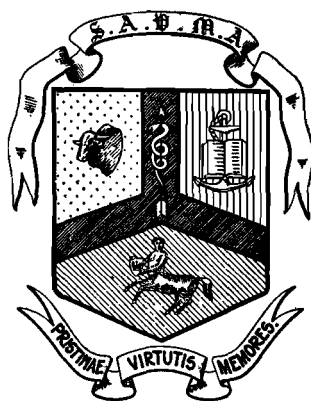


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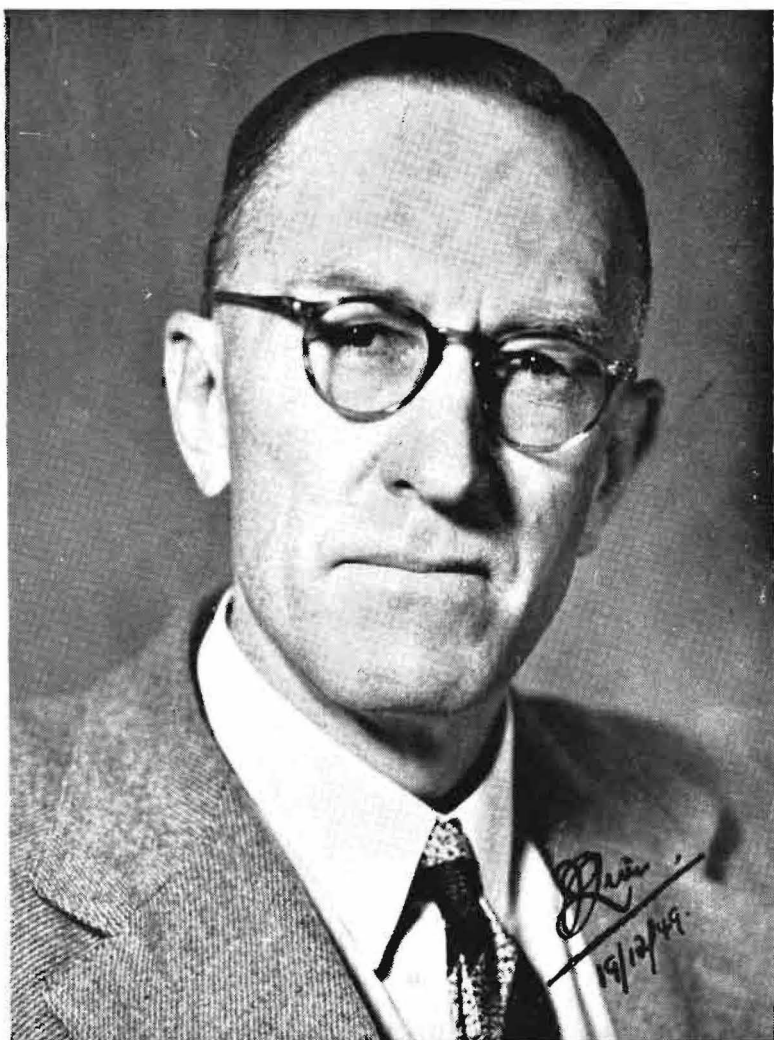
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THE LATE DR. J. I. QUIN.

## EDITORIAL

### THE LATE DR. J. I. QUIN: A TRIBUTE

It is with profound regret that we have to record the death of Dr. J. I. Quin during the night of March 19th, 1950. At the time of his death he was returning to Pretoria by train from Nelspruit after a short trip to the lowveld, during which he acted as host for the Union Government to a party of visitors headed by the Director-General of the Food and Agricultural Organization of United Nations.

When the Onderstepoort staff arrived for duty on the Monday morning, it is no exaggeration to say that they were stunned by the tragic news.

It is not the intention to deal with the many academic and scientific honours that were showered upon him during his comparatively brief career. Those can well receive attention on a more fitting occasion. It should be borne in mind, however, that he was one of the first group of students to graduate from the newly established Veterinary Faculty in 1924, was appointed to a post as research officer the following year, and eventually by sheer merit attained the very pinnacle of his profession.

Only on the 1st December, 1949, did he assume duty as the fourth Director of Veterinary Services, a worthy incumbent of the post founded by his former mentor, the father of our profession in South Africa, Sir Arnold Theiler. To seek a simile in our national sport, the old brigade of the club side had been compelled by the dictates of Father Time to hang up their boots. Quin was nominated as the new captain and immediately set to work to mould his available talent into a team. No-one can gainsay his infectious enthusiasm, his refusal ever to spare himself, his unbounded faith in the ability of the personnel at his disposal and his clear appreciation of the difficulties ahead. Some may have queried a decision or an opinion of his, but none the motive, the fixity of purpose or the impartiality. In little more than three months it was apparent that he was building up a team which, with him as captain, showed signs of being able to hold its own in open company. There is no knowing to what heights he might have risen as the leader. At the early age of 50, Higher Authority decided otherwise. Not only our profession, but the animal industry of the country and, through it, the sub-continent as a whole has suffered a loss, the extent of which will never be known.

While we commiserate with ourselves, we offer to his widow, his three children and the other members of his family our very sincere sympathy. There is no doubt that his conscientious approach to his difficult task contributed to his sudden demise. We trust it may be a measure of solace to them to know that we share with them in this irreparable loss.

# RABIES IN THE NORTHERN DISTRICTS OF SOUTH WEST AFRICA \*

L. VON MALTITZ

A very serious outbreak of rabies in cattle occurred in the districts of Outjo, Otjiwarongo and Grootfontein in 1948. The disease is spreading south and will probably sooner or later appear in Omaruru, Okahandja and Windhoek districts.

This paper is presented at the request of the Chairman, in the hope that it might serve a useful purpose in drawing the attention of other South West African veterinarians to certain peculiarities of the disease as it occurs here, and to possible circumstances which might obscure the clinical picture of rabies in cattle.

## HISTORY

Suspected cases of rabies in dogs were reported from Ovamboland and the Caprivi Strip in 1925 and 1926, and a case was reported in a native child bitten by a dog in Swakopmund in 1928. Although Ostertag and Kuhlenkampff (1942) state that rabies does not occur in South West Africa, it must be pointed out that the first positive case was diagnosed histologically and biologically in the Okavango in 1938, when brain material of an affected dog was sent to Onderstepoort from Runtu. A second positive case was diagnosed from Runtu in 1947.

The Native Commissioner at Kuring Kuru in the Okavango reported in 1935 that a Sister at the Finnish Mission Station at Lupala was bitten by a rabid dog. Eighteen natives were subsequently bitten by mad dogs and by a hyena in one case. He also stated that most of the dogs in the Okavango and Western Caprivi Zipfel areas appeared to be infected and a large number of dogs was destroyed by the natives.

Further reports were subsequently received from the same source to the effect that cattle contracted rabies in the British territory north of Kuring Kuru, and strange behaviour was reported by the natives in cattle, donkeys, pigs, goats, fowls, cats and wild animals. A hyena, which is usually a timid animal, became aggressive and attacked a native in his hut.

Several samples were sent to Onderstepoort at the time, but these were all too decomposed for diagnostic purposes, owing to the unsatisfactory transport facilities. The first positive case was only diagnosed in 1938 in the Okavango (referred to above).

A padre of the Roman Catholic Mission at Nyangana reported the presence of rabies in the Okavango in 1934, and believed that the disease was introduced from Angola across the river.

In October, 1935, the District Surgeon at Ondongua reported that

\* A paper read at the Annual Conference of Veterinary Surgeons of South West Africa at Windhoek, 30th September, 1949.



isolated cases of rabies occur in Ovamboland every year, and that 18 human cases were treated in 1935. He also reported that the disease was seen in oxen, a horse and a pig, but unfortunately it could never be positively diagnosed from material sent to Onderstepoort from time to time.

In 1937, a woman died eight weeks after she was bitten by a wild grey cat (*Felis ocreata?*) on a farm in Grootfontein district, in the police zone of South West Africa. Unfortunately, no specimens were sent to Onderstepoort, but no more cases were seen on the farm or in the vicinity.

In 1945, there was a suspected rabies outbreak involving 11 farms in the Gibeon - Mariental districts in the police zone. The Government veterinarians at Windhoek and Mariental investigated this outbreak, but the material collected was found negative at Onderstepoort and no further suspected cases were reported from that area.

From time to time brain specimens were sent to Onderstepoort from other districts in South West Africa, but all these were negative until May, 1948, when positive histological and biological tests were made on material taken from a bovine on farm Petersburg near Outjo.

#### HISTORY OF THE 1948 OUTBREAK

A farmer reported that he lost a few cattle from a disease which he termed "bulksiekte," on a farm situated on the northern boundary of Outjo district and Game Reserve No. 2. From his description of the disease a form of plant poisoning was first suspected. The farmer then moved his stock to Petersburg, a farm just south of Outjo, and reported four more deaths there.

The farm was then visited by the State veterinarian at Otjiwarongo, but only one sick cow was found. The animal was paralysed, salivated freely, strained and groaned from time to time.

At post mortem, pneumonia was found, which could have been caused by the farmer when he dosed the cow with Gow's remedy three days previously. No further lesions were seen, and a diagnosis of lamsiekte seemed to be correct, especially because the latter disease is very prevalent in this area. The skull was opened, however, and brain material sent to Onderstepoort in order to "eliminate the possibility of rabies". Both microscopical and biological tests were positive for rabies.

This first case illustrated the danger of making a wrong diagnosis of lamsiekte, especially as most cases are seen in the last stages of paralysis and coma, when the typical symptoms of the second stage are absent, and the macroscopic observations at post mortem are similar to those which are seen in lamsiekte.

After the first case was found, an extensive investigation was undertaken in the northern portion of Outjo district, and the histories obtained made it clear that rabies caused the death of many animals. No sick animals were seen, but eight farms were placed under precautionary quarantine. Soon afterwards, a mad jackal attacked a native herd boy on one of these farms, and material taken from this jackal was positive for rabies (Farm No. 33, 17/5/48). The native received a course of antirabic injections and suffered no ill-effects.

During the 18 months from March, 1948, until the end of September, 1949, 15 farms have been placed under quarantine for rabies which was confirmed at Onderstepoort. Sixteen other farms have also been placed under quarantine where the history of losses definitely pointed to rabies. Positive specimens were collected from two jackals, one dog, one goat, two cows, one calf and eight oxen. These figures refer to specimens sent from Otjiwarongo only, and up to the end of September, 1949.

#### TRANSMISSION

Neitz and Marais in 1932 mentioned the fact that in the Free State outbreaks, only members of the family *Viverridae* were responsible for transmitting the disease to man and cattle, and thought of the possibility that the virus changed its nature so that adaptation to this family could have occurred. In later reports, Neitz and Thomas also mentioned dogs and cats.

There is probably no connection with the disease as it occurs in South West Africa, but in this outbreak jackals were probably the most important carriers. Two cases were found positive in jackals so far, and on many farms the owners have seen jackals which behaved in a very queer manner. On two farms, dogs developed symptoms and died after killing jackals which were apparently tame and approached the homesteads. The brain of one of these dogs was positive for rabies.

A striking feature of this outbreak is the fact that up to now no cases have been reported in the *Viverridae*. Farmers and natives have been especially questioned on this point. Hardly any mungoose were seen in the north of Outjo district, where the disease first made its appearance, and this was ascribed to the peculiar limestone formation of the ground in that area, which was apparently unsuitable for the usual type of burrow.

The disease spread south from there, and although there is possibly a slight decrease in the jackal population on the farms further south, members of the family *Viverridae* are found in very large numbers near Outjo and Otjiwarongo. Once these small carnivora are found to transmit the disease here, one could expect a very serious deterioration in the rabies position in South West Africa.

#### EPIZOOTOLOGY

A feature of the outbreaks here is the widely differing nature of the incidence on individual farms. Losses in cattle have been very heavy on some farms, e.g. Neins, where 90 out of a total of 2,000 head of cattle were reported to have died of rabies over a period of three months. Material was sent away for confirmation in only two cases, which were both positive, but the possibility of another disease, e.g. anthrax or plant poisoning, or lamsiekte on this farm could not be excluded, and this figure must therefore be taken with reserve.

On Gelukwater, however, 38 deaths were reported due to rabies, and this figure is probably correct, since 14 animals in various stages of the disease were seen in one kraal when the deaths were investigated. There were about 450 cattle on Gelukwater at the time, and the deaths took place over a period of three weeks. No further cases were then

reported for more than a month, when one ox again showed symptoms of rabies and died.

On several other farms only three to four deaths took place over several months, although there were no fewer jackals on these farms than on others where many oxen died. Even where 14 or more cattle died of rabies in a week a peculiarity of the disease was that no deaths would take place for up to three months afterwards, when one or two cases would suddenly appear again in cattle.

On two farms a number of deaths took place only among the oxen which were fed bonemeal containing rough pieces of about 2½" long and 1" wide. Material taken from the dead oxen was positive for rabies. Other animals in the same camp, which had no access to the bonemeal, remained healthy.

The bonemeal troughs were made of cement and were large enough to allow 15 or more animals to feed at the same time. I have also observed an infected animal standing near a bonemeal trough dripping saliva on the contents of the trough, and venture to suggest that a combination of the above factors would create a very real possibility of transmission direct from one bovine to another.

This suggestion is made tentatively, because transmission by ingestion has never been regarded as of any importance in the spread of the disease, but it is conceivable that infection can be produced through damaged buccal mucosæ. One also wonders whether it is purely a matter of coincidence that the losses from rabies were limited to one or two cases in six months on farms where the owners immediately destroyed the first case and took pains to disinfect all bonemeal troughs thoroughly.

#### SYMPTOMOLOGY

##### *Cattle:*

The mad form is most frequently seen. A paralytic form also occurs as one sometimes sees cases which are comatose with no previous history of excitement or of bellowing.

In the mad form the prodromal stage is marked by restlessness, but the rapid emaciation is a striking feature. The rumen appears to be empty and the animal may stand near the water, but does not often attempt to drink. The symptoms of hydrophobia, which are so well marked in man do not appear in cattle.

Uneasiness increases and salivation becomes more marked as the animal passes into the second stage, in which bellowing is the chief symptom. Where a number of cattle are affected at the same time, the bellowing is so disturbing that the owners cannot stand it for longer than a few hours, and the animals are usually destroyed in this stage.

In one outbreak, 14 cattle in various stages of the disease were seen in one kraal, and here continuous straining was the main symptom. While straining, the head and neck was stretched forward and the animal bellowed. There was no diarrhœa or constipation, but a lot of gas was passed. Together with the salivation, the symptoms in these cases were very suggestive of some form of irritant poisoning.

Affected animals may attempt to butt one another, but apparently

with no intention of doing any serious harm. Cows with small calves may attack human beings.

After 24 hours, a swaying gait develops. The paralysis starts in the hind quarters and becomes worse until the animals can no longer get up.

In the last stage, the disease resembles lamsiekte very closely. There is paralysis, salivation and later coma, and the bellowing becomes so weak that only an occasional groan is heard. Death soon follows.

#### *Dogs:*

The symptoms which were seen in dogs during the present outbreak, followed the usual text book description, e.g. abnormal behaviour, seeking dark corners, peculiar expression in the eyes, "fly catching" or snapping at imaginary flies, biting sticks and stones, howling in a peculiar way with change in the voice. (This change, which is due to paralysis of the vocal chords, was not heard in cattle.)

One dog attacked its owner, and another bit several sheep in a flock. Death follows paralysis, but the dogs are usually destroyed by the owners very early during the course of the disease.

#### *Jackals:*

The complete picture could not be seen, but from the descriptions obtained from farmers, the course follows more or less the same pattern as in dogs. In the early stages the jackals show no fear of human beings or of dogs and often attempt to drink water at the troughs near the house. In one case, a farmer was sitting on the drinking trough with his two dogs near him, when a jackal approached and tried to drink water a yard or two away from him.

Jackals also attack sheep, dogs and native herd boys in the veld. One jackal tried to bite at the wheels of a passing car along a country road. In the later stages they turn in circles, and have also been found paralysed in the hind quarters. In this stage they are particularly dangerous, since they are then usually surrounded by a number of cattle sniffing at them, and manage to snap at the noses of the more inquisitive ones.

Some farmers claim that they can distinguish the howling of an infected jackal, so that the vocal chords are also apparently affected. It appears that death does not always follow complete paralysis, as there have been cases where jackals were found dead with their jaws clamped around an iron fencing pole or some similar object near the homestead.

### DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

When a farmer reports that he has been losing cattle from some form of poisoning, and describes symptoms of severe abdominal pain, straining, salivation and bellowing, then one usually thinks of rabies. The local term "bulksiekte" (bellowing disease) is a very good descriptive name for the disease as it occurs in this area, because there is no other disease in which this symptom is so marked or so consistently seen.

The diagnosis is easy when a good history of the case can be obtained, or where several animals can be observed. During the excit-

able stage the symptoms are typical, but where only one comatose animal is found, one has to rely solely on the laboratory tests on brain material. Post-mortem examination of all comatose animals in this area should be undertaken with care, and probably a sound procedure where the diagnosis is either *lamsiekte* or rabies, would be to remove the brain only. There is usually a well-marked hyperæmia of the cerebral meninges in affected cattle.

The following diseases should be considered with regard to differential diagnosis:—

(a) *Lead poisoning*: Old motor car batteries and paint are found on almost every farm, and cases of lead poisoning are therefore frequently met with. The main symptoms are gastro-enteritis and straining, together with nervous disturbances such as stupor, delirium and coma in acute poisoning. In chronic cases, tucked up abdomen, straining and groaning, together with the extensively involved nervous system, which may give rise to symptoms such as paralysis, partial or general, can further obscure a diagnosis of rabies. An important point to consider here is the duration of illness, since one seldom sees bovines which live longer than five days from the onset of symptoms of rabies.

(b) *Lamsiekte*: This disease is very prevalent here. In the last stages, all the symptoms resembling rabies are present, and the post-mortem picture is the same, except for the cerebral hyperæmia and possible foreign bodies in the rumen in rabies. The history is of primary importance where the animal is found comatose, but one must bear in mind that the paralytic type or dumb rabies, and also possible cases of atypical rabies, may show the same symptoms as those seen in *lamsiekte*.

(c) *Anaplasmosis*: There is emaciation, weakness and a tendency sometimes to attack approaching persons. The anæmia is usually well marked, however, and bloodsmears will establish the presence of the parasites in the latter disease.

(d) *Distemper in dogs*: Owners often overlook the importance of the further course of the disease in the early stages of suspected rabies, and usually destroy the dogs at the first sign of suspicious symptoms. The Lentz body of canine distemper may be confused with atypical Negri bodies, and the differential diagnosis has at times been very difficult histologically. (Refer to "The Diagnosis of Rabies in South Africa" by Neitz and Schulz.)

(e) *Vermiosis*: Intestinal worms may cause emaciation and irritation in dogs. *Cysticercus cellulose* infection in the brain of the dog may cause symptoms which may be mistaken for rabies.

(f) *Paralysis of piglets in South West Africa*: Paralysis of the hindquarters in piglets occurs in this area. The cause has so far not been definitely established, but the paralysis seems to be due to some form of deficiency. (Trace elements?)

(g) *Poisons*: Strychnine and some plant poisons.

(h) *Encephalitis*: Secondary infections of the brain, trauma and tumors of the brain.

Other diseases which can cause confusion, but which have so far not been seen here, are:—

(i) *Pseudo-rabies*: Symptoms are chiefly those of severe itching, but usually there are no symptoms of madness, and Negri bodies are not seen.

