to be *T. colubriformis*. The remaining two rabbits appeared healthy and were given a fourth feed at the commencement of the fourth week. Two days later, 23 days after the initial feed, one of the rabbits was killed. Numerous adult specimens of *T. colubriformis* were recovered from the duodenum, and most of the females contained

several segmented eggs *in utero*. Examination of the fæces of the remaining rabbit showed that trichostrongyle eggs were present. These findings showed that *T. colubriformis* easily sets up an infection in rabbits, and that it develops to maturity in this host in about the same time as in sheep

Fæces from the remaining rabbit were now collected and incubated in order that larvæ could be obtained to set up an infection in sheep. Fortunately, while the larvæ were developing, two of the writer's experimental ewes lambed, and these lambs were placed in a wire-screened box and only allowed to suckle under supervision on a cement floor so as to prevent any possibility of their acquiring a natural infection. In the meantime trichostrongyle larvæ appeared in the fæces culture of the rabbit, and about 200 of these were drenched to the one lamb, the other lamb being kept as a control. On the twenty-third day after infection the fæces of the infected lamb showed the presence of trichostrongyle eggs, whereas those of the control lamb remained negative. A week later, 30 days after the



commencement of the experiment, both lambs were slaughtered. A fair number of *T. colubriformis* were recovered from the duodenum of the infested lamb, whereas a careful examination of the abomasum of the control lamb showed that trichostrongyles were absent. Further, fæces of the latter lamb showed no trichostrongyle eggs. There can thus be little doubt that the infection set up in the one lamb was caused by the larvæ recovered from the rabbit's fæces, and not from a natural infection.

Summarising, we have then shown that *Trichostrongylus colubriformis* larvæ recovered from sheep can readily set up an infection in rabbits, that these parasites easily grow to maturity in this host and are able to produce eggs whose larvæ are capable of setting up an infection in sheep.

What inferences are to be drawn from these results? Are rabbits and hares to be regarded as a potential danger to the sheep-rearing industry, and must their extermination or control be resorted to in order to combat

effectively this parasitic disease? In South Africa, where hares are relatively very scarce, the writer does not think that these results are of any more than academic interest. In countries, especially in sheep rearing countries, where rabbits may be abundant, rabbit reservoirs of infection have to be seriously considered in any campaign for the effective control of these parasites.

Remarks on Trichostrongylus pietersei *le Roux, 1932.*

The above nematode was described by le Roux (1932) from material collected from black-head Persian sheep imported from Norval's Pont, Cape Province. An examination of sheep and goat material from Willowmore, Cape Province, also revealed the presence of this parasite. Further examination of material from various parts of the Union led le Roux to conclude that this species was "present only in sheep and goats on the arid central plateau, known as the Karroo, or in animals originally imported from that area not many months prior to the collection of the material."

The writer has recently re-examined all the trichostrongyle material collected at various times from sheep and goats at Onderstepoort and stored in the departmental collection; this examination revealed the presence of only a single male specimen of the above species. The containers unfortunately give no data from which area of the Union the hosts originally came prior to postmortem examination at this institute. In addition to this material the writer has also examined trichostrongyle material specially collected for him from different Karroo areas; no examples of *T. pietersei* were present in any of these materials.

The following data were obtained from a study of the hare material, which was unfortunately considerably shrunken. Length, males 4·2 to 5 mm., females 4·3 to 4·8 mm. Diameter head 0·01 to 0·011 mm. for males, 0·011 for females. Excretory pore in slight depression 0·12 to 0·148 mm. from anterior end. Bursa typical for genus with stout dorsal ray (fig. 1) showing four well defined terminal digitations. Spicules (fig. 2) similar to those figured by le Roux, being unequal and similar; left 0·148 to 0·167 mm. long; right 0·141 to 0·155 mm. long; distal hook 0·039 to 0.044 mm. from tip in right spicule and 0·033 to 0·044 mm. from tip in left spicule; proximal hook 0·063 to 0·072 mm. from tip in right spicule and 0·066 to 0·074 mm. from tip in left spicule. Gubernaculum 0·075 to 0·078 mm. long. Female tail 0·072 to 0·096 mm. long similar in shape to that found in other members of this genus. Vulva a longitudinal oval aperture, 0·05 mm. by 0·03 mm. wide situated 1·02 to 1·08 mm. from posterior extremity. Ovejectors directly opposed, their combined lengths 0·528 to 0·576 mm. Eggs thin-shelled, oval and morulated *in utero,* 0·078 to 0·084 mm. long by 0·042 mm. wide.

Host: *Lepus saxatilis saxatilis.*
Habitat: Duodenum.
Locality: Stilbaaistrand, Cape Province.

The above description differs in some respects from le Roux's in that the males are smaller, which may be due to different methods of killing and fixing; the spicules are slightly longer, being 0·129 and 0·137 mm. long for the right and left spicule respectively in le Roux's material. However, the male specimen recovered from sheep material at Onderstepoort had spicules of the same length 0·15 and 0·137 mm. as in the writer's hare material, and it would thus appear that the spicule lengths are capable of considerable variation. The gubernaculum in both the writer's hare and sheep specimens was practically of the same length, and somewhat smaller than le Roux's measurement (0·098 mm.). The writer, however, does not attach much importance to these differences, and does not consider them to be of specific significance.