(j) *Borna disease*: Intranuclear Joest-Degen bodies microscopically.

(k) *Heartwater*: Microscopical examination of the brain for the presence of *Rickettsia ruminantium*.

(l) *Equine and Ovine Encephalomyelitis*.

From the above list of diseases the importance of the histological and biological tests of brain material in suspected cases of rabies becomes obvious. A good description of the case with history and all other relative information which may be available, should be forwarded together with the specimens.

Some State veterinarians have made histological examinations of brain material from suspected cases in the field, but this entails a certain amount of risk and should preferably be left to the laboratory.

A more complete evaluation of the histological and biological tests as carried out at Onderstepoort can be obtained from "The Diagnosis of Rabies in South Africa" by Neitz and Schulz (1949), but mention should be made here of the following excerpts from the above paper to show that Negri bodies may, under certain circumstances, be confused with the following:

(a) The non-specific Lyssa-like, bodies which may be found in the nerve cells of normal cats and mice, and the possibility that similar bodies may be found in other normal animals.

(b) The Lyssa-like bodies in rabbits injected with *B. pyocyaneus* (and the inference that caution should be exercised in test mice injected with decomposed material).

(c) Emanuiloff bodies of normal horses.

(d) The Lentz body of canine distemper.

An additional point to remember is that an immediate diagnosis of rabies is not regularly obtained histologically in animals which suffered from the natural infection.

#### COLLECTION AND DESPATCH OF MATERIAL

An easy method of opening the skull in horned cattle, and one which to my mind is safer, is to remove one horn and the attached portion of the skull in such a way that a small portion of the brain comes away with it. The horn provides a useful lever, and the entire brain can usually be removed easily. When the skull is sawn through in the middle, it is still sometimes necessary to complete the division with a wedge or bone chisel, and there is always some danger of small chips of bone or infected material coming into contact with the face of the operator. Use a saw with a broad blade in preference to the usual post-mortem saw with a blade of about 1" wide.

It is useful to remember that the postal regulations state that the material should be sent in sealed tins, and owing to the difficulties in

complying with these regulations it has now been found necessary to send the specimens in small wooden boxes by rail. The boxes must be well marked "Danger" and "Suspected Rabies". Special labels are supplied by the Department for this purpose, and their liberal use is insisted upon.

### CONTROL

In an article in *Hoard's Dairyman* (Vol. 94, No. 7, April 10, 1949), Dr. Bailey states that rabies in wild animals is self-limiting and will probably eliminate itself in those species. He therefore recommends control measures which centre round the domestic dog only, and claims that proper restraint, together with vaccination of dogs, will successfully control the disease.

Dr. Bailey bases these statements on conditions in the United States, where the Department of Agriculture reported 10,540 deaths from rabies in one year (1944) and where the incidence of the disease is increasing every year, but he is probably right when the disease appears in an area which is chiefly populated by man and domestic animals.

In the Orange Free State, experience has shown that the disease in the Viverridæ has definitely not eliminated itself, and strict restraint of domestic dogs, combined with an attempt to poison mongoose on a fairly large scale, has so far not eradicated rabies in that province.

Even if jackals and other wild carnivora are successfully exterminated in the northern districts of South West Africa, which is not very probable, there is no possibility of eradicating the mongoose. The carriers will, therefore, always be there and the disease will remain enzootic.

Measures to control the disease by restraint and destruction of domestic dogs and cats will show results in urban areas, and limit the incidence of the disease in human beings, but these measures will have little effect on the incidence of rabies on the farms, where jackals and other wild carnivora abound.

Quarantine measures are useful in helping to prevent movement of possibly infected cattle to clean areas. The spread of the disease to areas further south which are clean at present will be slowed down, but probably only postponed, by legislation prohibiting the movement of cattle into these areas from infected districts.

Publicity, with the object of making farmers thoroughly familiar with the disease and its dangers, is a useful weapon for fighting rabies. The symptoms must be recognised early so that effective isolation of affected animals can be applied.

Phophylactic vaccination of animals on a large scale is a possibility which will have to be further explored, particularly with regard to expense and practicability before it can be recommended under our conditions. There are also other objections against its use, e.g. that the immunity produced decreases after some months, so that an animal which becomes infected with the natural virus in that stage may develop the disease in a form which, while transmissible, cannot be recognised as rabies.

Veterinarians who are continuously exposed to infection in rabies

areas will find the information contained in "The Prophylactic Treatment of Rabies in Human Cases" by Dr. Turner of the Union Health Department, Cape Town, very useful. The following points in that paper may be mentioned here very briefly:

Only 5 - 15% of persons actually bitten by rabid animals develop rabies, but once the symptoms appear, the disease proves rapidly and inevitably fatal. There have been no authenticated cases of recovery reported.

Because of the occurrence of neuromparalytic accidents in a very small percentage of cases treated with vaccine, the treatment should not be lightly embarked upon. (0.008% non-fatal accidents and 0.003% fatal accidents occur, when killed vaccines are used.) There may also be other local and general reactions, and persons suffering from chronic malaria may have a relapse induced by vaccine treatment. The Semple type of killed vaccine (phenol) is used in South Africa at present.

The degree of protection given by present-day vaccines is much more limited than is generally realised. L. T. Webster in the *Am. J. Hyg.* states that vaccine given before infection is effective only under limited conditions, and with regard to vaccine given after infection he states that all workers except Fermi (who used rats) "have failed to demonstrate a significant protective effect of vaccination following experimental exposure to rabies virus by any route."

Vaccine treatment, however, offers the only hope of saving lives in persons infected with the virus.

In view of the above, Turner stresses the importance of local treatment of bite wounds within 30 minutes of infliction if possible. Tincture of iodine is probably as effective in destroying the virus if applied within 30 minutes, as fuming nitric acid. Anti-serum injected locally after a bite, may destroy the virus before it reaches the cord.

Very potent vaccines have, however, been prepared recently, and there now appears to be justification for new hope with regard to more effective treatment of the disease.

#### ACKNOWLEDGMENT

To Dr. Van der Merwe, who supplied most of the information with regard to the history of rabies in South West Africa from the official files of the Director of Agriculture, Windhoek.



# THE PARALYTIC ACTION OF HISTAMINE ON THE RUMINAL MUSCULATURE

R. CLARK,  
Onderstepoort

## INTRODUCTION

In his publication "A Study of Drugs Affecting the Motility of Bovine Rumen," Dougherty (1942) reported that the intravenous injection of histamine caused paralysis of the rumen. In the discussion the same author also states that it has been proved that the histamine content of the rumen can be increased by increasing the protein content of the diet. The possible significance of this finding is discussed and the author expresses the hope that further work will be done in this connection.

This important finding appears to have received little further attention and is not even mentioned in recent textbooks on veterinary physiology.

The present author is engaged on a series of experiments on drugs affecting ruminal motility in sheep, during which work the above finding was confirmed. The object of this communication is to focus attention on what may well be a significant factor in the ætiology of ruminal stasis.

## EXPERIMENTAL METHOD

Merino sheep with permanent ruminal fistulæ are being used throughout the trials, and the general procedure is as described by Quin (1938). The cavity of the rumen is connected directly to a rubber diaphragm tambour for the registration of pressure changes.

## RESULTS

The intravenous injection of 1 to 2 mgm. of histamine was found to produce a prompt and complete cessation of ruminal movements without affecting the general intra-ruminal pressure. It was also noted that the sheep defæcated repeatedly from 5 to 10 minutes after the injection.

Simultaneous recordings from the rumen and an intra-rectal balloon during such an experiment are shown in figure 1 (at the end of this article).

As will be seen, the injection of 2 mgm. histamine caused a cessation of ruminal movements lasting 30 minutes and hypermotility of the rectum, which persisted for 7 minutes. This split effect in the digestive tract of the sheep is of great interest and once again emphasises the peculiarities of the ruminant fore-stomachs.

In order to demonstrate that the ruminal paralysis was caused by the specific action of histamine, the antagonistic action of three anti-histamine drugs was investigated. The preparations used were *Anthisan* and *Phenergan*, both kindly supplied by Maybaker Ltd., and *Thephorin* (Roche).

It was found that the administration of 50 mgm. of any one of these drugs completely reversed the action of histamine within 6 to 10 minutes (see figure 2). Conversely, their prior administration blocked the action of histamine.

In order to determine whether the ruminal paralysis caused by histamine was of myo-genetic or central origin, a sheep was narcotised with chloral hydrate and the abdominal organs exposed. The left vagus was isolated in the cervical region, ligatured and severed. The threshold strength of faradic stimulation to cause a strong contraction of the rumen and reticulum was determined. An intravenous injection of 2 mgm. of histamine was then given and the immediate reaction of the abdominal organs observed. The rumen and reticulum gave no response, but the whole of the intestine assumed a wrinkled appearance due to spasm of the circular muscle layer.

The distal end of the left vagus was then stimulated with the previously determined strength of current, and the reticulum and rumen were seen to contract as strongly as they had prior to the injection of histamine. This was repeated with identical results at intervals of from 2 to 20 minutes after the injection.

### DISCUSSION

Barsoum and Gaddum (1935) showed that the muscle of the alimentary tract of most species responds to histamine by an initial contraction followed by relaxation, but that the time relations of these reactions vary considerably. The rectal cæcum of fowls, for instance, shows a contraction period of only a few seconds, whereas the ileum of the guinea-pig contracts for over an hour. In both instances, however, contraction is followed by relaxation, during which period the tissue is insensitive to further additions of histamine, but responds normally to other stimuli such as acetyl-choline.

It may well be that the muscle of the rumen obeys this general rule, but the period of contraction must be extremely short, as it could not be observed in the experiment already described. Attempts to demonstrate the contraction phase in isolated strips of rumen have also failed.

This problem was also studied in fistula sheep by giving minimal doses and recording the effect as soon as possible after injection. The results were as follows:—

<i>Dose of Histamine</i> <i>i.v. mgm.</i>	<i>Effect.</i>
.1	No effect.
.25	Inhibition for 4 minutes
.50	„ „ 10 „
1.0	„ „ 11 „
2.0	„ „ 30-40 „

It will, therefore, be seen that the smallest dose giving any demonstrable effect caused direct inhibition and the duration of inhibition was proportional to the dose. It can, therefore, be said that, at least for practical purposes, even minimal doses of histamine caused cessation of ruminal movements.

Ruminal paresis occurs in many conditions known to be associated

with high protein feeding and disturbances of digestion. Such diseases include acetonæmia of cattle, milk fever and possibly pregnancy disease of ewes. Hoflund and Hedström (1949) have shown the connection between disturbances in rumen digestion and acetonæmia. Geeldikkop also usually occurs on a high protein diet and is invariably complicated by ruminal paralysis.

Hoflund, Quin and Clark (1948) showed that excess protein suddenly introduced after a period on a diet low in both protein and readily available carbohydrate (starches and sugars) caused inappetence and even sudden death associated with the liberation of large amounts of ammonia in the rumen. Clark, Oyaert and Quin (in press) demonstrated a similar effect after dosing urea. Later, Clark and Lombard (in press) showed that ruminal paralysis could be caused by the absorption of alkali from the rumen.

Ruminal stasis associated with a high protein intake may, therefore, either be due to excess alkalinity of the rumen (ammonia) or to the presence of toxic amines (histamine?) or to a combination of both these factors.

The recent introduction of potent anti-histamine preparations has opened the way for further work. These drugs have already been advocated for the treatment of "certain forms of bloat," but without any apparent proved physiological basis.

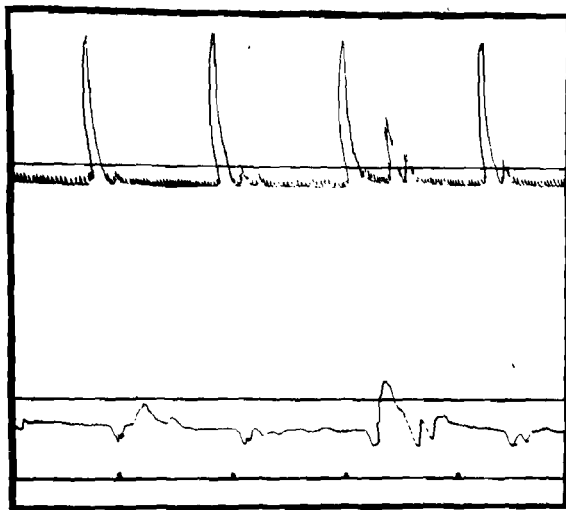
#### SUMMARY

1. The intravenous injection of as little as 0.25 mgm. of histamine causes cessation of ruminal contractions in sheep. The duration of the atony is proportional to the dose.
2. The paralysis can be reversed or prevented by the administration of anti-histamine drugs.
3. The rumen paralysed with histamine is still capable of responding to faradic stimulation of the vagus nerve.
4. The possible significance of histamine in the ætiology of certain forms of ruminal stasis is discussed.

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## Histamine

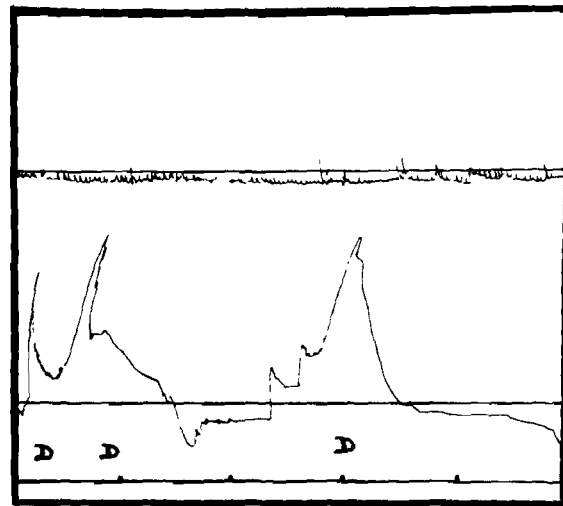


### Normal

Simultaneous Recording of Rumen (above)  
and Rectum (below)

Time marks 1 minute.

## Histamine (cont.)

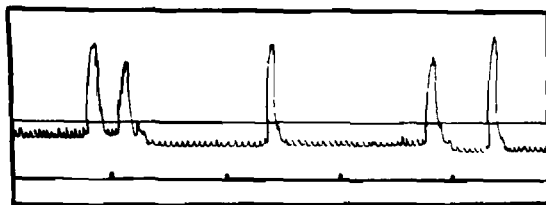


Recording 3.8 minutes after the injection  
of 2mgm. histamine intravenously.

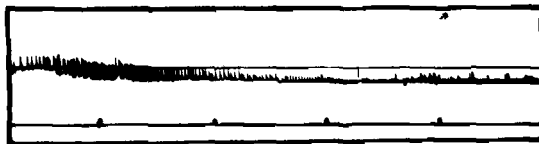
Note : Perosis of Rumen (Persisted 30 minutes)  
Hypermotility of Rectum (Persisted 7 minutes)

D - Defaecation.

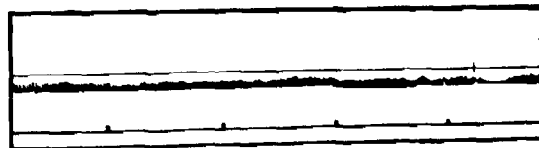
## Histamine



Normal

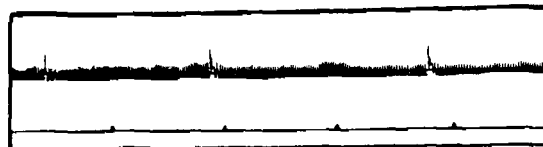


Immediately after 2mgm. histamine i.v.



6-10 minutes after histamine.  
50 mgm. Phenergan injected intramuscularly  
10 minutes after the histamine.

## - Treated with Phenergan



Immediately after Phenergan



6-10 minutes after phenergan.



10-15 minutes after phenergan.

# A NEW VIRUS DISEASE OF THE MUSCOVY-DUCK [CAIRINA MOSCHATA (LINN)] PRESENT IN NATAL

V. R. KASCHULA,  
Allerton Laboratory, Pietermaritzburg

The muscovy has long been regarded as an extremely hardy breed of duck. This characteristic, together with its rapid maturing qualities, has made it a popular breed. On farms in Natal they are left largely to fend for themselves, and very rarely has any trouble been encountered. Due to war-time meat shortages, they gained popularity in urban areas and in the Durban area at least, they are an important source of food.

It came as a surprise to find out that many more householders in the Durban area bred these ducks than was imagined. Even the poultrymen were unaware of this fact. Many householders kept small flocks of 20-30 for their own purposes in order to augment the restricted meat supplies. The total muscovy population is quite considerable and probably surpasses that of the other species of ducks. Vague reports had been received from time to time of mortality among ducks in the Durban area. On 27/5/47 a report was received from a veterinary practitioner in Durban who had carried out a post-mortem examination of a dead muscovy. The information he gave was that a very large number of this breed had died and many were sick. It was, therefore, decided to investigate the mortality at Glenwood, near Durban.

As a result of further reports of mortality received by the Government veterinary officer, Durban, it was decided to visit Durban North and Malvern.

## RESULTS OF INVESTIGATION

This showed that mortality among the muscovy ducks was extremely high. At Glenwood, one farmer had lost 150 ducks out of a total of 200, and of these 120 had died in the course of three days. At Fynnlands, 300 had died out of a flock of 450. A farmer at Malvern had suffered losses to the extent of 400 out of 500 ducks, while at Durban North, 80 out of 85 ducks had succumbed. In addition to these, losses had taken place in many small flocks. So far, this disease has only been observed in the Durban area. Only ducks of the muscovy breed were affected. In some flocks made up mainly of muscovies, but containing other breeds of ducks, only the muscovies died; the other ducks remained healthy throughout. On other farms, fowls, geese, turkeys and other species of ducks running with affected muscovies remained healthy. It is not possible to assess the numbers of ducks that died, but it is suggested that a conservative estimate would be in the thousands. From the nature of the mortality there was little doubt but that the condition was an extremely infectious one.

## SYMPTOMS

The first sign of illness was a complete loss of appetite which coincided with a marked rise of temperature up to  $109.2^{\circ}$ . Thirst was a characteristic symptom in all cases. The sick muscovy drank water continuously and as a rule would be found lying dead near the water supply. It is possible that the water in the containers became infected by the sick ducks, and when healthy ducks drank this water they acquired the infection. Shortly after the rise in temperature, the beak and legs became at first pale and then later assumed a pale purplish pink colour which would probably indicate cyanosis. A greenish watery diarrhoea, which soiled the vent and tail feathers, was a fairly common symptom. In some cases there was a slight mucous discharge from the nose and eyes. The eyes remained bright and the sick muscovy would "hiss" vigorously up to just before death if approached by anyone. In some birds that may have had a mild attack of the disease and recovered, other symptoms were observed. There was a paralysis of the limbs, especially in young ducklings, and they adopted what might be termed a creeping method of locomotion. In addition, some ducks, after an attack of the disease, showed a loss of voice. It must, however, be mentioned that some of these when killed showed an infection of the air-sacs with *Aspergillus fumigatus*.

Death followed a collapse of temperature and coma. The birds were found in a normal position with the head twisted to one side and resting on the ground.

The incubation period varied from 2-4 days, and the course of the disease lasted from 2-5 days. The spread in a flock was very rapid and usually reached its peak in a few days, and then would pass off after a week or ten days. Ducks of all ages were susceptible, but younger ducks were more so than older or breeder ducks. Heaviest losses were suffered with ducks about 2-3 months old.