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A Skin Condition seen in Rhodesian Ridgeback Dogs: Report on Two Cases.

H. P. STEYN, J. QUINLAN and CECIL JACKSON, Onderstepoort.

During January, 1938, two Rhodesian Ridgeback pups were imported from Salisbury, Southern Rhodesia, to Pretoria District. At the time of their importation Mr. H. Kingcome, M.R.C.V.S. wrote to Dr. du Toit, the owner of one of the pups, warning him that the breed is subject to a peculiar " skin growth " which he called " Trichiasis spiralis." He pointed out that one of the pups showed signs of developing the condition, and that the only successful method of treatment was to remove the lesions surgically. The treatment, he said, had to be undertaken early otherwise considerable difficulty would be encountered in bringing about a cure. He also briefly described the technique he adopted in removing the lesions.

Case 1. — The first pup, a male about 4 or 5 months old, was admitted for treatment on the 18th March, 1938. At the time of admission there was one small abscess situated in the mid-dorsal line near the base of the neck. A second fairly large abscess was situated in the mid-dorsal line between the anterior angles of the scapulæ, and a third immediately behind this. These were opened and drained as thoroughly as possible. The situation of the abscesses, however, made it impossible to create counter-openings to ensure thorough drainage, and one had to be satisfied with daily cleaning and treatment on general principles.

A few days after these lesions had been opened, a peculiar cord-like development extending downwards from each abscess cavity towards the spine was noticed, and it was then realized that this was the condition described by Mr. Kingcome. In addition to this cord-like development beneath each cavity, these abscesses differed from the ordinary abscess in that there was a persistent marked painfulness to manipulation, and that several days after opening the abscesses tufts of hair could be found in the depths of each lesion.

On the 19th April the dog was operated upon for the removal of the old abscess cavities with their cord-like attachments. The lesions were well advanced and removal proved exceptionally difficult. It was found that the " cord-like structures," which have been described, consisted of tubular growths, somewhat funnel-shaped, with the narrow ends extending down to the spine. These tubular growths were lined by epithelium and contained matted tufts of hair and sebaceous matter. There were three structures below the most anterior cavity and one each below the remaining two cavities.

The after-treatment proved to be very difficult, as proper drainage could not be ensured and healing was protracted. Healing was almost complete when a recurrence of the condition took place. The dog was operated upon a second time on the 7th June. Lesions exactly similar to those already described were again present, except that the tracts were now narrower and more typically of a fistulous nature. Healing was even more protracted than before, and the dog was not fit for discharge from hospital before the 5th September, very nearly six months from the date of admission.

On the 17th December, 1938, the dog was brought back with an apparent recurrence of the condition. Under treatment a large abscess developed at the site of the most anterior lesion. This was opened dorsally in the midline and counter openings were made on either side of the neck and setons introduced. Healing was again slow and the dog was discharged on the 20th March, 1939.

There has been no further recurrence up to the present, and one may assume that a permanent cure has now been effected.

Case 2. — The second case, a litter brother of the former, was admitted for treatment on 11th July, 1938, showing marked sensitiveness in the sacral region, immediately anterior to the root of the tail. Examination revealed two small apertures close together in the skin in the midline of the sacrum. These apertures appeared to be enlarged hair follicles with a number of matted coarse hairs protruding from each, and under each a cord-like structure about half the thickness of a lead pencil extending down towards, and apparently attached to the spine.

On the 12th July, 1938, these lesions were removed operatively. They were similar to the previous case, but very much smaller. The more anterior lesion was very successfully removed. It was found to extend through the subcutaneous tissue down to the bony sacrum and after all attachments of soft tissue had been dissected away from it, as far as this was possible, it was pulled free by steady firm traction. It is impossible to state definitely how far the lower end penetrated but it seemed to have extended down into some interosseous space in the sacral region. The second lesion was removed as thoroughly as possible, but its course could not be followed as well as that of the first. The operation wounds were sutured, as there was no evidence of infection, and healing took place rapidly by first intention.

This dog has not had a recurrence of the condition, although he has developed very marked signs of sensitiveness in the affected region on two or three occasions. One, however, feels justified in claiming a permanent cure in this case.

Operative Technique. — The technique is essentially simply in principle, but one or two points of especial significance should be mentioned.

It is advisable to incise around the externally visible lesion and not

through it. The external orifice of the lesion can then be conveniently grasped in a pair of artery forceps and the cord beneath it drawn taut so that one is able to follow its course more readily. In dissecting away the cord great care must be exercised not to incise it, as once this has been done it becomes extremely difficult to follow its course. In addition the interior of the tubular cord is a potential source of wound infection and incising this structure therefore reduces the likelihood of healing by first intention.

Histology of Excised Lesion. — Transverse sections were cut at various levels. They show (see figure) that the structure removed is a thick-walled, hollow tube composed chiefly of fibrous tissue and lined by squamous stratified epitthelium. The junction of this epithelium with the underlying connective tissue is smooth; papillæ are rare. In the surrounding connective tissue hair follicles are embedded which open through the lining epithelium and contain hairs, the shafts of which extend into the lumen of the tube. Connected with these follicles are sebaceous glands. More peripherally occur sweat glands. This connective tissue is bounded peripherally by subcutaneous tissue.

In the lining epithelium, stratum basale, stratum squamosum, stratum granulosum, and stratum corneum are readily distinguished. The last is well-developed, so that a hyperkeratosis exists. A similar hyperkeratosis affects the hair follicles. The latter are usually compound, transections of half-a-dozen or more hair-shafts being seen. The contents of the lumen of the main tube consist of scaly debris desquamated from the stratum corneum, and towards the superficial extremity of the lesion this has undergone partial calcification. In addition numerous hair shafts of very variable diameter are present in the debris. Most of these exhibit a distinct cuticle, pigmented cortex, and well-developed medulla; in some the medulla is absent.

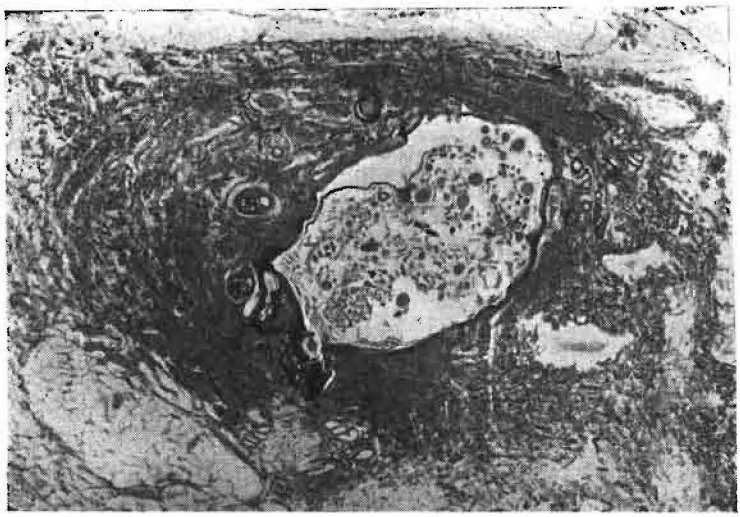
One of the sections made discloses a diverticulum of the main cavity where the connective tissue is hyperæmic and infiltrated by leucocytes, chiefly neutrophiles, which migrate through the epithelium and accumulate so as partially to plug the lumen with inspissated pus. This diverticulum contains a hairshaft and doubtless represents an inflamed hair-follicle. A section taken from the deepest portion of the lesion shows that at this level the accessory structures — follicles and glands — are no longer present.

Discussion.

Hieronymi (1924) defines dermoid cysts as lying deep in the subcutis and having no connection with the epidermis. Although the lesion under consideration has all the histological features of a dermoid cyst, it does not appear possible to describe it by the term "cyst," since in addition to having an elongated tubular shape it opens to the exterior and is in continuity with the epidermis. The lesion is, therefore, surgically a sinus and not a cyst.

For these reasons it is suggested that the lesion be described as a "dermoid sinus." However, we must emphasise certain characteristics of these lesions which cannot be considered as typical of a sinus. These are that the lesions apparently have a tendency to grow, are very painful, and that although a small amount of discharge matting the hairs at the opening of each sinus is noticeable, yet suppuration is very slight. In a recent case (e.g. case No. 2) the main symptom may be painfulness, and only careful examination either by parting or clipping the hair will reveal the lesion.

We are unable to state that abscessation, such as that seen in case No. 1, is a common occurrence, but this complication effectively disguises the



TRANSVERSE SECTION THROUGH LESION.

Note squamous stratified epithelial lining, deficient at one part and showing hyperkeratosis. Outside this, connective tissue representing the corium and containing hair-follicles, two of which (left, below) are transected at or near their opening into the main cavity; sebaceous glands (e.g. associated with the hair follicles at the right) and sweat glands (further to the right and below). Outermost the fatty subcutis which contains the lesion. Numerous hair shafts (cut transversely) and the debris of keratinised epithelium occupy the lumen. 30 X. (Path. No. 21595).

real nature of the lesion and a correct diagnosis is only likely to be made during the after-treatment of the abscesses.

The term "Trichiasis spiralis" mentioned by Mr. Kingcome, which appears to have gained a certain currency in Rhodesia and even in the Union, seems to us to have some drawbacks. Trichiasis is a condition of ingrowth of hairs from their distal ends, that is, a retroversion. No such phenomenon is operative in the disease under consideration. Further, when coupled with

the adjective "spiralis" (doubtless used to express an intertwining or matting of the hairs protruding from the orifice of the lesion — a feature which is of very minor importance) it would seem liable to confusion with the condition Hypotrichosis cystica (German: *Schrotauschlag;* also *Rollhaar-cysten*) of which a spiral contortion of congenitally imprisoned hairshafts within their follicles is a leading characteristic. Lastly, a superficial resemblance to the name of a well-known parasite (*Trichinella spiralis*) might prove embarrassing to students and perhaps even to practitioners.

The indications from the cases examined by us — the early age at which the lesions became clinically apparent, the sites of occurrence along the mid-dorsal line of the body, and the fact that the disease is, since it has not been previously described, apparently confined to the Ridgeback breed — incline one to the assumption that the condition is congenital in origin and indeed that an hereditary influence is a determining factor in its occurrence.