## POST-MORTEM LESIONS

The only external lesion shown was soiling of the vent feathers as a result of the watery greenish diarrhoea. The ducks would usually be found dead near the water supply.

On opening the cadaver, the most characteristic lesions were the changes found in the liver and spleen. The liver was enlarged and showed numerous yellowish grey pin-point necrotic foci disseminated throughout the substance of this organ. The consistency was very friable and soft but not hyperæmic. The bile was very dark green and the gallbladder usually full.

The spleen was sometimes markedly enlarged, while in other cases this organ was apparently normal as far as size was concerned. The characteristic necrotic foci as seen in the liver appeared fairly consistently in the spleen.

There was often hyperæmia and œdema of the lungs, but in some cases no changes were noted. The heart showed epi- and pericardial petechial hæmorrhages, and the myocardium appeared often to be soft in consistency. The pancreas was very prominent and mottled, with grey opaque and translucent areas. Lesions were present in portions

of the alimentary tract. No alterations were seen in the œsophagus, proventriculus and gizzard, but a catarrhal enteritis was usually present. This varied from mild to severe in intensity, with the small intestine as the chief site. The kidneys were sometimes of a lighter colour than normal, probably due to a form of degeneration.

#### EXPERIMENTAL WORK

(1) Two adult muscovies were injected on 27/5/47 subcutaneously, each with 1 cc. of the pooled sample of citrated blood from freshly dead muscovies. The temperatures were recorded twice a day. On 30/5/47 both ducks recorded a peak temperature of 109.2°. There was a slight watery diarrhoea. The temperature gradually fell from this point and the legs and beak became noticeably pale. They lost their appetites from 29/5/47, and both were dead on the morning of 2/6/47. Post-mortem lesions resembled those seen in the muscovies in Durban. Cultures and blood smears were negative.

(2) Heartblood was collected in saline from the above two dead muscovies. After mixing the blood and saline well, the mixture was centrifugilised in order to deposit the blood cells. This was to facilitate filtration through Berkefeld and Seitz filters. The supernatant fluid was then clear and it was divided into two portions, one for filtration through a Berkefeld filter candle N size, and the other through a special E. K. Seitz filter. The specimens were tested bacteriologically before and after filtration for the presence of organisms, but were found to be negative both before and after filtration. Two groups each consisting of three muscovies were chosen for the injection of the two filtrates. As the original mixture of blood and saline was very weak, it was realised that the virus had been greatly diluted. It was, therefore, decided to inject 4 cc. of the filtrate subcutaneously. Each duck was placed in a separate pen, which was wired up, and no temperatures were taken. Sufficient food to last a week was supplied and the water receptacle was filled daily through the wire gauze, but not handled in any way. In this manner the chance of spreading this disease in any other way but by the injections was precluded. The other muscovies kept similarly acted as controls.

The results of these experiments revealed that two of the three muscovies injected with Seitz-filtered material died in six and eight days respectively, while the third showed no illness whatsoever. The three muscovies injected with Berkefeld-filtered material all died in six, seven and eight days respectively. These dead muscovies revealed typical post-mortem lesions as seen in muscovies dead from this disease in Durban. Cultures and blood and organ smears were negative. The control ducks remained unaffected.

The following other laboratory animals were tested out with fresh known virulent material:—

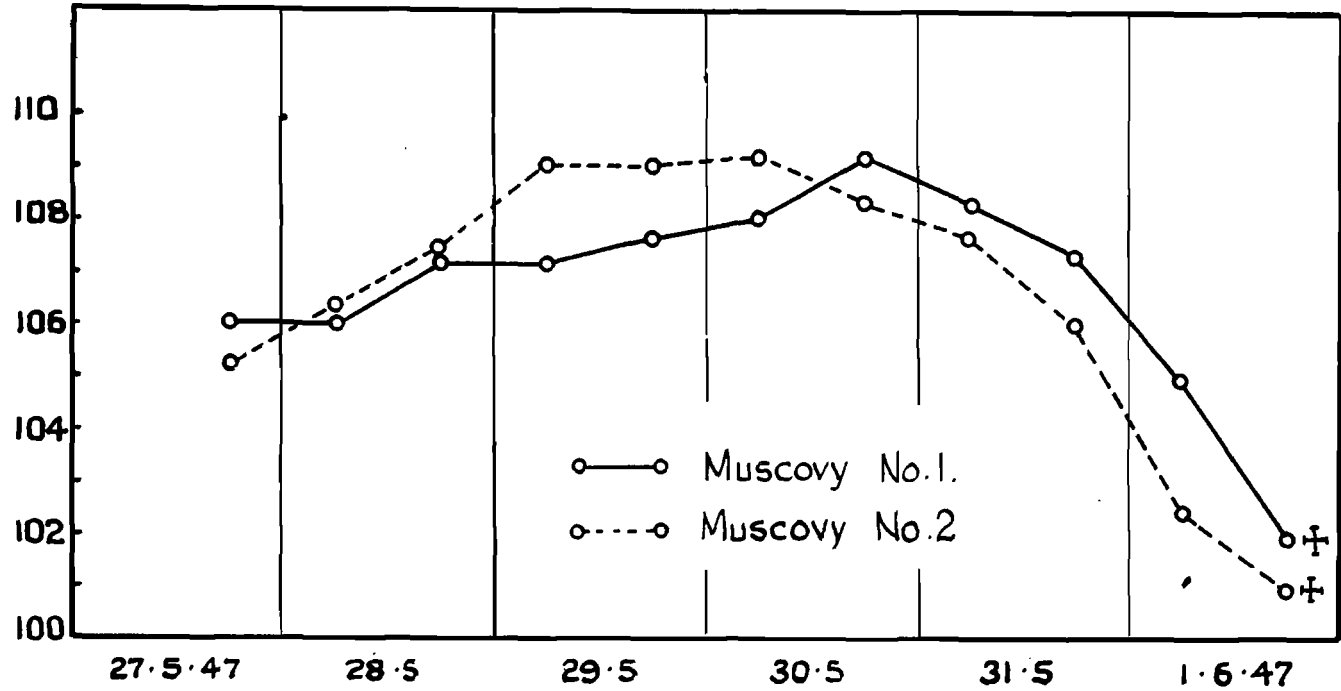
(3) Two guinea-pigs were injected subcutaneously with 2 cc. each of heartblood. Result: negative.

Two guinea-pigs were injected intraperitoneally with 1 cc. of blood. Result: negative.

(4) Two pigeons were injected subcutaneously and two were injected intraperitoneally with blood. Result: negative.



# Temperature Reactions of 2 Muscovy Ducks infected artificially.



(5) One fowl was injected subcutaneously, one intravenously and two intraperitoneally with blood. Result: negative.

(6) A muscovy which had recovered a month previously was injected subcutaneously. Result: negative.

(7) Three Pekin ducks obtained from Potchefstroom specially for this experiment were injected, two subcutaneously and one intraperitoneally. Result: negative. Not even a rise in temperature was recorded.

(8) One muscovy was dosed per os with blood saline mixture. All the symptoms developed in the muscovy and for a week it seemed as if the duck was going to die, but it survived and eventually recovered.

(9) A blood saline emulsion was stored in the refrigerator for 14 days and then injected subcutaneously into a muscovy, which died in six days, showing all symptoms and lesions typical of the disease.

(10) Cultures from the heartblood, liver, spleen and bile were prepared from all the cases examined.

The following media were used:—

Blood Agar, McConkey's and Liver broth. No growths were obtained.

In view of the reports of Graham Brandley and Dunlop (1938) and Hendrickson and Hilbert (1932) of the isolation of *Pfeifferella anatipestifer* from ducks in epidemics in America, their recommendations in regard to suitable culture media were followed, but again no growths were obtained under anærobic conditions. The epidemics with which they dealt were, however, not in muscovies, but in other species such as Pekin, etc.

In all the above experiments the muscovy ducks were about six months old and were found to be very susceptible to the disease.

Coles (1947) injected the following small animals at Onderstepoort with virulent material and obtained no positive reactions:—

- (a) Stanley crane, subcutaneously.
- (b) Pea-hen, subcutaneously.
- (c) White mice, intracerebrally.
- (d) Budgerigars, subcutaneously.
- (e) Canaries, subcutaneously.
- (f) White rats, intramuscularly and subcutaneously.
- (g) Ferrets, subcutaneously.

He also confirmed that pigeons, fowls, rabbits, guinea-pigs and Pekin ducks were not susceptible.

#### SUMMARY OF EXPERIMENTS

The disease was shown to pass a very fine filter (special E. K. Seitz). It was very virulent for the muscovy, Stanley crane, pea-hen, white mice, white rats, budgerigars, canaries, ferrets and rabbits. The disease produced a high temperature in muscovies and was negative bacteriologically and blood and organ smears were negative.

#### DISCUSSION

##### (a) *Virus and its specificity:*

In view of the fact that organ and blood smears were negative,

that the blood was bacteriologically sterile and that the causal agent passed through a special E. K. Seitz and a Berkefeld N filter, it is considered that the disease is due to a small filterable virus. A virus disease of ducks has been encountered in Holland, Bandet (1923) being the first to describe it. It resembled fowl plague, but was not transmissible to fowls. De Zeeuw (1930) described outbreaks in ducks which were in contact with large numbers of fowls. These fowls did not develop the disease, nor could it be transmitted artificially. Bos (1942), dealing with the same disease, concluded that it was a specific virus of the duck and was not related to fowl plague. Unfortunately, muscovy ducks were not employed in these experiments.

On a recent trip to the United States of America, the writer visited Dr. K. F. Hilbert on Long Island and was shown cases of *Pfeifferella anatipestifer* and duck cholera in Pekin ducks. This muscovy disease does not resemble these diseases. During this visit an epidemic of a virus disease was raging in the Pekin duck ranches of Long Island, causing very heavy losses in Pekin ducklings. This virus seems to resemble that encountered by Bos in Holland. In studying this virus, Fabricant (1949) was able to isolate Newcastle Disease from some ducklings, but Newcastle Disease had nothing to do with the epidemic in question, and appeared to be inapparent infections associated with the Newcastle infections in chicken populations.

It is interesting to recall that the Newcastle Disease outbreak occurred in fowls in the same area in 1945, Kaschula *et alia* (1946). No cases occurred naturally in ducks, but in experimental work at Allerton, muscovy ducks developed symptoms when injected intracerebrally. Apart from having occurred in the same locality, there is nothing in common between the viruses of Newcastle Disease and this disease of the muscovy ducks. Newcastle Disease was not present in South Africa during this outbreak. This virus is thus regarded as a new one, specific for the muscovy duck. Unfortunately, neutralization tests against Newcastle Disease were not done, but there appears to be nothing in common with it.

From the experiments conducted and from the report in the field, it would appear that the disease described is specific only for the muscovy duck, as, so far no other species of bird or animal has been found to be susceptible. Perhaps one would have expected the disease to be transmissible to other species of ducks such as the Pekin and Aylesbury, etc., but when one compares the origin and some of the characteristics of the muscovy with those of other species, one realises that the muscovy is a species apart and absolutely unrelated. In this connection Brown (1906) states "nearly all the races of domestic ducks are denizens of the Eastern Hemisphere, and almost without exception appear to be descended from the Mallard or wild duck. The Muscovy is originally a native of South America, where it is found in the wild state." The muscovy inhabits the hottest portion of tropical America. During the day it lives in swamps, where it finds suitable food, and towards the evening may be seen sitting in rows on the lower branches of large trees, descending thence to make inroads into the maize plantations and cornfields, where it does considerable damage, plucking up at

the same time the manioc and tapioca plants. While other species of ducks have an incubation period of 28 days for their eggs, the muscovy requires 35 days. It is also a commonly known fact that the cross between a muscovy and other ducks is a hybrid which is sterile. From the above data it is then not surprising that this muscovy duck virus disease is not transmissible to the other species of ducks. On many occasions Pepin and Aylesbury ducks or their hybrid with the Muscovy were in direct contact with the disease, but were not affected by it.

(b) *Mortality, morbidity, natural transmission and immunity:*

The mortality varied from 10 - 95%, usually above 50%. All ages are affected, but it appears as if younger Muscovies are the most susceptible. The heaviest losses were observed among Muscovies varying from a few weeks to six months old. Once the disease appears in a flock it spreads rapidly, the morbidity being in the vicinity of 100% within a few days. Due to the rapid course, the epidemic as a rule passes over in about ten days. The disease has been found to travel distances of up to a mile from the nearest outbreak, and the method of transmission would suggest the possibility of some winged insect being a vector.

Mosquitoes are fairly plentiful in this area. The Muscovy is also very fond of flies, so there is the possibility that flies may carry the infection even perhaps in a mechanical way. However, the rapid spread once in a flock is thought to be chiefly through the agency of infected drinking water.

In one severe outbreak 300 Muscovies died and 150 survived. After seven months the disease broke out again among one-month-old ducklings, but none of the birds which had been through the disease re-developed it. This would suggest that immunity lasts for at least seven months and that there is a possibility of "carriers."

#### DIFFERENTIAL DIAGNOSIS

This disease must be differentiated from the following diseases:—

- (a) *Botulism*: Is the cause of tremendous epidemics in America. Kalmbach (1930), and elsewhere. It is frequently seen in South Africa. This is, of course, not transmissible, and there are no post-mortem lesions. Paralysis is the chief symptom.
- (b) *Spirochaetosis*: This is transmissible to other ducks, rabbits, fowls, etc., and *S. anserina* is detectable in smears. It does not pass a filter.
- (c) "*Tampan paralysis*": due to the toxin of *Ixodes pilosus*. This is not transmissible and a negative post mortem is obtained.
- (d) Duck cholera due to *Pasteurella avisepticus*. This is transmissible to fowls, pigeons, rabbits, etc., does not pass a filter, grows on culture media and bipolars may be seen in smears.
- (e) Duck virus of Bandet (1923) resembling fowl plague. This occurs in other species of ducks. Apart from numerous petechial hæmorrhages, post mortems are generally negative.
- (f) *Aspergillosis*: This is a fairly common disease, seldom causing

heavy losses. The fungus can be cultivated bacteriologically. It is usually confined to the air-sacs and peritoneum.

- (g) *Salmonellosis*: This disease usually affects ducklings, though losses may occur in adult ducks. *S. anatum*, *S. typhimurium* and *S. pullorum*, the usual causes in South Africa, are easily isolated bacteriologically.
- (h) *Pfeifferella anatipestifer*: epidemic as seen in America by Hendrickson and Hilbert (1932). This bacterium can be isolated on blood or serum agar under anærobic conditions. It was, however, not found in Muscovies, but in other species of ducks.
- (i) *Poisoning and dietetic errors*: The post-mortem lesions are usually confined to the intestines and proventriculus. This could also not be transmitted to ducks by injection of blood.
- (j) *Leucocytozoon infection*: This is an important disease, causing heavy losses along the Great Lakes of North America. It is transmitted by black flies and is due to a protozoon easily seen in slides.

#### CONTROL

As this is apparently a contagious disease, movement of Muscovies in affected areas should be controlled. As, however, little is known of the transmission, naturally no steps have as yet been taken.

#### SUMMARY

1. A new severe acute infectious disease of the Muscovy duck is described.
2. The causative agent is apparently a small filterable virus, which should be regarded as a new one.
3. The disease is specific for the Muscovy duck and attempts to transmit it to other small laboratory animals and birds have failed to produce a visible disease process.
4. The disease is apparently contagious, but probably winged insects play a rôle in spreading it to other flocks.
5. The chief symptoms are complete loss of appetite, pale legs and beak, a high temperature in the early stages, diarrhoea, thirst and listlessness, and the very marked collapse temperature after the crisis.
6. The main post-mortem lesions are a multiple focal necrobiosis of the liver with a catarrhal enteritis and petechial hæmorrhages of the heart and pulmonary œdema.

#### ACKNOWLEDGMENTS

I wish to record my grateful thanks to Dr. A. S. Canham, Officer in Charge of Allerton Laboratory, for his advice and help; to Professor J. D. W. A. Coles of Onderstepoort for his advice and assistance in carrying out some of the experiments; to Dr. J. Viljoen, Government Veterinary Officer, Durban, for reporting outbreaks and gaining information, and to the technical assistants for their help.

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# ON THE CONTAMINATION OF THE MILK SUPPLY OF THE CITY OF PRETORIA WITH TUBERCLE BACILLI

M. W. HENNING and W. G. VAN ASWEGEN

It has been known for some time that tuberculosis is widespread in urban dairy herds in South Africa (Green, 1933; Henning, 1949), but as facilities are not provided for the general application of the tuberculin test to all suspected herds, this study was designed to inquire into the extent of the contamination of milk supplied to the city of Pretoria. By examining bulked milk samples in Johannesburg, Pullinger (1942) reported a contamination of 2.5 per cent with tubercle bacilli. In Cape Town, Horwitz (1944) found tubercle bacilli in 3.4 per cent of the milk from herds supplying the city of Cape Town. Pullinger (1942) attributed the comparatively low percentage of contamination of the Johannesburg milk supply to the low incidence of tubercular mastitis in South Africa; he believes that dairymen generally dispose of their cows as soon as there is evidence of a persisting mastitis.

## THE METHOD

The method employed was based on that used by the Medical Research Council (1933) and by Pullinger (1934). The test was a biological one that was confined to guinea-pigs.

Composite samples of raw milk were obtained from two hundred different herds supplying milk to the city of Pretoria. After thorough mixing of the milk in the different cans from individual herds, a few ounces were removed from each can and mixed in a container. Approximately 100 cc. of the milk was centrifugalized at 3,000 r.p.m. for 30 minutes. At the beginning of the test, 2 cc. of the gravity cream was thoroughly mixed with the deposit of 100 cc. of milk from the same sample, 1 cc. of this mixture being injected intramuscularly into the right thigh of each of two guinea-pigs; but later one guinea-pig was injected with 1 cc. of the gravity cream from each sample and another with the corresponding sediment emulsified in 1 cc. of normal saline.

The guinea-pigs were killed from 6 to 8 weeks after the injection. On post mortem, the presence of tubercle bacilli in the lesions was demonstrated microscopically. If any guinea-pig died before the lapse of six weeks it was carefully autopsied, and if no evidence of tuberculosis could be found, another guinea-pig was injected in the same way with milk obtained from the same herd. In doubtful cases the suspected spleens were ground up, emulsified and used for the injection of further guinea-pigs. After a further six or eight weeks the sub-inoculated guinea-pigs were killed, autopsied and the suspected organs examined microscopically. After the primary test, seven of the two hundred samples were found to be positive, yielding tubercle bacilli on a microscopical examination of the lesions. Twenty-four presented lesions,

which were regarded as doubtful. Portions of six of the suspected spleens were ground up, emulsified and sub-inoculated into a second series of guinea-pigs and portions were examined histologically. Two of the guinea-pigs sub-inoculated with one of these suspected organs developed lesions in which tubercle bacilli could be demonstrated microscopically, thus increasing the number of positive milk samples from seven to eight, i.e. a 4 per cent contamination. No tubercle bacilli could be detected in the guinea-pigs injected with the remaining five suspected organs. It is believed that the lesions in some of the doubtful organs were probably due to a *Brucella* infection.

Some of the suspected organs and some of the positive ones were examined histologically by Dr. M. de Lange, of the Section of Pathology, but he was unable to differentiate on purely histo-pathological grounds between the tuberculosis and the brucellosis lesions found in guinea-pigs.