SUMMARY.

Two cases of skin lesions in the Rhodesian Ridgeback dog have been described. From the histology and clinical nature of the lesion, which is of an unusual type, the name "dermoid sinus" has been suggested. Observations on the treatment and diagnosis of the condition have been made.

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A Case of Myiasis Produced by *Chrysomyia marginalis* Wiedemann.

By H. O. MÖNNIG, Onderstepoort.

The available records of *C. marginalis* as a cause of myiasis in domestic animals are few and rather vague. In South Africa it is a very common fly, but it has not been found to infect wounds on sheep, although it has always been looked upon with suspicion and there are indications that it may be in the process of evolution as a parasite, or myiasis-producer.

C. marginalis, commonly known as the "large bluebottle," is a relatively large fly of a blue, sometimes a bottle-green colour and with dark bands across the body at the borders of the segments, especially on the abdomen. The eyes are coppery-red, contiguous in the male and separated in the female by a light yellow area. The anterior margins of the wings are conspicuously black.

Patton (1921) states "*Chrysomyia marginale* is a common African

species, and though mainly breeding in decomposing bodies of animals and birds, will occasionally lay its eggs in, or near, diseased tissues." The same author (1922) writes: "*Chrysomyia marginalis* Wied. — This is essentially an African species, being distributed from the North to the South; Austen records it from Quetta. I have not seen any species from India. Its larvæ occasionally cause cutaneous myiasis, much in the same way as do the larvæ of *megacephala.* It normally breeds in decomposing animal matter."

Curson (1924) states in connection with "infection of wounds by blowflies," that Patton examined material sent from Zululand and identified, *inter alia, C. marginalis.* In none of the above cases is it mentioned from what species of animal the larvæ were taken and under what circumstances. Myiasis due to this fly is apparently comparatively rare and may occur only on certain species of animals and under particular conditions.

In regard to sheep, Smit (1929), who collected and identified numerous specimens of maggots from wounds on sheep all over the Union, states: "The large bluebottle, *Chrysomyia marginalis,* which is so common all over the country, is often thought to be a sheep blowfly, but up to the present its maggots have not been taken from live sheep . . . It breeds in carrion very readily, where its eggs are laid in huge white masses, often by several females working together. As its maggots are active agents in devouring carrion that would otherwise feed the sheep maggot-flies, it may be considered a beneficial fly, but because it is so closely allied to the green- and -blue sheep blowfly and to the banded blowfly, and belongs to the same genus, we feel bound to look upon it with suspicion and should not be surprised to find it attacking sheep at some future date."

The object of this note is to report on a case of myiasis in a calf, in which *C. marginalis* was concerned.

The calf was running in an experimental paddock at Onderstepoort and was suffering from sweating-sickness, a condition in which there is a hyperæmia of the skin with copious perspiration. The calf was seen at 3.30 p.m. on the 27th January, 1939, and numerous small maggots were discovered on various parts of its body. The calf was placed in a stable later in the afternoon and died there overnight. The next morning at 9 a.m., at the post-mortem examination, numerous maggots, all in the second larval stage, were found distributed over the body, mainly in the flanks and along the ventral aspect of the abdomen, where the skin was reddened and moist, but no wounds were present.

The maggots were allowed to develop in meat and later to pupate in sand. On the 6th February, adults of *C. marginalis* began to emerge and several days later adults of *C. albiceps* and *Lucilia sericata* also appeared. The *C. marginalis* adults were kept in a cage in the laboratory and supplied with sugar-water and meat, but no eggs were laid and the flies began to die 22 days after having emerged. During this period maggots were taken

from several calves and sheep with infected wounds, but no *C. marginalis* was found in these instances.

The above case indicates that it is desirable to note carefully the condition under which maggots occur on animals, where such maggots are collected for indentification, in order to obtain a clear impression of the conditions which will attract flies of the type of *C. marginalis.*

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A Rickettsiosis New to South Africa.

By K. SCHULZ, Onderstepoort.

The object of this preliminary note is to record the occurrence of *Rickettsia ovina* in the monocytes in blood, intima, and lung smears of two sheep sent in during January, 1939, by the Government Veterinary Officer, Mr. J. G. de Wet, District Grootfontein, South-West Africa.

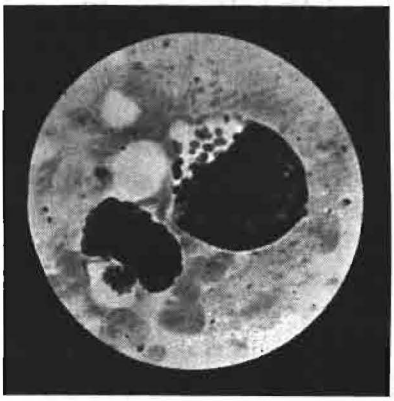
The rickettsiæ were more prevalent in the smears of one animal than in those of the other. A field containing two affected cells is reproduced below.

It may be of interest to mention briefly the observation recorded by Mr. de Wet regarding the above outbreak.

Severe losses, confined to the sheep only — cattle and goats not being affected — have occurred on this farm over a period of about 12 months. The symptoms appear very suddenly and animals which had a normal appearance the previous evening are found dead the next day. In fact a number of cases are noticed to ail only for about two hours prior to death. Not a single animal suffering from this disease has yet recovered. The animals lie on their side with outstretched legs, and head turned backwards, and appear unconscious. The eyes are protruberant and appear glassy. A febrile condition is suspected.

At autopsy ticks were numerous on all sheep examined, but no heartwater transmitting ticks could be found. The changes noted on post mortem simulated those of heartwater; namely hydropericardium, hydrothorax, ascites, subepicardial, and subendocardial hæmorrhages, hyperæmia and oedema of the lungs, tumor splenis with prominent Malpighan bodes, soft and pulpy kdneys. To exclude this disease, the hippocampi of the above two sheep and those of other animals sent in at the same time were examined for *Rickettsia ruminantium,* although the veterinary officer stated that no heartwater ticks were found on the sheep in that vicinity. The result of the histological examination of this material was, however, negative for heartwater.

The former Government Veterinary Officer of Grootfontein, Dr. Sigwart, described similar symptoms in sheep on the same farm, but in



From Blood smear of sheep, No. 416. Path. number 22544. Magnification 1250X.

addition mentioned that paralysis of the hind-quarters and opisthotonus were seen in some of the sheep shortly before death. He applied the vest pocket test for cyanide poisoning with negative results. The presence of arsenic could not be demonstrated in the material sent to Onderstepoort for that purpose. He found numerous blue ticks *(Boophilus decoloratus)* and bontleg ticks *(Hyalomma egyptium)* on the sheep and stated that the veld on this farm was dry and showed signs of overstocking. In addition a fairly marked verminosis (wireworms, tapeworms and nodular worms) was recorded in some sheep. A blood smear sent in by him proved to be negative on microscopical examination and in the brain material of affected sheep the causal organism of heartwater could not be found.

From the above it would appear that the heavy mortality among the sheep cannot be attributed to a rickettsia infection alone, but possibly also to contributory factors such as verminosis, tick infestations, and nutritional disturbances.

Much to our regret, as no further cases occurred by the time the diagnosis was made, it was impossible to investigate the outbreak further.

Lestoquard and Donatien (1937) described *Rickettsia ovina,* a parasite of the monocytes of the blood of sheep, in Algiers and Anatolia. They were able to exclude anthrax, piroplasmosis, and pernicious anæmia as possible causes of the mortality. By inoculating bone-marrow and blood of affected sheep subcutaneously into susceptible ones, a febrile reaction was produced and *R. ovina* was demonstrated in the peripheral blood. Based on the results of their experiments they suggest that *Rhipicephalus bursa* is the vector of this rickettsia.

As *R. ovina* occurs exclusively in the monocytes, and presents a morphology similar to that of *R. canis* and *R. bovis,* and as it is often frequent in the peripheral circulation, it can be easily differentiated from *R. ruminatium,* the causal organism of heartwater.

It may be mentioned here that my colleague Mr. W. O. Neitz was good enough to show me a preparation in which he had already found *R. ovina* in a blood smear of a sheep from the Brits area in the Transvaal prior to the arrival of the material from South-West Africa. As this was a single case he refrained from publishing it.

Obviously no research can be undertaken on this new condition until active outbreaks are discovered from which further material, including live ticks, can be obtained. This brief note is therefore published in the hope that veterinarians in the field who encounter similar conditions will report them to enable further investigations to be made.

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An Unusual Case of Strongylosis

By C. C. WESSELS, Government Veterinary Officer, Bredasdorp.

Although strongylosis with its usual complication is encountered in the Western Province, where horse breeding is still carried out on a fairly large scale, the unusual acute infection described here is worthy of record.

Subject. — A thoroughbred brown filly 9 months old belonging to a well-known breeder of thoroughbreds.

History. — At 7 a.m. when the animal was fed she appeared to be normal. At 9 a.m. she was reported to be off colour with symptoms of inappetance and general depression. At midday it was noted that there

was abdominal distention. The symptoms of depression and dullness became progressively worse with the animal swaying from side to side until 6 p.m., when she collapsed and died. The next morning I was called in to perform a post mortem.

Autopsy. — There were approximately 8 gallons of blood in the peritoneal cavity. On the parietal peritoneum in the xyphoid region there was a hæmorrhage 9" x 9". It was impossible to determine which vessel was involved as the tissues were permeated by the blood clot. When a cut was made into the clot large numbers of live strongylus larvæ oozed out. Surrounding the border of the hæmorrhage there was marked serogelatinous infiltration which, on section, also contained similar larvæ.

Further examination of the parietal peritoneum revealed hæmorrhagic cysts varying from 0·5 to 2 inches in diameter. In each of these cysts one or several larvæ were found. These cysts were found throughout the peritoneal and pelvic cavities and also involved the visceral peritoneum. In some places larvæ *(S. edentatus)* could be found under the serosa without the presence of a hæmatoma and when these foci were cut out there was slight exudation of purulent matter.

Some of the external iliac and mesenteric lymph glands revealed suppuration. The anterior mesenteric artery at its origin was the seat of an aneurism which was filled with a crawling mass of strongylus larvæ. A portion of the ileo-cæco-colic artery which was dissected out also contained similar larvæ. The latter artery showed sausage-like dilations (Aneurysma fusiforme) and in parts the lumen was almost obliterated and so blocked with the larvæ that it was difficult to open up the artery with pointed scissors. The gastro-intestinal tract also revealed a few adult strongyles. This could be expected from the age of the animal and the fact that most of the larvæ had not yet completed their life cycle.