No effort was made to demonstrate the presence of *Brucella* in the lesions, although it is conceded that some of them might have been due to brucellosis. It should be pointed out, however, that it is not uncommon for tubercle bacilli to occur in conjunction with *Brucella* in the same organs or guinea-pigs injected with milk contaminated with both organisms (Pullinger, 1935). Moreover, as the authors have demonstrated the presence of tubercle bacilli in the suspected organs of one of the doubtful cases, it is not impossible that more positive cases would have been detected if all the suspected organs had been sub-inoculated.

TABLE I.

*Summary of results obtained by injecting guinea-pigs with either gravity cream, sediment or a mixture of sediment and gravity cream.*

Herd from which sample was obtained	No. of guinea-pigs injected with gravity cream alone	Result	No. of guinea-pigs injected with sediment alone	Result	No. of guinea-pigs injected with a mixture of gravity cream and sediment	Result	Remarks
I	—	—	—	—	2	P2	
I	1	P	1	N	—	—	
I	1	N	1	P	—	—	
I	1	?	1	P	—	—	
II	—	—	—	—	2	P2	
III	1	P	1	P	—	—	
IV	1	P	1	N	—	—	
V	1	P	1	N	—	—	
VI	1	P	1	N	—	—	
VII	1	P*	1	N	—	—	
VIII	1	P	1	N	—	—	

P — Positive. N — Negative. ? — Guinea-pig died before lesions could develop.

\* — Doubtful case: spleen was emulsified and sub-inoculated into two guinea-pigs, both of which developed lesions in which tubercle bacilli could be demonstrated.



## SUMMARY AND CONCLUSIONS

Composite samples of milk obtained from two hundred different herds were examined; eight of these (4 per cent) proved to be contaminated with tubercle bacilli. From one positive herd, however, four different samples were taken at varying intervals, all proving to be contaminated, increasing the number of positive samples to eleven. Two of the positive samples were detected in guinea-pigs injected with a mixture of gravity cream and sediment. The rest were detected in guinea-pigs receiving either gravity cream alone or sediment alone. Seven of the guinea-pigs injected with gravity cream alone became tuberculous, while only one of the seven that received sediment from the same samples developed lesions. In the case of only one sample did tuberculous lesions develop in the guinea-pig injected with sediment and not in the one that received the corresponding gravity cream. In another case, tuberculosis was produced in a guinea-pig by an injection of sediment, but the animal which received the gravity cream died before lesions could develop.

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The Hon. Secretary,  
South African Veterinary Medical Association,  
P.O. Onderstepoort.

Dear Sir,

### *Fees of Veterinarians.*

I beg to inform you that the Veterinary Board at its last meeting, held on the 31st October, 1949, discussed the matter of veterinarians' charges for professional services rendered.

The Veterinary Board arrived at the conclusion that many complaints regarding what clients consider to be exorbitant charges would be obviated were there a better understanding *ab initio* between client and practitioner in regard to the question of costs in relation to prognosis, particularly in cases in which a protracted illness is anticipated and the prognosis is unfavourable. I was instructed to suggest to your Association that all veterinarians be notified of the Board's opinion through the medium of the Association's *Journal*.

Yours faithfully,  
*Registrar of Veterinarians.*

# THE TRANSMISSION OF LUMPY WOOL (*ACTINOMYCES DERMATONOMUS*) BY BENZENE HEXACHLORIDE DIPS

P. W. THOROLD

A farmer of the Vryheid district, Natal, complained that after compulsory dipping for scab in the recommended .01% benzene hexachloride dip a number of cases of lumpy wool occurred in his flock. The B.H.C. dip used was of the wettable powder type and contained 40% technical B.H.C.

The object of the work outlined below was to determine whether benzene hexachloride dips could become sources of infection of *Actinomyces dermatonomus*.

*Actinomyces dermatonomus* was isolated from an infected sheep by seeding flakes of dried epithelium on to serum agar and Sabouraud's dextrose agar. The morphology, cultural characters, pathogenicity, transmission and treatment have been well described by Mason and Bekker, Bull, Steyn, and Albiston. All cultures used for transmission and culture tests were six-day serum cultures incubated at 37°C — this method gave a heavy yield consisting mainly of conidia and a few filaments.

The fungicidal properties of different strengths of benzene hexachloride were tested on species of *Microsporum*, *Trichophyton*, *Achorion* and *Sporotrichum* (all cultures contained large numbers of spores).

## CULTURE TESTS

Dilutions of benzene hexachloride were made up to contain different amounts of the gamma isomer; to one set of dilutions was added 0.03% copper sulphate as a control; the solutions in 500 cc. flasks were then autoclaved at 20 lbs. for 30 minutes. A benzene hexachloride dip specimen from a farmer's tank made up to strength 0.012% B.H.C. one month previously, through which a large number of stock had passed and consequently contained a high percentage of organic material, was also used. The tests were controlled by saline and serum cultures. Two and five days after inoculation material from each flask was seeded on to different media. The results are indicated in Table I.

TABLE I

Pure cultures in Benzene Hexachloride Solutions.	Growth after 2 and 5 days.	
	Actinomyces dermatonomus	Fungus Species
0.005% Gamma B.H.C. . . . .	No growth	No growth
0.005% Gamma B.H.C. and Scabs and Exudate	"	"
0.01% Gamma B.H.C. . . . .	"	"
0.015% Gamma B.H.C. . . . .	"	"
0.02% Gamma B.H.C. . . . .	"	"
0.03% Gamma B.H.C. . . . .	"	"
0.005% Gamma B.H.C. 0.03% CuSO <sub>4</sub> . 5H <sub>2</sub> O . . . .	"	"
0.01% Gamma B.H.C. 0.03% CuSO <sub>4</sub> . 5H <sub>2</sub> O . . . .	"	"
0.015% Gamma B.H.C. 0.03% CuSO <sub>4</sub> . 5H <sub>2</sub> O . . . .	"	"
0.02% Gamma B.H.C. + 0.03% CuSO <sub>4</sub> . 5H <sub>2</sub> O . . . .	"	"
0.03% Gamma B.H.C. + 0.03% CuSO <sub>4</sub> . 5H <sub>2</sub> O . . . .	"	"
0.012% Gamma B.H.C. (ex dip tank) . . . . .	"	"
Saline . . . . .	Good growth	
Serum . . . . .	"	

From the above table it is evident that benzene hexachloride at the given concentrations did not favour the growth of these organisms in vitro.

## TRANSMISSION OF ACTINOMYCES DERMATONOMUS

Merino sheep were used, all being over two years old with  $\pm$  four months' wool growth. A Friesland heifer was included to determine whether the strain was host-specific or not.

An area on the animal's back was infected with a serum culture and allowed to dry; the area was then slightly scarified (scarification was not found to be a necessary adjunct to a positive reaction), well wetted with benzene hexachloride solution, and thereafter damped daily with water for three weeks, except where mentioned. The results are indicated in Table II.

TABLE II.

Sheep infected with Actinomyces dermatonomus, then treated with B.H.C.	Damped daily with water.	Not damped.
0.005% Gamma B.H.C. . . . .	+	—
0.01% Gamma B.H.C. . . . .	+	—
0.015% Gamma B.H.C. . . . .	+	—
0.02% Gamma B.H.C. . . . .	—	—
0.03% Gamma B.H.C. . . . .	—	—
0.005% Gamma B.H.C. + 0.03% CuSO <sub>4</sub> . 5H <sub>2</sub> O . .	—	—
0.01% Gamma B.H.C. + 0.03% CuSO <sub>4</sub> . 5H <sub>2</sub> O . .	—	—
0.015% Gamma B.H.C. + 0.03% CuSO <sub>4</sub> . 5H <sub>2</sub> O . .	—	—
0.02% Gamma B.H.C. + 0.03% CuSO <sub>4</sub> . 5H <sub>2</sub> O . .	—	—
0.03% Gamma B.H.C. + 0.03% CuSO <sub>4</sub> . 5H <sub>2</sub> O . .	—	—
0.012% Gamma B.H.C. (ex dip tank) . . . . .	+	—
Saline (Control) . . . . .	+	—
Serum (Control) . . . . .	+	—
Bovine — pure culture . . . . .	+	—
Bovine — scab and exudate . . . . .	—	—

The transmission tests in the above table were repeated once, with the same results.

Where a positive reaction occurred, raised yellow pustules were noted five days after infection. These developed into typical lesions with exudate and scabs after 21 days. In all cases the lesions healed without treatment after dipping was discontinued.

From the above it is evident that any of the organisms mentioned soon lose their viability in benzene hexachloride and that these dips, *at the strengths used for the control of external parasites*, are unlikely to act as reservoirs of infection of *Actinomyces dermatonomus*. It is apparent that a moist fleece is necessary for the growth of *Actinomyces dermatonomus* and that the benzene hexachloride *per se* did not stimulate its growth, since in the absence of repeated dampenings no cases occurred with any of the B.H.C. concentrations used. It is also clear that with concentrations of .02% and over, no growth occurred, even when the skin was damped. Where the organism is present in the wool, a few days' rain or misty weather and optimal conditions pertain for the organism to penetrate the superficial layers of the dermis and produce the condition. Copper sulphate in a dilution of 0.03% appears to control the condition quite successfully and it is suggested that when prevailing weather conditions favour the possibility of an outbreak of the disease, copper sulphate could advantageously be added to the dip at the rate of 3 lbs. per 1,000 gallons dipwash. This would control the fungi which may be present but have caused no damage at the time of dipping. In view of Steyn's experiments, it could not, however, be regarded as being of value for curative purposes.

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# THE PROPHYLACTIC EFFECT OF ANTRYCIDE METHYL SULPHATE AGAINST *T. CONGOLENSE* IN GUINEA-PIGS

G. D. SUTTON

Curd and Davey (1949) reported that antrycide gave a prophylactic effect against *T. congolense* in mice. Wilson (1949) found the same with *T. simiae* in pigs. An investigation was made with *T. congolense* in guinea-pigs to see whether the same effect could be produced.

Guinea-pigs were injected subcutaneously with a solution of antrycide methyl sulphate in distilled water. The dose given was 1 mgm. per 200 grammes live weight. The treated guinea-pigs were challenged later with blood from guinea-pigs heavily infected with *T. congolense*. This blood was mixed with equal parts of 1 per cent sodium citrate and 1 ml. of the mixture injected subcutaneously. The strain used was recently isolated from cattle and established in guinea-pigs.

The results are given in the table on the following page.

## CONCLUSION

Antrycide methyl sulphate had a prophylactic effect against *T. congolense* in guinea-pigs for approximately three months. It prevented the development of a fatal disease, but there is a possibility that the guinea-pigs might be harbouring the trypanosomes in their bodies without being demonstrable.

## REFERENCES

- CURD, F. H. S., and DAVEY, D. G. (1949): "Antrycide": A new Trypanocidal drug. *Nature*, Vol. 63, No. 4133, pp. 89-90.
- WILSON, S. G. (1949): Treatment of *T. simiae* Infections with "Antrycide." *Vet. Rec.*, Vol. 61, No. 28, pp. 397-398.

Injected with Antrycide	Controls	Period between injection and challenge	Blood Smears	Result	Sub-inoculation	Result
6	3	18 days	All negative All positive	All lived All dead by 13th day	Not done —	— —
6	3	30 days	All negative All positive	All lived All dead by 15th day	Not done —	— —
6	3	69 days	All negative  All positive	All lived  All dead by 12th day	Pooled blood injected into 4 guinea-pigs  —	2 became infected and died. 2 remained uninfected  —
8	3	95 days	All negative  All positive	All lived  All dead by 16th day	Divided into two groups of 4. Pooled blood from each group and injected into 2 guinea-pigs  —	All lived. No infection could be found  —
9	3	132 days	7 positive 2 negative  All positive	5 died by 16th day. Remainder, including 2 which showed blood infection, lived till 36th day, when destroyed All dead by 16th day	Not done  —	—  —
8	3	170 days	All positive All positive	All dead by 15th day All dead by 15th day	— —	— —

# IS PRIVATE PRACTICE TAKEN FOR GRANTED ?

A. D. THOMAS and R. EVERY

Most of the articles by private practitioners appearing in these pages have been descriptions of unusual clinical cases or records of some achievement or other in the fields of surgery, medicine or obstetrics. Little has been written to show what private practice really amounts to in humdrum work. In other words, what constitutes the bread and butter of the average veterinary surgeon in this country.

Since the veterinary service rendered by private practitioners has made vast strides in the past 15 years and undoubtedly is destined to advance still more, it is only right that articles appear from time to time to illustrate the type and variety of the work.

We hope that others will also contribute from their wider experience, not merely to amplify our figures, but particularly to assist in presenting a true cross section of private practice in its varied branches and in the different provinces.

In order to give a sample of our own work in as brief a form as possible, we have tabulated below species by species all the cases dealt with in this practice during one calendar year, that is from January 1st to December 31st, 1949.

## D O G :

<i>Condition.</i>	<i>Number.</i>	<i>Condition.</i>	<i>Number.</i>
Abscesses .....	50	Distemper immunisation .....	79
Alopecia .....	1	Dystokia .....	18
Anal Glands occluded .....	4	Exanthema .....	1
Arthritis .....	6	Eclampsia .....	2
Ascites .....	9	Eczema (moist) .....	82
Balanitis .....	1	Encephalitis (non-distemper) .....	3
Biliary Fever .....	299	Enteritis and Diarrhoea .....	47
Black Tongue .....	1	Epistaxis .....	1
Bronchitis .....	2	Entropion .....	1
Burns .....	1	Export certificates .....	5
Calculi: Bladder .....	3	Fractures .....	55
"    Urethral .....	5	False pregnancy .....	2
Castration .....	7	Foreign body: larynx .....	1
Cataract .....	1	"    "    palate .....	1
Cerebellar Ataxia .....	2	Gastro-enteritis .....	10
Conjunctivitis .....	20	Hæmatoma .....	2
Constipation .....	17	Helminthiasis treated .....	25
Cystotomy .....	1	Hepatitis .....	4
Dermatitis .....	15	Hernia: inguinal .....	2
Dermoid Fistula .....	7	"    umbilical .....	2
Dewclaws removed .....	8	Hodgkin's Disease .....	1
Dislocation .....	15	Hysteria .....	1
Distemper (includes 2 hard pad) .....	250	Hysterotomy .....	1
		Hysterectomy .....	1

<i>Condition.</i>	<i>Number.</i>	<i>Condition.</i>	<i>Number.</i>
Interdigital cyst .....	2	Pleuritis .....	1
Luxation eyeball .....	9	Proctitis .....	2
Luxation harderian gland .....	1	Prolopse rectum .....	4
Lymphadenitis .....	1	Posterior paralysis .....	25
Mange: Demodectic .....	23	Pyometra .....	2
"    Sarcoptic .....	20	Peritonitis .....	1
Mastitis .....	1	Ranula: inflammatory .....	2
Mesalliance .....	2	Rickettsia canis .....	1
Metritis .....	7	Rickets .....	2
Meningitis .....	1	Ringworm .....	21
Nasal catarrh .....	2	Ruptured diaphragm .....	1
Neoplasms .....	16	Ruptured tendo achilles .....	1
Nephritis .....	18	Seroma .....	2
Obesity .....	3	Sitfasts ticks) .....	1
Oophorectomy .....	88	Stomatitis .....	12
Ophthalmia .....	8	Tails docked .....	55
Osteomyelitis .....	1	Teeth extractions and scal-	
Oesophageal stenosis .....	1	ings .....	20
Othæmatoma .....	2	Toe amputation .....	1
Otorrhæa .....	47	Urinary incontinence .....	1
Papillomatosis oris .....	22	Urticaria .....	9
Paralysis nictitating mem-		Valvular insufficiency .....	4
brane .....	1	Vulvitis .....	2
Pharyngitis — Tonsillitis .....	100	Wounds .....	160
Phlegmosis .....	3	Unknown .....	204
Pruritus — "Pimplly Itch" .....	26		
Phymosis .....	1		
Pneumonia .....	6		
Poisoning .....	6		
		TOTAL .....	2,026

# B O V I N E :

Abscesses .....	4	Heartwater treated and post	
Actinomycosis .....	3	mortem .....	27
Afterbirth removal .....	18	Mastitis .....	15
Atony forestomachs .....	17	Metritis .....	3
Arthritis .....	1	Milk examination .....	6
Black quarter immunisation	10	Panaritium .....	1
Blood in milk .....	4	Pericarditis: Traumatic .....	2
Broncho-pneumonia .....	2	Pharyngeal obstruction .....	1
Bull ringing .....	6	Pleuritis .....	1
Contagious abortion bleeding	39	Prolapse uterus .....	1
Castration (burdizzo) .....	4	Ruptured uterus .....	1
Certificate of soundness .....	2	Redwater treated and post	
Dystokia .....	5	mortem .....	19
Emphysema subcutis .....	1	Stiffsickness: 3-day .....	5
Fractured spine .....	2	Sterility examination .....	50
Gifblaar poisoning .....	1	Teat operations .....	2
Gallsickness immunisation .....	79	White scours .....	1
Gallsickness treated .....	6	Unknown .....	31
Hoven .....	7		
Hypocalcæmia .....	3		
Heartwater immunisation .....	16	TOTAL .....	395



# C A T :

<i>Condition.</i>	<i>Number.</i>	<i>Condition.</i>	<i>Number.</i>
Abscesses .....	4	Oophorectomy .....	31
Castration .....	25	Parotitis .....	1
Constipation .....	3	Torsio uteri .....	1
D.D.T. poisoning .....	2	Wounds .....	1
Enteritis: infectious .....	6	Unknown .....	11
Fracture .....	1		—
Mange (notœdric) .....	8	TOTAL ..	97
Nasal catarrh .....	2		—

# H O R S E :

Box spavin .....	1	Phlegmosis .....	2
Botulism (mule) .....	1	Wounds .....	1
Iliac thrombosis .....	1	Unknown .....	3
Interdental necrosis .....	1		—
Jugular phlebitis .....	1	TOTAL ..	49
Horsesickness .....	9		—
Horsesickness immunisation	29		

It will be evident from the figures given that small animals make up the bulk of the practice: dogs 79% and cats 4%, while large animals: bovine 15% and horses 2%, play a somewhat minor rôle so far. Usually there is quite a fair sprinkling of pigs, milch goats, an occasional sheep and a variety of miscellaneous pets, like budgies, pigeons, tame meercats and monkeys, etc. For some unaccountable reason these apparently must have been remarkably healthy during the year under review. In regard to the type of cases encountered, it will be seen that specific infectious diseases, diseases of the skin, digestive and urinary system as well as minor surgical operations make up the bulk of the work.

## SEASONAL INCIDENCE OF SOME DISEASES

We have taken the two major diseases of the dog, viz. Biliary Fever and Distemper, and two subsidiary ones, Pharyngitis and Eczema (figures of which appeared sufficiently high and significant) and plotted these in the form of a small graph to show their season incidence. As was to be expected, Biliary Fever makes a beautiful peak in summer at the height of tick activity, while Distemper is rife for a similar period in winter.

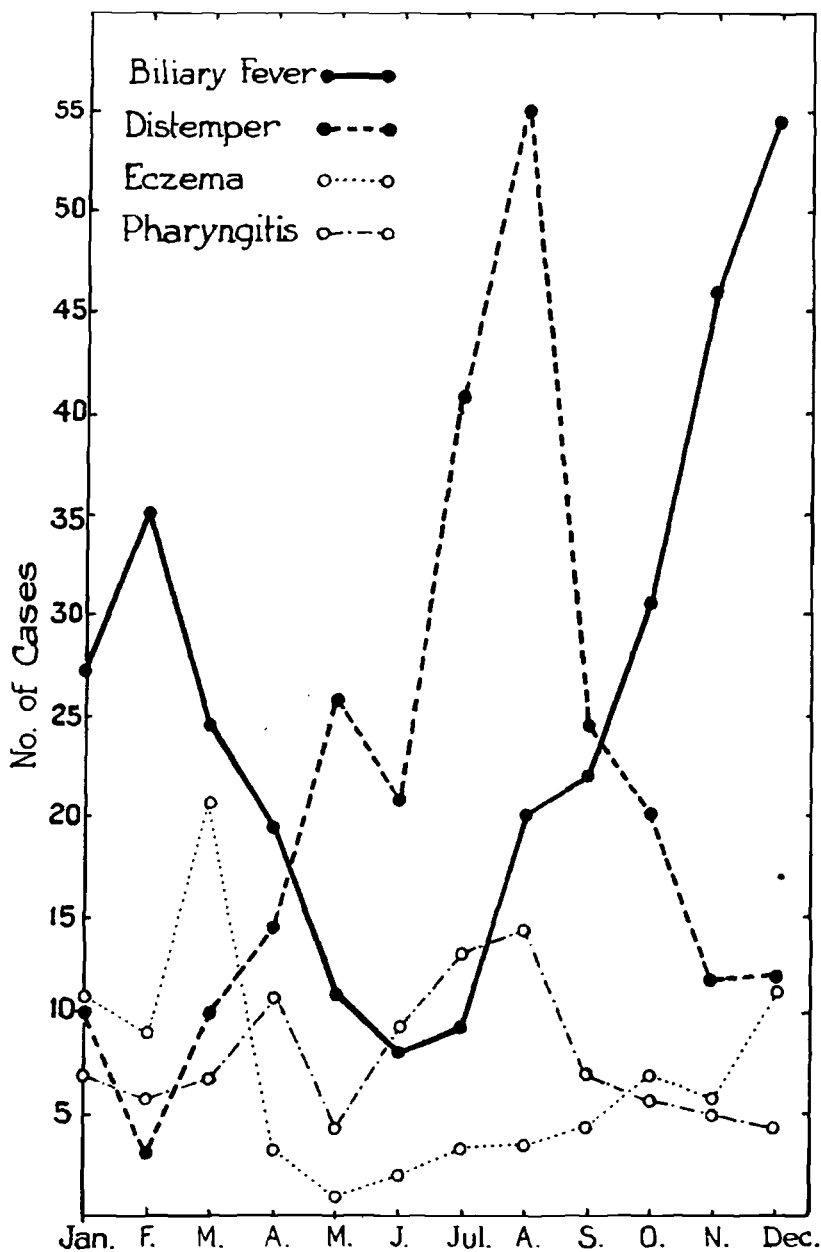
• From the private practitioner's point of view, this state of affairs is very satisfactory, as it results in a more even distribution of work throughout the year. The shape, height and location of these peaks probably differ from year to year according to climatic variation.

Pharyngitis comes into this graph on account of its likely connection with Distemper. It is interesting to note how small rises in this curve appear just before peak periods in Distemper. Our theory is that a number of essential Pharyngitis cases occur more or less throughout the year. At certain times, however, when Distemper infection is widespread, many cases are diagnosed as simple Pharyngitis (Distemper precursor) which would later develop into Distemper proper; hence the variations in curves tending to anticipate that of Distemper.

The curve for Eczema is somewhat disappointing. Although a seasonal occurrence for this disease is well known, we had hoped that it would be more pronounced than it is.

We believe that observations of this nature, comprising larger numbers, or running over several years, especially if correlated with climate, would yield far more interesting and significant results. This would naturally apply to seasonal diseases of large animals as well.

TABLE II



*Distemper:*

Our routine treatment for distemper is as follows:— When presented in the earlier stages, even when only a well-marked pharyngitis is present, we give a course of three injections of a polyvalent bacterial catarrhal vaccine in progressively larger doses at two-day intervals. At the same time we prescribe sulpha drugs, for preference the triple combination — diazine, merazine and thiazole. This is, of course, the only stage of the disease at which one can hope to influence its course beneficially and we have had more encouraging results with this method than when using hyperimmune serum. For intercurrent complications like enteritis and broncho-pneumonia, we use thalidine, and procaine penicillin in oil. Our repeated attempts to alleviate the encephalitis form have proved futile, and when the dog is only 3-5 months old we advise early destruction.

*Prevention.* We have had fairly satisfactory results with the B.W. virus serum method of immunisation. But a certain percentage of failures, together with the discovery that some batches of virus arrived here in a non-viable state, has somewhat shaken our faith in it. At the moment, we are trying out the Onderstepoort avianised strain of distemper vaccine, which so far promises exceedingly well. It is too early to say to what extent it will be successful.

*Biliary Fever:*

Our standard treatment is phenamidine 5%, 1 cc. per 10 lb. body weight, given on two consecutive days. We find the smaller initial dose less liable to produce shock and collapse, while it is quite sufficient to bring the temperature down. The two doses together thus increase the amount of drug given, but spreads it over a longer period of time. Above all, however, we find it an advantage to see the patient the next day, especially those cases in which the temperature has not come down and in which presumably the parasite may be drug fast, or in which additional treatment may be desirable. For relapses we give trypan blue or euflavine intravenously, but the question of repeated relapses still gives cause for much anxiety and has not yet to our knowledge been satisfactorily solved.

*Moist Eczema:*

In our hands, this condition yields usually quite readily to the treatment below:—

1. Clip hair short.
2. One application of camphor-phenol aa. This allays the itch and by coagulation produces a protective membrane on the raw surface.
3. Daily application of flavoline (a mixture of kaolin 60, sulphanilamide 15, boracic 15, pyridine 5 and acriflavine 0.5) in cod liver oil favours regeneration of the skin in a few days.
4. Diet — advise the omission of wheaten foods like bread, biscuits, etc.

## LARGE ANIMAL *versus* SMALL ANIMAL PRACTICE

Some criticism and even ridicule has been levelled at private practitioners for giving so much attention to domestic pets. The implication being that they could *more profitably* (for the farmer presumably) employ their time and energy treating large, and, therefore, more useful animals.

The impression has been created that small animal practice consists of ministering to the fads and fancies of affluent or eccentric lapdog owners. Many of those who believe this seem to fail to realise that by far the majority of people who own dogs are quite sane and reasonable and that their everyday life is the richer for the companionship and pleasure they derive from their pets.

Many dogs, of course, are kept for utilitarian motives such as sport, and especially as watchdogs. The latter in these days of all too common burglaries is indeed an insurance of no mean value. The point at any rate is that these people are prepared to pay for the treatment of their animals, and, what is more, as taxpayers they are entitled to, and should receive, a greater share of service in the State-sponsored programme of Veterinary Research than they do at present.

Actually, there are only a few practitioners who are privileged enough to be able to confine their work to small animals. The majority are quite content and even forced to combine both large and small animal practice and in so doing render an all-round service to their community. There again the townsman makes up to a certain extent the loss incurred in attending the animals of farmers, because it is a recognised and accepted fact that the fees usually charged do not adequately compensate for time employed in travelling long distances.

## THE NEED FOR RESEARCH

In conclusion, just a few words on research in relation to private practice. If private practice is to continue at the highest possible level, then it is imperative that it should keep abreast of the rapidly advancing progress of Veterinary Science.

This is being done to a limited extent by all too rare personal visits or contacts overseas and by reading the available veterinary literature. It can and should be done also to a far greater extent than at present, by frequent interchange of ideas at our professional meetings or through the medium of our excellent journal. We should get away from the mistaken idea that only accredited research workers can publish the results of their labours. There must be numbers of useful observations and experiences in the everyday treatment of disease, refinements of technique — simplifications and improvements in many spheres, labour-saving devices, etc., which would and should be shared for the common good and advancement of our work.

But all that is still not enough. We have our own particular prob-

lems also, which can not be solved in the field or in practice. They can be solved only if we can enlist the whole-hearted collaboration of our research colleagues and those under whose authority they work.

We are, indeed, fortunate in already having in this country a world-famed Research Institute at Onderstepoort. The fact that solutions have been found there to many of our more important veterinary problems shows that the facilities and the brains are there. Unfortunately, however, with the notable exception of avianised distemper vaccine, little or no systematic research has been made into the diseases of small animals.

There are several conditions which urgently require intensive study because, at the moment, we have no adequate or satisfactory treatment for them. To mention only a few:—

1. Nephritis in the dog, its cause and treatment.
2. Feline enteritis.
3. Eczema in the dog.
4. Several skin diseases in the dog, mostly unnamed because their etiology and pathology is so confused.
5. Rickettsiosis.
6. Infectious oral papillomatosis in the dog.

Not forgetting, of course, yet more research to improve treatment and prevention of the more prevalent diseases like Piroplasmosis, Distemper, etc.

We are confident that many of our colleagues will endorse the sentiments expressed above, and we trust that they will make individual and concerted efforts and take every opportunity in pressing for something to be done in this respect.

### THREE GENERATIONS OF VETERINARIANS

Dr. John Robinson, who graduated at Onderstepoort in December, 1949, is the third generation of veterinarians in the family.

His grandfather, J. A. Robinson, M.R.C.V.S., came out to South Africa during the rinderpest outbreak of 1896. Later, he was lecturer in Veterinary Science at Grootfontein and died in 1915.

John's father, Dr. E. M. Robinson, of Onderstepoort, is well known to most of his colleagues.

John has taken up an appointment with the Government of Southern Rhodesia, and is stationed at Bulawayo.

## BOOK REVIEWS

*EXPERIMENTAL SURGERY*, including Surgical Physiology, by J. Markowitz. Second Edition (1949); published by Baillière, Tindall & Cox, London. Price 40s.

The second edition of this remarkably stimulating book has been received. No better indication of the scope of the book can be given than by quoting from the Foreword by C. F. Schlotthauer, D.V.M.:—

‘This book fills an important need for laboratories using animals for medical research purposes. In it are described nearly all of the common operative procedures used in the experimental laboratory. It also should be useful to the veterinary surgeon, because the operative procedures described, although designed for experimental purposes, employ surgical technics which have been shown to be suitable for use on animals. Veterinarians should find the entire book interesting and helpful, but the chapters dealing with equipment, technic, sutures and instruments, thoracic surgery, intestinal obstruction and intestinal surgery will be of special interest to them.’

The author adopts what he himself describes as “the discursive style” which is, in fact, a clear, personal and often highly amusing manner of writing, a most refreshing thing to find in a text-book. For instance, in discussing the importance of the rôle of the liver in the metabolism of ethyl alcohol, Markowitz states “the liverless dog stays cheerfully drunk on a quantity that merely enlivens his intact brother. Many a patient with hepatic cirrhosis has discovered with mixed feelings that his capacity was not what it was in his hey-day.”

Physiology is usually classified as a pre-clinical subject, which, to the majority of students means an examination which must be passed before he will be allowed to study surgery and medicine. For this regrettable attitude the student is not entirely to blame. Modern physiology has lost much of the spirit of adventure and common sense approach which characterised it in the days of Claude Bernard. It has tended to become a class-room subject, bogged down with many bio-physical considerations and enlivened only by exasperating, sooty, and somewhat irrelevant experiments conducted on the gastrocnemius of the frog.

The emphasis on surgical physiology brings new light onto an old subject. It is not advocated that second-year physiology students should perform highly technical experimental operations, but it is suggested that the function of the liver can be more interestingly approached from the results of an Eck fistula than through the medium of a discourse on the migration of the ions and the formation of a Donnan equilibrium.

“Experimental Surgery” can be recommended to all veterinarians if only for stimulating and instructive reading. To those who practise the arts of medicine and surgery it will not only form a very useful reference book on operative technique, but the discussions will elucidate (and debunk) the rationale of many accepted curative procedures both surgical and medical. For the sake of future students, this book should be the constant companion of all teachers of physiology. — (R.C.)

*THOMSON'S ELEMENTARY VETERINARY SCIENCE*, Sixth Edition, 474 pages; revised by A. C. Duncan, F.R.C.V.S., B.Sc.; published by Baillière, Tindall & Cox. Price 15s.

Dr. Duncan has assembled much useful information on diseases and ailments of farm animals in this book. The text includes descriptions of such varied subjects as wounds, diseases and ailments of the feet, the joints and the bone; disturbances of the digestive, the respiratory and the nervous systems; the structure of the body and some of the main morbid processes; methods of controlling farm animals, and the definition of some of the more common terms used in veterinary literature.

The author has presented his subject matter in a clear and concise manner, and has tactfully avoided unnecessary detail. Yet, for a book on General Veterinary Science, no matter how elementary, the description of infectious, parasitic and deficiency diseases is disappointing. As the chief progress made in veterinary science during recent years concerns mainly these diseases, it is not clear what the author's reasons could be for not devoting more space to them.

Nevertheless, Thomson's Elementary Veterinary Science contains a great deal of very useful information and is regarded as a valuable textbook for agricultural students, farmers and stock-keepers, for whom it was primarily written. — (M.W.H.)

\* \* \*

*NEOPLASMS OF THE DOG*, by Professor R. M. Mulligan; Baillière, Tindall & Cox, London (1949); pages xi. plus 135, 59 plates; illustrated. Price 31s. 6d.

On the subject of tumours of the dog, if the reviewer were asked to say off-hand what are the highlights that come to his mind, he would reply: Firstly, the unique nature of the contagious venereal tumour as a biological problem; and, secondly, difficulties of pathological diagnosis peculiar to the canine species, including especially the question of the inter-relationship between certain sarcomas and tuberculosis in the dog.

The cardinal facts relating to the contagious venereal tumour are — or should be — well known. This problem is one which has intrigued the inquisitive, attracted the speculative, and challenged the discerning mind. The story is indeed virtually a classic and in future will find an important niche in the history of comparative pathology. The biological aspects significant in veterinary practice were worked out just after the turn of the present century and attracted among others a medical pathologist whose name was later to become famous above all others in the field of the pathology of neoplasia — James Ewing. Contributions to the literature have been made over the space of almost half a century since Sticker first described this neoplasm and so fortunately thought of trying to transmit it artificially. One might add, not only fortunately but also remarkably, since he had no reason to suspect that the tumour had arisen other than spontaneously in the particular case which he investigated.

It was Sticker also who first experienced that mental wrestle which was later to tax the brains and patience of many an experienced histopathologist confronted with sections of the contagious tumour of the dog — carcinoma? — sarcoma or what-the-deviloma?

It would have been pleasant and fitting to find in a book devoted — so praiseworthily — to the special field of canine tumours, an appreciation of the history, romance, difficulties and practical aspects of the contagious tumour problem, but the reviewer was disappointed in this respect. The

literature has not been adequately referred to, and it is not only the reviewer's theory of a relationship to heart-base tumours (Jackson, 1936) that has been omitted. The author embarks on a mistaken line of argument about the nature of the tumour cells. He fails to see that one cannot begin to deduce their nature from the cytology of the tissue (e.g. the subcutis) in which the tumours occur, for the simple reason that the cells had an exogenous origin (by grafting of tumour cells pre-existing in another individual). What is worse, Professor Mulligan, on this mistaken basis, labels the tumour "histiocytoma" — a most unfamiliar designation for the contagious venereal tumour, even if it were correct. One looks in vain in the index for a reference to the well-known terms — "venereal," "contagious," "transmissible". One finds "lymphosarcoma," but this refers only to lymphoid tumours of leucotic nature. A colleague looked into this book to see what the author had to say about the contagious venereal tumour, and after searching in vain for this or any familiar synonym, remarked, "What a pity, it has not been dealt with at all!"

Regarding this term "histiocytoma," it may be mentioned that it was Kaalund-Jorgensen and Thomsen (1937) who enunciated the theory of the reticulum cell nature of these tumours. Their work, however, is not referred to, so that it is not clear whether Professor Mulligan arrived at a similar conclusion independently of this influence. In any case, the reviewer (Jackson, 1944) was at great pains to point out the error of this and other views and to show for the first time what the cells looked like when adequate technique was employed. It was especially pointed out that the easy formalin fixation — paraffin embedding — hæmatoxylin eosin type of technique failed hopelessly to give a faithful picture of the cytology. Indeed, it is this that produces the artefacts and gives the familiar but erroneous appearances once more reproduced in Professor Mulligan's book in Plate L.

There are special pitfalls which beset the histopathologist when diagnosing tumours of the dog. Of these, the contagious venereal tumour is one. This possibility must always be remembered, when confronted with a tumour of ill-defined cell type and obscure histogenesis. But in addition, excruciating difficulties arise in the differential diagnosis from mastocytoma, especially because the specific basophil granules of the latter are only too often dissolved out by a watery fixative. These difficulties have not been appreciated. One does not gather from this work that the diagnosis of canine tumours is anything but easy and straightforward, that there is any need for caution, or any need to adapt technique to the special problems in this field. The book does indeed contain an appendix on histological technique, but here one learns merely how to do paraffin embedding, to stain a section with hæmatoxylin and eosin or a frozen section with Sudan III, or a bloodsmear with Giemsa. Surely, this appendix is a little out of place? Who is going to consult a work entitled "Neoplasms of the Dog" to obtain such information?

The second point mentioned about pathological problems peculiar to the canine species (in the first paragraph of this review) is apparently known only to a handful of pathologists — all of them, I believe, South Africans — and as this knowledge has not been published one cannot expect others to be aware of it. Nevertheless, he who blithely embarks on diagnosing obscure lesions labelled "endothelioma" (of lymphoid tissues), "reticulum-cell sarcoma" (accorded no mention by Professor Mulligan), "retroperitoneal sarcomas," needs to be warned as follows:—



Between sarcomas and tubercular lesions in the dog exists so closely graded a series of histological appearances, that after many years of experience the reviewer finds himself totally unable to draw any sharp borderline. Under certain conditions in the dog (which we understand all too little) tuberculosis assumes a highly and even exclusively proliferative nature, regressive changes being either overshadowed or even conspicuously absent. There are lesions exactly simulating neoplasms both macroscopically and microscopically and giving rise to metastases indistinguishable from those of sarcomata. The microscopic picture may give no hint to stain and search for acid-fasts. And even if this is done there is no assurance of their presence being disclosed. Yet on sub-inoculation a positive biological test may be observed, and in the experience of Dr. E. M. Robinson (of the Onderstepoort Research Institute) and the reviewer, if typing is undertaken one may expect to find the human type. The reviewer does not pretend to understand the ultimate implication of these facts for the relationship between atypical tuberculomas and sarcomas in the dog (and for all we may know, perhaps in other species), but facts they are and it is a duty to warn anyone dealing with canine pathology about their existence.

There is also much that can be said in praise of Professor Mulligan's work. He certainly has performed a labour of love, and has had the enthusiasm to collect a truly enormous collection of canine tumours (1,000 cases) largely by encouraging practising veterinarians to collaborate. The chapter on mammary tumours and tumours of the gonads (*cave*: look under "Neoplasms of Endocrine Glands") are the best. The classification here is logical and systematic and this is a very useful summary of the tumour types encountered, adequately illustrated save for the lack of a picture of a theca cell ovarian tumour.