Of significance here is that in several places in the intestine, especially the large gut under the mucosa, there were nodules varying in size from a pea to a walnut. These nodules on section revealed fresh blood clots in which strongyles could be found. These were probably nests of the larvæ after completion of their life cycle; from such foci they again escape into the lumen of the gut where they become sexually mature.

Diagnosis. — Internal hæmorrhage due to rupture of a blood vessel caused by strongylus larvæ.

The owner further informed me that some time ago he also lost a colt which at times showed periodic attacks of colic. On post mortem he had noticed inflammation only in the vicinity of the kidneys but when he had cut out the bowels he noticed masses of larvæ floating around in the abdominal cavity. However, he did not attach much importance to the worms as he thought they might have escaped from the gut. He also found a large quantity of blood in the abdominal cavity and therefore he suspected gut-tie; but no further evidence of this was found.

In my opinion this animal died from a similar condition.

Practically all the yearlings on this farm have spasmodic attacks of colic from time to time. They are unthrifty, in spite of excellent care and attention from a dietetic point of view. Further the grazing on this farm consists of marshy pastures where conditions are very favourable for strongylosis.

Conclusions. — The animal described was grossly infected with strongyle larvæ which caused aneurysms in some of the arterial trunks. The larvæ damaged the wall of an artery in the xyphoid region to such an extent that it ruptured and thus led to the death of the animal. Larvæ forwarded to Onderstepoort were identified as *S. vulgaris* and *S. edentatus.*

Jl. S.A.V.M.A.
X(4): 180—181
1939.

A Field Experiment in the use of Prontosil for the Treatment of Horse Sickness.

By WAKEFIELD RAINEY, Bloemfontein.

There is no intention, in submitting an account of the following small experiment, to claim that prontosil will cure horse sickness: what happened as the result of injecting prontosil into field cases of sub-acute horse sickness may have been pure coincidence. The experience, however, was so striking that it should be recorded in order that the experiment may be repeated by others.

An outbreak of horse sickness occurred in a large stable of polo ponies and hacks, and in the paddocks adjoining the stable, near Bloemfontein late in May and early in June, 1939. Some frost had been recorded and the disease was not of a very acute nature.

In all, nine cases were observed of which seven died and two recovered. Of the nine cases, four only received two injections each of 15 cc. of a five per cent. solution of prontosil at an interval of eight hours. All the five cases that did not receive injections died within 72 hours of illness being observed.

Of the four treated cases, two recovered rapidly and completely: one case recovered at the time, but had a relapse and died four days later after fast work to which a native groom had put the animal, in the belief that it was fully recovered. The fourth treated case was already *in extremis* when injected.

Of the two cases which recovered, one was down in the camp when discovered and unable to rise, with a temperature of 103°, at 2 p.m. By 8 p.m. that evening it had recovered its strength, the temperature was normal, and it was back in the stable feeding.

The writer observed two almost identical cases at 8 p.m., both with a temperature of 106° and both evidently somewhat distressed. One was given an injection of prontosil and the other was not. The injected horse was normal next morning and never "looked back." The uninjected horse still had a temperature of 106° the next morning and died twenty hours after treatment.

It has been said that the outbreak was not of an acute nature and in support of that statement it should be noted that each of the animals that was attacked — with the exception of one Shetland pony found dead in the camp — had been subjected, in the early stages of infection, to more or less vigorous work.

In each case the same history was given of noticeable "dullness" at work on one or two days before the cases were taken seriously, and before the writer found, on examination, a temperature of 106°. Advice regarding routine temperature taking, given after the first case, was, as usual, ignored. It seems likely that if the affected animals had not been worked in the early stages typical horse sickness would not have appeared, with the stated exception of the Shetland pony. Consequently the value of prontosil, if any, in the treatment of horse sickness should not be over-estimated, as the drug may have no curative effect in an ordinarily severe outbreak of the disease or in experimental laboratory cases.

The members of the polo club are, as might be expected, firmly convinced that prontosil saved the lives of those animals that recovered, and that those untreated animals that died would not have died if prontosil had been injected.

The writer, of course, is not convinced, but he confesses to having been impressed by the dramatic drop in temperature of each of the animals that was injected as compared with the persistent high temperature of those that did not receive the injection. But pending further experiment and reports by colleagues elsewhere, he would not have the moral courage, on a further occasion, to abstain from the use of prontosil or to advise an owner that prontosil had no effect on the course of sub-acute horse sickness.

OBITUARY.

James Robert Roe Hamilton, M.R.C.V.S.

We regret to announce the death of another old member of the Association. James Robert Roe Hamilton was a native of County Roscommon, Ireland. He studied at Trinity College, Dublin, and later went ranching in North Dakota. He gave up ranching after six years and entered the New Veterinary College, Edinburgh, where he qualified in 1894. Towards the end of the Anglo-Boer War, he joined the civil veterinary surgeons attached to the British

forces and thereafter transferred to the Veterinary Service of the Orange Free State.