Among miscellaneous points deserving comment are the following:— It is interesting that no less than four cases of primary liver cell carcinoma were encountered, a tumour which, in contrast with cholangiocellular carcinoma has been considered (perhaps erroneously) to be of great rarity in the dog. Professor Mulligan has probably encountered more intracranial tumours in the dog than has any other observer. The reviewer would have welcomed his going into details about them. It is not clear how one is to distinguish basal cell carcinoma (Plate XIX A) from sweat gland carcinoma (Plates XXI B and C). The diagnosis of basal carcinoma in the case of Plate XVIII D is open to some doubt as the tumour apparently shows pearl formation, described, however, as "mimicking of hair follicles."

There is no doubt that this work will have its uses and that veterinary pathologists the world over will require to refer to it and will often find it helpful. On the subject of breed peculiarities in susceptibility to neoplastic conditions nothing comparable has been published in respect of any domesticated species and this feature will be of interest to all practising veterinary surgeons.

#### REFERENCES TO LITERATURE

- BEEBE and EWING (1906): *J. Med. Res.* 15: 209 - 277.  
 JACKSON (1936): *Ond. Jl.* 6: 387 - 413.  
 JACKSON (1944): *Ond. Jl.* 20: 97 - 118.  
 KAALUND-JORGENSEN and THOMSEN (1937): *Z. f. Krebsf.* 45: 385 - 398.  
 STICKER (1904): *Z. f. Krebsf.* 1: 413 - 444.

C.J.

## SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION

*Minutes of Council Meeting held at 2.15 p.m. on February 2, 1950,  
in Prudential House, Pretoria.*

*Present:* J. H. Mason (President), . G. Boswell, P. S. Snyman, G. D. Sutton (Hon. Treasurer), R. Clark (Editor), S. W. J. van Rensburg, R. A. Alexander, H. P. Steyn, A. D. Thomas, G. Pfaff, W. D. Malherbe and M. de Lange (Hon. Secretary).

*Apologies:* P. J. du Toit and A. M. Diesel.

1. *Minutes of Meeting* held on November 10, 1949. Adopted.

2. *Arising from these minutes:*—

(a) *Restriction on the sale of drugs:* Letter from Secretary for Health re sale of hormones to public. Decided to collect as many instances as possible on the indiscriminate sale of hormones to the public and to submit this information to Secretary for Health.

(b) *Refresher Courses at time of General Meetings:* Report by sub-committee. Two courses proposed. Decided that details be published in *Journal*, and depending on response from members, either one or both courses to be held during the week preceding General Meeting, i.e. some time between 15th August and 15th September, 1950. A fee of one guinea per member to be charged to cover incidental expenses.

(c) *P.D.S.A. Cape Town — Appointment of Part-time Veterinarian:* Report of General Purposes Committee. After discussion decided to advertise post on terms of contract proposed by P.D.S.A. Suitable candidate to be selected, and if necessary interviewed, by committee consisting of Drs. Mason, Van Rensburg and Thomas.

(d) *P.D.S.A., Johannesburg:* Dr. Malherbe reports that person concerned had not responded to request to give written statement. Registrar R.C.V.S. to be informed accordingly.

(e) *Complaint of Member:* In view of Dr. Diesel's absence, matter held over for report to next General Meeting.

(f) *Dog Licences in Province of Transvaal:* No reply received from Provincial Secretary. Reminder to be sent. Secretary reports on phone conversation with Chief Provincial Inspector. Decided to watch situation closely for further developments and to obtain copy of Draft Ordinance if and when available.

(g) *Financial Matters:*

(i) *Financial Assistance to dependents of deceased Members:* Report by Finance Committee on three cases, one of which requires immediate assistance. After discussion recommendations of Finance Committee approved by Council.

(ii) *Assistance to Member:* Investigations proceeding and report to be submitted to Council in due course.

(iii) *Subscription two Members:* Council's approval obtained to withhold accounts for subscriptions in the case of two members.

(iv) *Students' Loans:* Proposed action of Finance Committee in case of two loans approved of by Council.

- (v) *Maud Bales Scholarship*: Available for 1950. Secretary of Faculty to be requested to submit details of scholarship to all South African Universities for publication in their calendars. Applications for current year from among students at present studying at Onderstepoort to close on March 31, 1950. Board of Trustees to be informed accordingly.
- (vi) *Benevolent Fund*: Proposed scheme for collections by members approved by Council. Finance Committee to obtain sanction from D.V.S. before scheme is published in *Journal*.
- (vii) *A.E. and C.I. Prize*: Faculty and students to be notified of prize.

3. *Co-operative Employment of Veterinarians*: Letter from Registrar of Veterinarians to be circularised among Council members and to be discussed at next meeting (copy of letter enclosed).

4. *City of Bloemfontein: Appointment of Consulting Veterinarian*: After discussion decided that no further action is called for.

5. *Facilities for Slaughtering of Poultry*: Letters Town Clerks Pretoria and Durban received. Matters raised already being dealt with by D.V.S. Noted and receipt to be acknowledged.

6. *Writing of Articles by Veterinarians in Lay Press*: Instances cited. Matter receiving attention of Veterinary Board.

#### 7. *Correspondence*:

- (i) *Letter Secretary for Agriculture re resolution 1948 General Meeting, etc.*: Contents noted and receipt to be acknowledged.
- (i) *Letter Health Officials' Association re Compulsory Pasteurisation of Milk*: Noted and receipt acknowledged.
- (iii) *Letter Secretary, Cape Western Branch re certain Proprietary Product* and report on analysis to be submitted to D.V.S. (Registering Officer).
- (iv) *Letter Dr. Pullinger re National Health Council*: Acknowledge receipt and submit to Dr. Snyman for report next Council Meeting.
- (v) *Letter Secretary, Pretoria Branch, re Petrol Saving*, to be submitted to Petrol Saving Committee for further action.
- (vi) *Letter Dr. Groenewald re Trace Element Deficiency*: Dr. Groenewald to be requested to write short article for *Journal*.
- (vii) *Letters D.V.S. re Interim T.B. Scheme*: Noted. Acknowledge receipt. Notice already in Sept., 1949, issue of *Journal*.

#### 8. *General*:

- (i) *Amendments to Veterinary Act*: Memorandum by Dr. Pfaff to be circularised to members of Council, Veterinary Board and Faculty. Approved.
- (ii) *Appointment of Lecturer by Pretoria University*: Resolved that letter expressing the views of Council be sent to University, with copies to Minister and Faculty.

The meeting closed at 5.45 p.m.

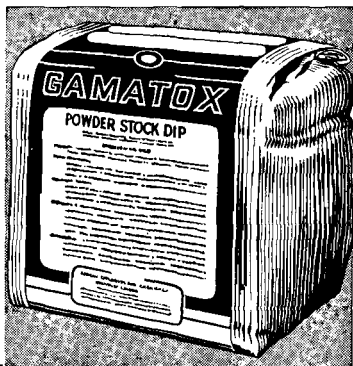
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*Hon. Secretary, S.A.V.M.A.*

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# A REVIEW OF OUR PRESENT KNOWLEDGE OF FACTORS AND DRUGS INFLUENCING RUMINAL MOTILITY.

R. CLARK,  
Onderstepoort

## INTRODUCTION

The presence of regular and strong waves of contraction of the reticulo-ruminal sac are essential to the proper functioning of the fore-stomachs. These movements not only mix the ingesta, so contributing to the mechanical breakdown of the food, but are also essential to the act of rumination and to the passage of ingesta down the alimentary tract. They are also associated with the belching reflex.

The treatment of ruminal stasis is one of the most common tasks of the veterinarian in cattle practice. Although great advances in ruminal physiology and pharmacology have been made in recent years, the author feels that many therapeutic measures used to-day are irrational in that they are primarily based on the use of ruminatorics, many of which are of doubtful value, rather than on the recognition and correction of the cause. The objects of this paper are, therefore, to review our present knowledge of the following:—

- (a) The physiology of normal ruminal motility.
- (b) The causes of ruminal stasis and their correction.
- (c) The relative values of the drugs advocated as ruminatorics.

## THE PHYSIOLOGY OF RUMINAL MOTILITY

While at rest, the rumen normally shows a wave of contraction every 40 to 60 seconds. This rate is increased during feeding and rumination. For details of the mechanics of the contraction the reader is referred to the publications of Schalk and Amadon<sup>18</sup> and of Phillipson.<sup>16</sup>

These contractions can only take place in response to motor stimuli via the vagi nerves. Hoflund<sup>12</sup> has shown that by severing branches of the vagi supplying different portions of the reticulo-ruminal sac, permanent paralysis of the whole organ or of selected portions can be brought about.

The passage of ingesta into the omasal groove is dependent on the presence of ruminal motility<sup>19</sup> and on the integrity of the vagal branch supplying the musculature of the reticulo-omasal office. If this nerve supply is interrupted stenosis of the orifice results.<sup>12</sup>

We must assume that the vagal stimuli originate in some centre (we may designate it the "*ruminal centre*"), which discharges a series of impulses regularly much as does the respiratory centre. To continue

with the analogy, it can be assumed that this ruminal centre either possesses an inherent rhythm, influenced by humoral and nervous factors, or that it discharges only in response to specific stimuli. The extraordinary regularity of the ruminal movements under a given set of conditions certainly favours the former hypothesis.

#### PHYSIOLOGICAL FACTORS AFFECTING RUMINAL MOTILITY

##### (a) *Nervous Stimuli.*

Ruminal contractions are markedly accelerated during feeding by the stimulation of sensory nerve endings in the pharynx. This reflex can also be initiated by the application of certain drugs to the pharynx, such as copper sulphate<sup>17</sup> and sodium bicarbonate.<sup>19</sup> Such stimuli usually also produce a temporary closure of the oesophageal groove. The stimulation of the rumen passes off within 10 to 20 minutes after the stimulus ceases.

Stimuli may also arise in the ruminal wall itself. The regurgitation and rumination reflex has been shown to be initiated by the contact of coarse food particles with the wall of the anterior portion of the rumen.<sup>18</sup> On the other hand, sheep have been kept for long periods on hand-picked green lucerne leaves, i.e. without any roughage, without any effect on the ruminal rhythm, although rumination was suppressed.<sup>6</sup> It is known that ruminants, especially cattle, require roughage in the diet in order to thrive.<sup>7</sup>

In most plain muscle, stretch, especially if rapidly applied, is a strong stimulus to contraction. Starvation reduces ruminal motility in sheep<sup>17</sup> and the moderate distention of the rumen with gas, normally seen during active fermentation, probably increases ruminal motility. The insufflation of the rumen with oxygen and other gases has been shown to stimulate contractions.<sup>10, 6</sup>

##### (b) *Humoral Stimuli.*

It is reasonable to suppose that the ruminal centre would be affected by the composition of the blood, but little is known of this aspect of the normal physiology. The paralysing effects of artificially induced alkalosis and the intravenous injection of histamine will be dealt with later.

#### THE CAUSES OF RUMINAL STASIS

##### (a) *Mechanical.*

Peritoneal adhesions will naturally interfere with the ruminal movements and may give rise to symptoms resembling those of stasis.

##### (b) *Interference with Nerve Supply.*

Hoflund<sup>12</sup> has shown that intermittent or chronic atony, as well as chronic stenosis of the reticulo-omasal orifice can be caused by vagal branches becoming involved in lesions of peritonitis.

##### (c) *Stimuli from the Abomasum.*

Wester states: "*The motility of the fore-stomachs is chiefly dependent on the state of the abomasum. Inflammatory stimuli of the*

*abomasal mucosa, pressure on the wall due to too great a quantity of ingesta or liquid, stimulus of cold on the abomasal mucosa, all give rise to atony."*

In the present author's opinion ruminal stasis usually develops as a result of chemical and physical changes within the ingesta of the organ itself. With a normally functioning rumen it is difficult to see why any of the above conditions, apart from inflammation, should arise in the abomasum.

#### (d) *Changes in the Ruminal Ingesta.*

(i) *Physical:* Drying out of the ruminal ingesta is a common cause of stasis in South Africa during the winter months. The essentiality of an adequate water content within the rumen has been stressed by Schalk and Amadon<sup>18</sup> and by Clark.<sup>4</sup> Ruminal contractions simply mould dry ingesta into a compact mass. Under such circumstances, rumination and the passage of ingesta are rendered impossible and the flora die out. Eventually the ruminal movements cease, due to lack of stretch stimuli and exhaustion.

At the time of an inadequate water supply, the feed is frequently highly fibrous and deficient, contributing further to depletion of the flora and the tendency to impaction.

(ii) *Chemical:* Clark and Lombard<sup>3</sup> showed that the intravenous injection of alkali resulted in immediate ruminal paralysis. The dosing of alkali into the rumen produced the same effect. As the rumen so paralysed responded normally to stimuli from the pharynx (feeding) and to the injection of carbamylcholine, it would appear that an alkalosis suppresses the ruminal centre.

Alkalinity of the rumen may develop by either over- or under-feeding of certain dietary ingredients.

The ruminal flora normally digest a portion of the dietary protein for their own use, but in the presence of excess protein and relative deficiency of sugars and starch, the process of deamination predominates, giving rise to the formation of ammonia and a consequent elevation of the ruminal pH. The resulting alkalosis may not only cause ruminal atony, but may also interfere with calcium metabolism and so give rise to milk fever or acetonæmia or both.<sup>8, 13</sup>

On the other hand, a marked deficiency of sugars or starch in the diet renders the natural formation of organic acids within the rumen impossible and the ingesta may go alkaline due to the accumulation of salivary bicarbonate. This alkalinity may also give rise to ruminal stasis and even "under-feeding acetonæmia."<sup>13</sup>

The maintenance of a slightly acid reaction within the rumen is, therefore, essential, not only to normal digestion but also to the whole metabolism of the ruminant animal.

#### (e) *Thermal Effects.*

Exposure to cold is frequently given as a cause of ruminal stasis. The dosing of large quantities of iced water has, however, failed to influence the ruminal motility in sheep.<sup>17</sup>

(f) *Psychic Effects.*

Psychic effects such as the removal of a new-born calf have also been mentioned, but the importance of such factors is difficult to assess.

(g) *Histamine.*

In 1942, Dougherty<sup>10</sup> reported that the injection of histamine caused ruminal paralysis. Clark has recently confirmed this finding.<sup>5</sup> The rôle of histamine in naturally occurring ruminal stasis is not known. Histamine, and probably other toxic amines, might be produced in the rumen by the bacterial breakdown of protein. In fact, Dougherty<sup>10</sup> has demonstrated that the histamine content of the rumen is increased by increasing the protein content of the food. Histamine might also be formed in the tissues during allergic reactions. If it be proved that histamine plays a part in the ætiology of certain diseases of ruminants, the use of anti-histamine drugs in bovine practice will be placed on a rational basis.

(h) *Febrile Diseases.*

Ruminal stasis frequently occurs as a complication to febrile diseases such as anaplasmosis, where it is almost invariably present. The cause of the stasis is not known, but its treatment forms an essential part of the treatment of the disease.

(i) *Specific Poisons.*

Ruminal atony may be brought about by many plant poisons.

(1) *Atropine:* Atropine inhibits the parasympathetic impulses and its administration results in immediate stasis of the whole digestive tract.<sup>6, 17</sup>

(2) *Prussic Acid:* Small doses of prussic acid cause ruminal stasis before any other symptoms are discernible.<sup>2, 17</sup>

(3) *Icterogenin:* This active principle of *Lippia rehmanni* causes ruminal stasis as well as paralysis of the biliary tract, regurgitative jaundice and photosensitisation.<sup>17</sup> *Lantana camala* also possesses an active principle of similar action, and there is little doubt that *geel-dikkop* is caused by an icterogenic toxin which develops in *Tribulus terrestris* under certain conditions.

## THE TREATMENT OF RUMINAL STASIS

Rational treatment can only be based on the diagnosis of the underlying cause. As already stated, in the author's opinion, this can usually be found in errors of diet as reflected in changes in the physical or chemical composition of the ruminal ingesta. It must also be remembered that stasis in itself, from whatever cause, rapidly leads to changes in the ingesta, thus setting up a vicious cycle.

In the majority of cases, therefore, the first step, in rational treatment is to restore, and endeavour to maintain, conditions within the rumen compatible with normal function. This will briefly consist of:—

- (1) The dosing of large amounts of water and massage where desiccation and compaction have taken place.



- (2) The adjustment of the pH by acidification. This can best be done by adding 50-100 cc. glacial acetic acid ( $1\frac{1}{2}$  to 2 bottles of vinegar) to the water dosed. This amount should preferably be divided into two doses at 3 to 4-hour intervals.
- (3) The maintenance of acid fermentation by adding 1 to 2 lbs. of sugar to the drench.
- (4) The restoration of the flora where this has died out due to protracted dysfunction. This can best be achieved by dosing fresh ruminal ingesta from a normal animal. It is of interest to note that the beneficial effects of such treatment has been known for centuries to the peasants of certain parts of Sweden, whose home remedy for ruminal dysfunction has long been the dosing of gruel fermented by the addition of a cud from a healthy animal. The practice has been taken over scientifically and cakes of dried cultured ruminal organisms are now supplied to veterinarians.<sup>14</sup> Even compressed bakers' yeast is effective, and kaffir beer should contain almost all the ingredients required of a good ruminal tonic.
- (5) The restoration of motility by the judicious use of ruminatorics and purgatives.

#### RUMINATORICS AND PURGATIVES

The following review of the pharmacological action of drugs used as ruminatorics and purgatives on ruminants is derived from the works of Quin and Van der Wath,<sup>17</sup> Dougherty<sup>10</sup> and the present author. Unreferenced statements refer to the author's unpublished experiments. The drugs will, as far as possible, be dealt with according to their mode of action.

##### 1. *Parasympathetic Stimulants.*

As the whole gastro-intestinal tract is activated by the parasympathetic system, these drugs are powerful ruminatorics and purgatives. It must be remembered that they also have the following actions:—inhibition of the heart, vasodilation and contraction and increased secretion of the bronchioles. The vasodilation results in a drop in blood pressure, and the resultant pressor reflexes usually cause an actual acceleration of the heart. The circulatory and respiratory systems are thus acutely embarrassed and the drugs are extremely dangerous in anæmic or debilitated animals. They can all be reversed extremely rapidly by the administration of atropine. Small divided doses should be given and the effects on the heart and respiration kept under observation. Drugs belonging to this class are:—

(a) *Carbamylcholine-Chloride*: (For doses and uses, see this journal, Vol. 20, p. 36.) Produces marked stimulation of salivation and ruminal motility and purgation within 10-20 minutes. Effects last 4-6 hours.

The drug is absolutely contra-indicated in prussic acid poisoning, as it acts synergically to produce circulatory collapse as well as to increase the duration and severity of the ruminal paralysis.<sup>2</sup>

(b) *Physostigmine* (Eserine): This drug acts by inhibiting the enzyme which normally destroys acetyl-choline, thus prolonging and increasing parasympathetic stimulation. Its uses and contra-indications are as for carbamylcholine.

(c) *Neostigmine*: This is a synthetic anti-cholinesterase resembling physostigmine but differing from it in that it appears to have a more selective action on the sacral portion of the parasympathetic system. The actions on the heart and lungs are minimal, while the ruminatoric effect is milder but adequate. Purgation is good, with few signs of colic. In the author's opinion this is the drug of choice whenever the extra expense is warranted.

(d) *Pilocarpine*: Dougherty<sup>10</sup> and Amadon<sup>1</sup> report very little reaction in cattle. Sheep show small rapid contractions of the rumen, copious salivation, frequent urination and defæcation.

(e) *Arecoline*: Large doses (34 mgm. or over) cause inhibition of ruminal movements in cattle, while small doses (4-6 mgm.) cause stimulation.<sup>1</sup> In sheep, inhibition was obtained by doses of 10 mgm. or over, but no reaction was noted from smaller doses. In a few instances a delayed secondary stimulation followed inhibition about two hours after injection. Arecoline would not appear to be a reliable ruminatoric.

(f) *Acaprin*: The marked vasodilation and purgation which follow the administration of acaprin to horses and dogs for the treatment of babesiosis indicate a parasympathetic stimulation. No ruminatoric action could be demonstrated in sheep.

## 2. *Strychnine*.

*Nux vomica* has long been used as a ruminatoric, but doses up to 3 gms. to merino sheep were entirely without effect over a period of 5 hours.

Strychnine sulphate (3 mgm. subcut.) caused an increase in the excursion, but not in the frequency of contractions.

Discussing the action of strychnine on the motility of the alimentary tract in man, Cushney<sup>9</sup> states: "*Any change which is produced by the small doses usually employed is inconsequential and could readily be obtained by other less dangerous drugs.*"

The beneficial effects obtained from the use of *Nux vomica* in cases of ruminal stasis and inappetence are probably due to the stomachic and tonic actions rather than to any ruminatoric effect.

## 3. *Ammonium carbonate*.

No action could be demonstrated with the advocated doses in sheep.

## 4. *Tartar Emetic*.

Referring to cattle, Dougherty<sup>10</sup> reports as follows: "*Tartar emetic was practically without effect on one animal, but in the other increased the strength of contractions slightly when 150 grains in solution were injected into the rumen. Recordings were made for three hours. Little change was noted when a 120-grain dose was injected in the same animal.*"

No effect could be demonstrated in sheep.

## 5. *Veratrine*.

Wester<sup>19</sup> regards *Pulv. veratri albi* as an excellent ruminatoric. The dose for this purpose to cattle is 5 gm. administered in water or thin gruel. He also uses doses of 10 gm. given in milk or sodium bicarbonate to produce vomition in cases of overloading of the rumen.

These results have been confirmed in sheep using *Veratrina hydrochloric* injected intravenously. A dose of 5 mgm. caused marked stimulation of the rumen within 20 minutes, and the action lasted for nearly two hours. When given 10 mgm. i.v., sheep showed copious vomition.

## 6. *Oxygen Insufflation*.

Working with cattle, Dougherty<sup>10</sup> reports that oxygen insufflation of the rumen markedly increases motility. In fact, he states: "*The increased activity was more marked than that obtained by the use of any of the drugs tried in these experiments.*" It was also found to stimulate the rumen paralysed with either atropine or epinephrine. The action is apparently partially mechanical, because stimulation was also obtained with CO<sub>2</sub>, CH<sub>4</sub> and Air. Oxygen, however, appeared to produce a more marked effect.

Numerous tests have been made on sheep, but the stimulation ceased as soon as the intraruminal pressure returned to normal. Neither could any beneficial effect be obtained in cases of atropine or alkali-produced atony. This may be due to a species difference.

The use of the procedure in cattle should perhaps receive more consideration.

## 7. *Ethyl Alcohol Intravenously*.

The use of ethyl alcohol intravenously has recently been advocated for the treatment of ruminal atony occurring as a complication of anaplasmosis in cattle.<sup>11</sup> The authors claim success, although they state that the treatment does not affect the rumen of normal cattle.

The treatment was tested on normal sheep, the dose being 5 to 6 cc. of 95% alcohol or about one-tenth of the dose recommended for cattle. Very irregular results were obtained in six trials, which can be summarised as follows:—

Time after dosing.	Effect (6 Trials).
(minutes)	3 Slight acceleration.
5-10	2 No change.
	1 Complete inhibition.
10-20	All normal.
30-60	General reaction in all cases. Restlessness, dyspnœa, cramps of abdominal muscles. Retardation of ruminal movements and irregularity of strength followed by:—
	3 Complete inhibition.
	2 Retarded and irregular.
	1 Normal.
(hours)	4 Normal.
2-4	2 Inhibited.
6	5 Normal.
	1 Inhibited.

As will be seen the drug tended to cause inhibition rather than stimulation. Neither could any beneficial effect be demonstrated in sheep in which the rumen had previously been paralysed by alkalinisation.

#### 8. *Purgatives.*

Many substances known to have a purgative action are advocated for use in cases of ruminal atony. Several such drugs have been tested for ruminatoric action on fistula sheep, but none was found to have any specific stimulating effect on the fore-stomachs. In view of Wester's<sup>17</sup> opinion that the motility of the rumen is markedly influenced by disturbances further down the alimentary tract, this does not necessarily indicate that their use is irrational. The main drugs under this class are:—

(a) *Sodium sulphate*: Webster considers this to be the best purgative for cattle, "*especially as it often passes directly into the abomasum if given in strong concentration and because its action is not severe.*" Neither sodium nor magnesium sulphate influenced the ruminal contractions of normal fistula sheep when introduced directly into the rumen.

(b) *Anthraquinone derivatives*: One proprietary veterinary remedy of this class was tried on fistula sheep without any demonstrable effect when administered through the ruminal fistula.

(c) *Phenolphthalein*: Dougherty<sup>10</sup> reports no effect in cattle.

(d) *Mercurochrome*: Parkin (1935)<sup>15</sup> introduced the use of mercurochrome — 220 soluble for the treatment of *Anaplasmosis*. The drug was given intravenously to cattle in doses of 0.4 to 1.3 gm. and definitely caused purgation.

When corresponding doses were tried on fistula merino sheep the drug was found to cause complete ruminal paralysis, which appeared immediately after injection and persisted for some four to five hours. In spite of this the animals showed purgation during the same period. In fact, the action on the bowels was more satisfactory than shown by any other drug in trials on sheep to date. This split action of simultaneous inhibition of the rumen and stimulation of the intestine has also been reported to be caused by histamine<sup>5</sup> and is of great physiological interest.

#### SUMMARY

1. The physiology of ruminal motility and the known causes of atony are reviewed.

2. It is emphasised that the rational treatment of ruminal stasis must be based on the recognition and correction of the cause and not merely on the administration of ruminatorics or purgatives.

3. It is argued that the physical, chemical and biological constitution of the ruminal ingesta must be restored to and maintained at normal so as to allow of active fermentation.

4. The efficacy of 16 different drugs used as ruminatorics is reviewed in the light of previous literature and of hitherto unpublished observations by the author.

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# THE CONDEMNATION OF CALVES AT JOHANNESBURG ABATTOIR WITH SPECIAL REFERENCE TO CALF PARATYPHOID

E. J. PULLINGER,  
Municipal Abattoir, Johannesburg

The object in preparing this survey has been to utilise abattoir data and abattoir material to ascertain the relative incidence of various pathological conditions to be found in slaughter calves. Special attention has been paid to the subject of Salmonellosis because of the public health significance of this infection. As a subsidiary purpose, attention has been given to ascertaining to what extent laboratory methods of diagnosing Salmonellosis can be applied to routine meat inspection.

Calves arrive at the Johannesburg Abattoir at an average rate of 150 per day. By far the majority of these are Friesland calves aged from two to six weeks and having an average dressed weight of 45 to 50 lbs., though animals are still classed as calves up to a dressed weight of 125 lbs. The reason for this heavy slaughtering of such young stock is that these animals represent the unwanted product of the dairy herds serving the Witwatersrand, the animals being marketed young because dairy farmers cannot or will not spare the milk necessary to feed them any longer.

In a country such as South Africa, which is seriously understocked with good quality cattle, the slaughter of so many dairy heifers is a national tragedy. The reason for this slaughter is an economic one in that dairymen, who farm under the relatively costly conditions imposed by municipal health departments and to whom every ton of cattle feed represents so much milk, maintain that it is cheaper to purchase heifers-in-calf rather than to rear their own heifer calves. At the 1948/49 price levels the salvage value on calves was five shillings for the hide, three shillings and nine pence for the offal, and a dressed carcase price varying with the grade but averaging out at about sixpence a pound. Thus a young calf was at that time bringing a return of about thirty-five shillings, from which had to be deducted any transportation charges together with a sum of five shillings made up of various charges and levies. The figures quoted constitute a very meagre salvage value, and yet if a calf is to be sold for slaughter it is clearly uneconomical to feed it a gallon of milk a day valued at two shillings a gallon, when any increase of weight of veal will only yield sixpence a pound.

## CAUSES OF CONDEMNATION

In Table I, figures are given showing the total slaughter figures and the condemnation figures for four years of operation. From these it will be seen that the condemnation rate does not reach two per cent

of the total, and, therefore, cannot be regarded either as a serious loss to the farmer or to the state. The main interest, in consequence, lies in analysing the figures from the public health and state veterinary medical points of view.

The chief causes for which calves are condemned are detailed in Table 2, from which it will be seen that by far the most important cause for condemnation is that of emaciation, while in some years a high proportion of calves are marketed while still too immature. These two causes which together account for from 48 to 72 per cent. of all condemnations, may both be regarded as avoidable ones and both reflect the economic position already mentioned. Bruising, which may also be regarded as an avoidable factor, ranges around the figure of 4 per cent.

From 12 to 28 per cent. of the condemnations are due to jaundice, two distinct types of which are seen. These are, firstly, pure jaundice and, secondly, jaundice associated with a general febrile condition. Pneumonia, pleurisy and peritonitis may be the cause of as much as 10 per cent. of all condemnations; these conditions are usually acute and the cause is frequently *Corynebacterium pyogenes*. The incidence of tuberculosis falls between 1 and 2 per cent., the cases seen being generally of well-developed infection in very young calves and probably nearly always congenital in origin.

The remaining conditions that account for an appreciable number of condemnations are fever and enteritis, the combined figure for these fluctuating between 4.5 and 13.5 per cent. These two conditions are purposely grouped together as they are often both present in one and the same carcase. Because fever and enteritis in calves are conditions intimately linked with Salmonellosis, an investigation has been carried out to ascertain to what extent calf Salmonellosis is likely to be encountered in South African abattoirs.

#### SALMONELLOSIS IN CALVES

Laboratory facilities being limited, it has not been possible to perform any elaborate survey, and every effort has been made to use simplified bacteriological methods designed to reduce laboratory requirements to a minimum. This procedure has had the advantage that it has been possible to work out the most simplified form of laboratory technique with a view to seeing whether bacteriological diagnosis could usefully be employed as a routine procedure for all cases suggestive of Salmonellosis. Thanks are due to Dr. Joan Taylor, of the Salmonella Reference Library, Central Public Health Laboratory, Colindale, London, for much help in both planning the technique and in rechecking the typing of atypical cultures; while thanks are also due to Dr. M. W. Henning, of Onderstepoort, for assistance in the typing of certain strains.

Bacteriological study was not confined to calves showing signs of calf paratyphoid, but was also extended to include a group of calves that had been condemned for a variety of conditions such as emaciation and pyæmia, and also to a group of apparently normal calves.

## TECHNIQUE

The medium used for isolating *Salmonella* was Desoxycholate Citrate Agar prepared in the standard manner. For testing a certain number of samples, Selenite enrichment broth was also used, but the advantages gained by this procedure were outweighed by the technical complications of preparing, storing and using this medium, and its use was therefore discontinued.

Cultures were prepared by sowing pulp from mesenteric lymphatic glands or from intestinal mucosa onto petri dishes of desoxycholate citrate agar and incubating them for 18 hours. Petri dishes were re-incubated where necessary for a further 12 hours. After incubation, likely-looking colonies were picked off onto moist agar slopes and after 18 hours' incubation, were submitted to serological classification. This was done by the slide agglutination technique, using type specific sera from the Medical Research Council Laboratory at Colindale. Strains that typed readily and fell into anticipated types such as *dublin* and *typhi-murium* received no further attention, but strains giving ambiguous results or appearing to fall into unexpected types were sent either to Dr. Joan Taylor or to Dr. Henning for checking.

## RESULTS

Calves examined bacteriologically, fell into four groups:—

- (a) Those showing enteritis with inflammation extending to the lymphatic glands and even to the whole system.
- (b) Those showing enteritis without extension to the mesenteric lymphatic glands.
- (c) Those showing no enteritis but condemned for conditions such as emaciation or pyæmia.
- (d) Those passed as apparently sound and normal.

In Table 3, figures are given relating to those calves suffering from some degree of enteritis, while in Table 4, are the records of the examination of calves showing no signs of enteritis. From Table 3 it will be seen that, out of a total of 100 carcasses condemned for enteritis, 18 per cent. were shown to be infected with *Salmonella*. Dividing this group of carcasses up into those showing enteritis with extension of inflammation and those showing enteritis without extension, the incidence of *Salmonella* infection was 15 out of 76 or 19.2 per cent. for the severe type and 3 out of 24 or 11.5 per cent. for the mild type. The group of mild cases of enteritis included ones that could readily have been overlooked at the time of inspection.

In the second series of calves, the results of which are recorded in Table 4, it will be seen that *Salmonella* were isolated from 3 per cent. of apparently normal animals. The fact that none of the group of 50 calves condemned for reasons other than enteritis was infected with *Salmonella* was probably fortuitous. In the three cases where apparently normal calves were infected, the organisms were confined to the intestinal tract and showed no signs whatever of invading the



tissues. It is worth noting that 21 per cent. of calves showing severe signs of enteritis gave entirely negative cultures on desoxycholate citrate agar.

## DISCUSSION

The figures quoted in Tables 3 and 4 referring to the incidence of *Salmonella* infection should be regarded as minimal ones, because, in the present investigation, comparatively little effort was made to isolate *Salmonella*, whereas it is well known that the more effort that is made (i.e. by multiple cultures and the use of enrichment media), the more likely are *Salmonella* to be isolated from infected carcase material. Nevertheless, even using the present simple technique, at least 18 per cent. of calves condemned for enteritis were infected with *Salmonella*. No figures are known of entirely comparable investigations done elsewhere, because nearly all the survey work that has been done has been confined to calves actually dying of disease, or has been carried out upon epidemic material. For example, Lovell and Hughes (1935), working in Southern England, reported that 2 per cent. of deaths among calves were due to *S. typhi-murium*, while Field (1949), working in South Wales, reported that 14 per cent. of all cattle carcasses sent to knackeries for disposal, were infected with *Salmonella*.

The figure of 18 per cent. found in the present survey was so high that it seemed necessary to seek an explanation. The most convincing one to be found is in the method frequently employed for marketing calves. Certain livestock agents and stock speculators operate by collecting or purchasing calves in the country areas and accumulating large consignments at suitable points. When a consignment is sufficiently large, it is dispatched to market, but during the period of collection the calves are herded together in dirty yards and are fed a thin maize gruel which such very young animals cannot digest satisfactorily. This combination of sudden weaning and dirty conditions are thought to be the factors responsible for the heavy incidence of salmonellosis.

From the point of view of the meat inspector, it is very clear that the possibility of *Salmonella* infection of calves must always be borne in mind, and carcasses even showing only slight signs of fever or enteritis should be seized and condemned. This is particularly important because veal is extensively used in preparing processed meat products such as brawn, gelatine, veal loaf, veal and ham paste, etc., where the temperature of processing, though sufficient to kill *Salmonella* bacteria, may not be sufficient to destroy pre-formed toxin. Moreover, in preparing these products, mincers and various utensils might become contaminated with *Salmonella* with devastating effect on other uncooked products such as sausages which are prepared with the same apparatus.

The finding of as high a figure at 3 per cent. of intestinal carriers among apparently normal calves is disturbing from the public health viewpoint, though it confirms the belief commonly held by epidemiologists that calves can become temporary intestinal carriers of *Salmonella* without suffering any ill-effects, and that the development of invasive infection only results from the intervention of some predisposing factor such as bad calf management. From the public

health standpoint, the fact that the transient bowel infection is light, and that any invasion of the tissues is associated with the onset of symptoms of inflammation, is reassuring. For the rest, animal entrails are always contaminated with an abundant and a varied assortment of bacteria and should at all times be regarded as a potential source of danger.

The fact that 21 per cent. of carcasses showing definite macroscopical evidence of congestion and inflammation gave negative cultures on desoxycholate citrate agar, is significant. One possible suggestion is the presence of some chemical irritant, though no such irritant has been identified. An alternative explanation is that the congestion is physiological, due to a general vaso-dilatation throughout the splanchnic area, resulting from some unidentified factor in the slaughtering procedure.

From the work that has been done in the present investigation, it seems unlikely that the routine examination of calves by bacteriological means could be a practical proposition.

Under the best conditions the primary culture of suspected material requires 24 hours, and a further 24 hours is needed for typing suspicious-looking colonies. If enrichment medium is used or if any difficulties are experienced in typing a strain satisfactorily, yet more time is required. Adding to this time lag, the fact that negative cultural findings offer no guarantee that *Salmonella* are not present, it will be realised that it is undoubtedly safer to condemn all calves showing even the slightest trace of fever or enteritis; particularly is this the case in view of the relatively low food and monetary value of a calf carcass.

#### SUMMARY

The main causes for condemning calf carcasses at Johannesburg Abattoir are listed. The majority of condemnations (i.e. from 51 to 76 per cent.) are due to the avoidable causes of emaciation, immaturity and bruising. From 7 to 14 per cent. of all condemnations are for enteritis and fever, and of these, 18 per cent. were found to be due to *Salmonella* infection. Of a group of 100 apparently normal calves, 3 per cent. were found to be carrying *Salmonella* in the intestinal tract at the time of slaughter. It is recommended that all calves showing even the slightest trace of enteritis or fever should be seized and the carcasses and offal condemned.

#### ACKNOWLEDGMENTS

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TABLE 1.

*Calf Slaughter and Condemnation Figures: Johannesburg Abattoir.*

Years	1945/46	1946/47	1947/48	1948/49
Total calves slaughtered ..	46,871	42,146	43,723	49,611
Total calves condemned ..	367	566	503	894
Per cent. condemned .. .	0.782	1.344	1.150	1.820

TABLE 2.

*Percentage of Condemnation of Calves for the Conditions Listed.*

CAUSES OF CONDEMNATION	PERCENTAGE CONDEMNATIONS			
	1945/46	1946/47	1947/48	1948/49
Emaciation .. . . .	47.8	64.0	36.0	38.5
Immaturity .. . . .	0.5	5.9	16.6	11.6
Bruising .. . . .	4.1	4.5	3.5	3.2
Gangrene .. . . .	0.5	0.3	1.0	0.9
Pleurisy and/or Peritonitis	7.1	1.2	0.8	1.1
Pneumonia .. . . .	3.3	3.5	6.1	5.7
Tuberculosis .. . . .	1.1	0.9	2.1	0.8
Pyæmia .. . . .	2.1	1.8	11.5	10.9
Jaundice .. . . .	18.3	12.7	11.7	13.3
Fever and/or Enteritis ..	7.0	4.5	9.0	13.5

TABLE 3.

*Incidence of Salmonella Infection in Slaughter Calves showing Lesions of Enteritis.*

Pathological Lesions	No. Calves Examined.	No. showing <i>Bact. coli.</i>	No. showing <i>Salmonella.</i>	<i>Salmonella</i> Types Isolated.
Enteritis with Extensions of Inflammation	76	63	15	13 <i>S. dublin</i> 1 <i>S. typhimurium</i> 1 <i>S. pretoria</i>
Enteritis without Extensions of Inflammation	24	16	3	3 <i>S. dublin</i>
TOTAL .. . . .	100	79	18	16 <i>S. dublin</i> 1 <i>S. pretoria</i> 1 <i>S. typhimurium</i>

TABLE 4.