The last nine years of his service with the Union Government were spent in Durban where he retired in 1924. He was of a genial, kindly disposition and was a great lover of animals. To his wife we tender our sympathy in her loss.

J. S.

THE ASSOCIATION.

Council Meeting held at Carlton Hotel, Johannesburg, 23rd August, 1939.

Present:— S. T. Amos (President), C. J. v. Heerden, P. J. du Toit, R. Alexander, D. G. Steyn, C. Jackson, A. C. Kirkpatrick, J. Quin, and S. W. van Rensburg (Hon. Sec.).

Apologies for absence from: A. M. Diesel, H. H. Curson, H. O. Mönnig, A. S. Canham, A. D. Thomas, and J. G. v. d. Wath.

The Secretary explained that Dr. Quin had been asked to attend in view of the discussion on hormone treatment.

1. *Minutes* of the meeting held on 4th April, 1939, having been circulated, were taken as read and were confirmed.

2. *Arising from these:*

(a) *Arrears.* — The Secretary informed the meeting that a member whose case was considered at the last meeting, had since died. It was decided that his arrear subscriptions be written off.

(b) *Dr. Schulz.* — A letter dated 7th July, 1939, was read, in which this member thanked the Council for the assistance received from the Association in pressing his claims for compensation, and made a donation of £12 12s. 0d. towards the Benevolent Fund as a mark of appreciation. The Secretary stated that he had already thanked Dr. Schulz for this donation.

The meeting decided that the legal expenses (£3 3s.) incurred in presenting Dr. Schulz's case to Parliament be paid by the Association.

(c) *International Veterinary Conference.* — Dr. du Toit stated that nothing further had been heard regarding the payment of expenses.

(d) *Journal.* — The meeting was informed that no alteration would be made in the publication of the Journal.

(e) *Advance.* — It was decided that the balance of £4 10s. still due by the widow of a late member be written off.

(f) *Permanent Force.* — Dr. du Toit reported that further progress had been made with the veterinary military organisation and it was hoped that he would be able to give full details to the next meeting.

(g) *Footner Memorial.* — The President reported that about £55 was received, which would be adequate for erecting and maintaining a headstone.

3. *New Members.* — On Dr. du Toit's proposal it was unanimously decided to recommend acceptance by the General Meeting of the following new members: C. F. B. Hofmeyr, P. R. Mansvelt, A. J. McGilvray, D. T. Mitchell, G. C. Muller, Miss M. R. Silverman, N. C. F. Steenkamp.

4. *Clerical Assistance.* — The Secretary intimated that it was not possible to get a suitable assistant to do the clerical work and typing at the present rate of remuneration. The meeting accordingly sanctioned an increase to £3 10s. per month.

Auditing. — The Secretary reported that Mr. J. H. van Wyk, who audited the books during the past two years, had suggested that Council consider the advisability of getting a chartered accountant to audit in view of the trust money held by the Association. During the subsequent discussion various members expressed satisfaction with the manner in which the books were audited, and it was decided not to make a change at present. On the President's suggestion it was decided to pay a gratuity of £5 5s. for auditing.

Office Accommodation. — Dr. du Toit and the Secretary were deputed to consider the possibility of obtaining suitable office accommodation.

5. *Hormone Treatment:*

In introducing the subject the President stated that the alleged beneficial effect of certain hormones has opened up a wide field which might be exploited by unscrupulous persons, and that this matter is causing grave concern to the Jockey Club of S.A. He read a copy of his letter of 13th June, 1939, to the Durban executive of the Club and of 13th May, 1939, to Messrs Roberts & Co., New Bond Street, London. The latter replied that no information could be obtained from the highest veterinary authorities in Great Britain, and that even Major Reynolds, Veterinary Surgeon of Newmarket, denied all knowledge of any beneficial effects from gland treatment in race horses.

The Secretary read the correspondence with the Jockey Club and an article published in an overseas periodical.

Dr. Quin then addressed the meeting, giving a detailed explanation of the action and effects of certain hormones. He indicated that it was exceedingly difficult to separate the wheat from the chaff, and that this treatment lends itself to wide abuse in the hands of ignorant people. He considered that there was need of further investigations by means of properly conducted experiments.

Important points that were raised in the subsequent discussion were that this treatment was apparently already being applied by unscrupulous persons, that the continued use of hormones is likely to cause serious damage to the body, that the Jockey Club should sponsor the suggested experiments, and that when hormone treatment of animals was necessary it should only be done under strict veterinary control.

On a suggestion from Dr. du Toit, a committee consisting of Drs. Quin, Quinlan, Alexander and the Secretary, was appointed to draft a reply to the Jockey Club. Copies of this, marked "Confidential," first to be submitted to members of Council.

The President thanked Dr. Quin for coming over and supplying the meeting with the desired information.

6. *General:*

(a) A letter dated 15th August, from the Secretary, N.V.M.A., was referred to the General Meeting.

(b) An advertisement of a certain hospital was considered. Decided that the Secretary should make further investigations and refer the matter to the Veterinary Board.

(c) A suggestion from a member that the General Meeting be called "Congress" could not be accepted, as this would be unconstitutional.

(d) Mr. Van Heerden suggested that the Minister of Agriculture be asked to open the General Meeting. Agreed.

The Meeting closed at 10.45 p.m.

S. W. J. VAN RENSBURG,
Hon. Sec.-Treas., S.A.V.M.A.

Registered Veterinary Surgeons of the Union of South Africa.

ADELAAR, T. F.
ALEXANDER, R. A.
ALLCHURCH, W. B.
AMOS, S. T. A.
AMOS, V. J. E. (Miss)

BADENHORST, J.
BARNARD, W. G.
†BARNES, W. G.
BARRIE, N.
BEKKER, J. G.
BELONJE, C. W.
BERGH, M.
BISSCHOP, J. H. R.
BLOMEFIELD, L. C.
BOARDMAN, N. H.
BOSWELL, J. G.
BOGUE, J. Y.
BRANDSEN, J. G.
BROOKES, J. W. A.
BROWN, M. H. V.
BRÜMMER, B. J.
BUCK, J.

CAMERON-DOW, G. E.
CANHAM, A. S.
CANNON, G. T.
CARLESS, F. J.
CHALMERS, J.
CLAPHAM, W. S. B.
CLARK, R.
CLEMOW, E. T.
CLOETE, J. H. L.
COLES, J. D. W.
COOPER, V.
CORDY, C. H.
CROWHURST, J. W.
CURSON, H. H.

DALY, L. L.
DATNOW, RACHEL
DE BOOM, H. P. A.
DE KOCK, G .v. d. W.
DE LANGE, M.
DE VILLIERS, O. T.
DE VILLIERS, S. W.
DE VILLIERS, W. B.
DE WET, J. M.
DICKSON, J. L.
DIESEL, A. M.
DORÉ, J. L.
DUNNING, F. J.
DU TOIT, P. J.
DU TOIT, R.
DYKINS, W. A.

EDWARDS, L. T.
ERASMUS, C. J.
†EWING, S. H.

FICK, J. F.
FLIGHT, C. H.
†FOOTNER, W. E.
FOREST, J.
FOURIE, J. M.
FOURIE, P. J. J
FRANZ, H. G. J.
FREAN, J. R

GARRAWAY, R. S.
GAVIN, F. C.
GRAF, H.
†GREEN, W. J. B.
GRIST, A. G.

HAIG, D. A.
HAMLYN, W.
HARBER, A. F.
HARTIG, E. R.
HEARN, W. E.
HELLBERG, H. F.
HENNING, M. W.
†HODDER, A.
HOFMEYR, C. F. B.
HORWITZ, B. M.
HOWIE, A. M.
†HUTCHINSON, F.

JACKSON, C.
JACKSON, S. T.
JONES, W.

KEPPEL, J. J. G.
KIND, G.
KIRKPATRICK, A.
KLUGE, E.

LAMBRECHTS, M.
LUND, A. E.

LYONS, J. H.

MacGILVRAY, A. J.
MAINPRIZE, J. L.
MALHERBE, W. D.
MANSVELT, P. R.
MARAIS, I. P.
MARÉ, C. v. E.
MARTINAGIA, G.
MASON, J. H.
MATTHEW, A.
McCALL, D. B. J.
McINTYRE, G.
McNAE, A.
McNEIL, J.
MEARA, P. J
MEESER, J. N.
MÖNNIG, H. O.
MORFORD, L.
MORICE, Miss J. A.
MORTON, D. D.
MULLER, G. L.

NEITZ, W. O.
NELSON, E. C.
NESER, M. M.
NICOL, J.

O'DOWD, L.
OSRIN, R. B.

PAINE, R.
PARKIN, B. S.
PEROSSI, E. T.
PILKINGTON, J. K.
POWER, W. M.
PULLINGER, E. J.

QUIN, J. I.
QUINLAN, J.

REID, J.
ROBERTSON, P.
ROBINSON, E. M.
†ROBINSON, J. M.
ROBINSON, M. C.
ROSSITER, L. W.
RUNCIMAN, B.
RYKSEN, W. J.

SANDROCK, T. H.
SCHEUBER, J. R.
SCHULZ, K. C. A.
SCHOEMAN, J. H.
†SIMPSON, F. C.
SHARPE, C. M.
SIMSON, W. A.
SMITH, J. I
SMITH, P. R. B.
SNYMAN, P. S.
SPREULL, J.
STARKE, N. C.
STERNE, M. P
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Registered Veterinary Surgeons in the Union of South Africa—(Contd.)

VAN ASWEGEN, W. G.
VAN DER LINDE, N. T.
VAN DER VYVER, B. J.
VAN DER WALT, S. J.
VAN DER WATH, J. G.
v. d. WESTHUIZEN, J.
VAN DRIMMELEN, G. C.
VAN HEERDEN, C. J.
VAN RENSBURG, S. W. J
VERNEY, F. A.
VILJOEN, J. H. B.
VILJOEN, N. F.
VILJOEN, P. R.
WADLOW, C. H.
WALKER, J.
WATT, G.
WATT, J. S.
WEBB, J. L.
WESSELS, C. C.
WILLIAMS, J. G.
WOOLLATT, S. B.
WRIGHT, F. B.
†YOUNG, B.
ZIEHN, T.
ZWARENSTEIN, J. J.

† deceased.

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