*Incidence of Salmonella in Slaughter Calves showing no Lesions of Enteritis.*

Pathological Lesions	No. Calves Examined.	No. showing <i>Bact. coli.</i>	No. showing <i>Salmonella.</i>	<i>Salmonella</i> Types Isolated.
Jaundice, Pyæmia, Fever, etc., without Enteritis	50	34	—	—
Nil (normal calves)	100	51	3	1 <i>S. pretoria</i> 2 <i>S. dublin</i>

# THE VETERINARY INSPECTION OF TABLE POULTRY

P. S. SNYMAN.

Official ante and post-mortem inspection of poultry intended for human consumption seems, for reasons unknown, to be neglected throughout all civilized countries of the world. It is difficult to understand why this should be so, as poultry has since ancient times formed part of the source of the meat supply of man. It is even looked upon as a very essential article of food in hospitals and nursing homes. One would have expected that the inspection of poultry would have developed along with that of other meat-producing animals.

The only occasion where some form of inspection is compulsory is in connection with ritual slaughtering. The mosaic food laws forbid the eating of birds which belong to the carrion-eating species and even with the modern methods of Schacten the examination falls far short of that of cattle and sheep. In the latter, an examination of the thoracic organs takes place, while in poultry a cursory examination for blemish only is made.

In the middle ages, many edicts concerning the slaughtering of food animals were passed for health, religious and other reasons, but poultry was never mentioned, although equines and dogs were frequently dealt with.

Ostertag's "Handbook of Meat Inspection," 1902 edition, generally recognised as the most complete and authoritative treatise on meat inspection of that period, contains merely five pages devoted to poultry disease in the form of an appendix. It deals with fowl cholera and fowl diphtheria only, besides the methods of determining the age in fowls, ducks and pigeons.

The comprehensive German Imperial Law of 1900, concerning the inspection of food animals and meat, exempted fowls and game from inspection at frontiers, but the right was reserved for the Federal Council to prohibit the importation of poultry in case of outbreaks of epizootics in foreign countries.

Even in spite of the large amount of knowledge gained on poultry diseases and on the causes of "meat poisoning," modern handbooks on meat inspection still regard inspection of table poultry as of academic interest rather than of practical importance. The reason for this is that inspection of poultry for human consumption is not compulsory in any country.

The only country in which there is a recognised system of table poultry inspection is in the U.S.A., and even there it is applied on a voluntary basis only. It is only the keen competition in the poultry marketing field that has stimulated packing houses to adopt a voluntary inspection scheme under official control.

In the Union, the only inspections that are being carried out on table poultry are those under by-laws framed under the Food, Drugs and Disinfectants Act No. 13 of 1919. Inspections are limited to the examination for soundness of dressed carcasses offered for sale at municipal markets and of those displayed in butcheries and fishmongers' shops. Even this is done in a haphazard manner. The inspection of the dressed carcase only is very unsatisfactory, as a proper examination can only be made if the internal organs are present.

As no local authority takes any interest beyond stopping the sale of the unsound product, a position has been allowed to develop where dealers in table poultry slaughter when and where they like. For economic reasons, and probably also on account of ignorance, this is often done under the most repugnant of conditions.

With the development of the poultry industry in South Africa the State concentrated on the marketing of eggs. At first grading was made compulsory for the export trade and, during the war, grading and price fixation were also introduced for eggs for inland consumption. Nevertheless the trade in table poultry has escaped the notice of the Government. The determination of quality and soundness of the chickens was left to private enterprise without any supervision.

The practice of freezing poultry, plucked, but without removing the internal organs, stimulated a tremendous trade in table poultry. Firms in the cities have established depôts in the country districts where slaughtering, plucking and freezing take place before the removal of the carcase to the central depôts in the cities for storage.

Poultry is collected by country stores over very wide areas, where it is reared under all sorts of unsanitary conditions. In one of our large cities most of the poultry consumed originates from native reserves and locations where, in the absence of the most simple methods of sanitation, nature provides for the removal of waste material by calling to her aid the pig and the fowl.

Rearing poultry for the table is a very expensive undertaking and, as there is no great demand for birds of better quality, the supply is very small. Many farmers have contracts with hotels, poultry shops, etc., for the delivery of first-grade dressed poultry, but, when the supply reared by themselves becomes exhausted, they buy at markets or from natives.

The following is a summary of the more common conditions and diseases of poultry which make the meat unsuitable and loathsome for human consumption.

(1) *Pyæmia and septicæmic conditions.* Birds, unlike mammals, have no lymph glands. Consequently the process of the filtering out of organisms does not take place, so that infections are usually septicæmic in nature. Pathogenic organisms are, therefore, always present in the flesh and other edible parts of a diseased bird.

Staphylococci and streptococci, which cause pus formation and blood poisoning, are pathogenic for both man and birds. The former may cause lesions of impetigo in man indistinguishable from that in fowls. Staphylococci may become the cause of food poisoning by

contaminating kitchen utensils and the hands of the housewife. Many obscure outbreaks of food poisoning may very well be attributed to the consumption of chicken.

(2) *Salmonella*. Birds as well as animals, including man, are susceptible to intestinal disorders caused by the paratyphoid group of organisms. Excluding *Salmonella pullorum* and *gallinarum*, which are not true paratyphoids, approximately 54 of the total number of 150 *Salmonella* types have been isolated from poultry. Of the 54 species that have been isolated from fowls, 35 are pathogenic for man, and their importance in food poisoning is well known. Turkeys and ducks are very susceptible to paratyphoid infection.

Darby and Stafseth have placed the more important types isolated from poultry in the following groups:—

*Group B*: Commonly referred to in meat inspection in connection with meat poisoning as the Aetrycke group, which includes *S. paratyphi B.*, *S. typhi-murium* and *S. derby*.

*Group D*, i.e. Gaertner-enteritidis group: *S. enteritidis*, *S. eastbourne* and *S. dublin*.

*Group E*: *S. anatum*.

*S. paratyphi B* is a common cause of paratyphoid fever in man.

*S. typhi-murium* is the commonest and most widespread of the paratyphoids, and is the most common species encountered in food poisoning. It is a natural pathogen of rodents and is also found in poultry and pigs.

*S. enteritidis* and *S. dublin* have frequently been isolated from cattle and man. They cause a very severe disease in man, especially in infants.

*S. anatum* is one of the causes of "keel" disease in ducklings and is frequently found in man and pigs.

The literature of the last few years tends to stress the importance of paratyphoid disease in ducks. This is on account of the increasing number of food-poisoning outbreaks caused by ducks' eggs. Paratyphoid in birds, as in other animals, is generally a disease of the young. Adults may also become infected, but their mortality is low, the disease being more of a chronic nature with occasional diarrhoea and emaciation. Often on post-mortem examination the only lesion seen is a chronic oöphoritis.

*S. typhi-murium* and *S. enteritidis* are the chief species associated with the conditions described above. *S. anatum* has only been isolated in America.

It is, therefore, evident that paratyphoid disease is of much greater importance in poultry than is generally realized.

Although adult poultry may not be highly susceptible to *Salmonella* types pathogenic for man, they can act as carriers, and their crops may become infected with the organisms. The carcass may become contaminated if the crop is not very carefully removed.

*Mycosis-Favus or fungus disease.* This condition is common in poultry, man and animals. It is, however, not a very serious disease in fowls, and for that reason frequently escapes notice, but may cause infection in children.

#### *Diseases and Conditions in Poultry not Pathogenic for Man.*

There is a long list of infectious diseases of poultry which, although they are not transmissible to man, nevertheless render the flesh of poultry unfit for human consumption. To these belong the large group of acute febrile diseases which produce harmful toxins or metabolic products which may cause poisoning. The carcasses derived from such diseased birds decompose very rapidly.

*Neoplastic diseases.* Tumours, both malignant and benign and including leucosis, are relatively common in domestic poultry, especially fowls. The incidence reported by various workers varies from 2% in birds up to eight monthse old and 20% in the older groups. Figures obtained from egg-laying competitions (by courtesy of Prof. Coles) show that the average mortality is between 15 - 20%, of which 15% are due to tumours.

The following figures are given for the condemnations of poultry slaughtered in the United States of America under the voluntary inspection scheme:—

Year.	Amount inspected, pounds.	Amount rejected, pounds.	Rejected, per cent.
1930 .....	22 million	1 million	4.42
1935 .....	23.2 „	.5 „	2.22
1942 .....	147.2 „	1.9 „	1.3
1944 .....	178.7 „	2.3 „	1.25

The major causes for condemnations were:—

Tuberculosis .....	40%
Emaciation .....	4%
Septicæmia .....	15%
Tumours .....	4%
Leucosis .....	8%
Peritonitis .....	4%
Abscesses, bruises .....	6%
Decomposition .....	11%
Other causes .....	18%

It is reasonable to presume that had no inspection taken place a very large amount, if not all, of the meat would have been sold and eaten.

The condemnation of meat for food, for æsthetic reasons, does not necessarily imply that the meat is infected with disease communicable to man or even that it is diseased. Fatigue, asphyxiation, œdema, tumours, and other conditions may not render meat dangerous, but their presence does make it most undesirable.

In order to supply the public with good wholesome table poultry,



free from disease and other repugnant and loathsome conditions, all poultry should be slaughtered under supervision and undergo ante- and post-mortem inspection. It is the duty of every municipality to provide the necessary facilities where poultry may be slaughtered and inspected, a service which the public has the right to demand.

It is the intention of the Veterinary Division to include poultry in the regulations framed under the Agricultural Produce Export Act of 1917, governing the slaughtering and inspection of animals and meat and meat products intended for export.

With the modern technique of quick freezing, there is a decided advantage in the transporting, packing and storing of drawn poultry, apart from the fact that a ban may be placed on the importation of undrawn poultry from the Union into Great Britain on account of Newcastle disease.

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# TUBERCULOSIS IN PIGS CAUSED BY M. TUBERCULOSIS VAR. HOMINIS

P. J. J. FOURIE, G. J. DE WET and G. C. VAN DRIMMELEN,  
Onderstepoort.

According to Topley and Wilson (1948), human tubercle bacilli are pathogenic for pigs, but are less virulent than the bovine type for this species. The human tubercle bacillus is, however, only occasionally found in pigs. Young pigs fed with human bacilli may develop extensive glandular and pulmonary disease.

Avian tubercle bacilli do not give rise to progressive disease in pigs, but they may multiply and become disseminated throughout the internal organs.

Cornell and Griffith (1930) review work done in England, where it was shown that 258 cases of proved tuberculosis in pigs examined at abattoirs consisted of:—

- 178 cases of infection with the bovine type,
- 70 cases of infection with the avian type,
- 50 cases of infection with the human type,
- 4 cases of mixed infection, bovine and avian,
- 1 case of mixed infection, bovine and human.

Outbreaks of tuberculosis in pigs in South Africa are not uncommon and usually the lesions are of a localised nature in the mandibular or pharyngeal glands. Local abattoir reports give the following returns:—

		No. of Pigs Slaughtered.	No. of Pigs Condemned.	No. of Pigs showing T.B. Infection.
Johannesburg	1947	95,697	4,707	440
	1948	112,897	5,713	403
	1949	218,752	10,467	710
Pretoria	1947	20,473	1,285	—(2.056%)
	1948	27,997	1,679	—(2.168%)
	1949	28,987	1,645	517

The type of organism responsible for the lesions has been determined in a number of cases over a period of many years. Usually the bovine type was found to be present, but on one occasion Dr. Robinson encountered a human type infection. (Personal communication.)

## CASE REPORT

A farmer in the Transvaal visited Onderstepoort in connection with condemnations for tuberculosis in a number of consecutive consignments of pigs slaughtered at abattoirs. In collaboration with the State Veterinarian of the area (Dr. G. J. de Wet) it was arranged to test all the pigs on the farm and to try to determine the source and nature of the infection.

On the 28th August, 1948, the intradermal tuberculin test was carried out on 350 pigs using mammalian tuberculin in the skin of the right ear and avian in the left, applied dorsally near the base of the ear. The result of this test was read on the 31st August. The three reactors were slaughtered and lesions demonstrated in all three. Five young pigs (about two months) showed very indefinite reactions. Since they were in poor condition they were also slaughtered, but no lesions were found.

A second test was carried out on the 17th February, 1949, when one reactor and four unthrifty negatives were slaughtered. The reactor showed typical lesions in the right mandibular lymph gland and suspicious foci in the right bronchial and right prescapular gland. The other pigs showed no lesions.

To date (18 months since the last test) no further cases have been found in pigs sent for slaughter, although some five hundred animals have gone to the abattoirs.

The material collected from the reactors was examined histologically and biologically. Tuberculosis was established by both methods. Acid-fast organisms were also found in smears from antiformin-treated portions of the glands. Fowls and guinea-pigs were used in the biological examination and lesions developed in the guinea-pigs only. This indicated that the organism concerned was of the mammalian type. Further typing tests in rabbits and guinea-pigs with material from Dorset's egg cultures of the glands of the first guinea-pigs, showed lesions only in the guinea-pigs. This was confirmed by a further test with material from a subculture on Dorset's egg medium. The result justifies a diagnosis of human-type infection.

Final confirmation was obtained by inoculating two calves 3815 and 3931 subcutaneously with massive doses of a fresh culture.

Three months after infection these calves were subjected to a comparative tuberculin test — using heat concentrated (H.C.) Onderstepoort tuberculin on the one side of the neck and P.P.D. (single) tuberculin, Onderstepoort, on the other side of the neck. These intradermal tests were carried out on the 28.1.50 to the 31.1.50. On the 1.2.50 the short thermal, subcutaneous tuberculin test, was applied in accordance with the directives of Gregory (1949). The results of these tests are presented in table No. 1.

TABLE 1.

*Showing Results of Comparative Test with H.C. and P.P.D. Onderstepoort Tuberculins as well as those of the Short Thermal Subcutaneous test in Calves 3815 and 3931.*

<i>Tuberculin test.</i>						
	28.1.50		30.1.50		31.1.50	
	H.C.	P.P.D.	H.C.	P.P.D.	H.C.	P.P.D.
3815	8.0 mm.	8.6 mm.	23.5 mm.	26.0 mm.	37.5 mm.	27.0 mm.
3931	6.0 mm.	7.2 mm.	12.0 mm.	20.0 mm.	25.0 mm.	21.0 mm.

<i>Short Thermal Subcutaneous test: 1.2.1950.</i>						
	0 hours	2 hours	4 hours	6 hours	7 hours	8 hours
3815	101.6°F	102.4°F	102.4°F	103.6°F	104.6°F	105.4°F
3931	102.2°F	101.9°F	102.1°F	103.2°F	104.1°F	104.8°F

Four months after infection the calves were slaughtered and at post mortem only very slight lesions in the glands near the site of infection were detected. Smears of the lesions showed acid-fast organisms, and guinea-pigs injected with the material developed generalised tuberculosis. The presence of only localised lesions in the calves speaks against a bovine-type infection.

The source of the infection could not be established. It was impossible to arrange for a medical examination of the personnel on the farm which consists partly of migrant labour. Since no further cases were found subsequent to the last test it is assumed that the source of infection had ceased to operate.

One difficulty encountered in applying the intradermal test in pigs was the reddening of the skin of the ears in white pigs, due to sunburn. It almost seemed as if more wounds were present on the ears of these pigs with unpigmented skins, and it is suggested that in a country like the Union with its strong sun in summer, the test in pigs should, where possible, be carried out during the winter.

#### SUMMARY

1. An outbreak of subclinical tuberculosis in domestic pigs is described.
2. The disease was controlled by slaughter of reactors found when the herd was tuberculin tested on two occasions only.

3. The cause of the outbreak was proved by biological tests on small and large animals to be *Mycobacterium tuberculosis* var. *hominis*.

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# EPISTAXIS IN RACEHORSES

## INCIDENCE IN SOUTH AFRICA

G. PFAFF.

In racing parlance a horse that bleeds from the nose during a gallop is said to have broken a bloodvessel. In a very exhaustive article in the *British Bloodstock Breeders' Review* (No. 4 of Vol. 2, pp. 266 to 281), J. B. Robertson showed that the tendency to break bloodvessels is inherited as a recessive character; a horse, however, is not likely to break a bloodvessel unless he carries the factor in a double dose. It is said that all bleeders trace back to Herod, which was foaled in 1758; certainly the names of Hermit (foaled in 1864), and of his grandson Gallinule (foaled in 1884), appear in the pedigrees of many bleeders in South Africa.

A vessel ruptures because of the strain that sudden engorgement imposes upon its abnormally thin or weakened walls. It follows, therefore, that the vessels most liable to rupture are those superficially situated and not supported by over-lying tissues, such as those in the walls of the nasal chambers and in the pharynx. Other vessels prone to rupture are those in the lungs, and (according to Robertson) in the bowels, in or about the capsules of the joints, in the urethra, and under the skin.

A liberal ration of hay, or bedding a horse on straw, was at one time blamed for bleeding from the nose. The indiscriminate and inexpert use of the stomach tube may leave tiny lesions resulting in hæmorrhage during a race. Some think that a horse may bleed if severely galloped when not quite fit, but not when in hard racing condition. One wonders, however, whether drugs may help to provoke epistaxis.

The bleeding may vary from a slight trickle from one nostril to profuse hæmorrhage from both nostrils. It usually stops spontaneously and rapidly when the horse is brought to a standstill, though in exceptional cases death may occur within a matter of minutes.

The course of the disease is unpredictable. A classical case is Hermit, which bled profusely in a trial gallop, yet ten days later won the Derby. In the same Derby ran The Rake, which bled six days earlier, but not in the race. Then there was Gregalach, which won the 1929 Grand National as a 7-year-old. Gregalach had five crosses of Hermit; the names of Toxophilite and Gallinule (notorious bleeders), and of Thormanby (a sire of bleeders), also appear in Gregalach's pedigree. In the 1933 Grand National, Gregalach broke a bloodvessel, or, as J. B. Robertson put it, "the curse of Hermit won through".

In my own experience was a two-year-old colt that bled slightly in a gallop, and 18 days later bled to death in a race, having worked

normally in the meantime. Strangely enough, the grandson of this horse produced a colt which also bled to death in a race on the same course at almost precisely the same spot. Fortunately, both horses were well clear of the field when they fell, thus giving the others a chance to go round them. Many horses bleed only once and not again. The exceptional few are chronic bleeders, with intervals of perhaps months between hæmorrhages. The very rare ones bleed to death on the first occasion.

Very few bleeders are even noticed by the racing public; those that bleed profusely spatter themselves and their riders with blood. The dangerous horse is the one that bleeds so much that he stops suddenly, thus interfering with other horses in the race; if he falls he may bring them down.

Deaths from the disease are not common. To my knowledge, in the past two and a half years, four deaths occurred during or soon after a race, but not within the period covered by this review, and are, therefore, not included in the figures below. Two followed profuse hæmorrhage from the nostrils (one horse died within a few minutes and the other about an hour later); the other two died two or three minutes after the race; they did not bleed externally, but post-mortem examination revealed extensive hæmorrhage from the lungs. A trainer told me that in the past two years two of his horses died during work; from the veterinary surgeon who did the post-mortem examination I learnt that death was due to cerebral hæmorrhage. Both horses were by the same sire.

The object of this note is not to discuss the cause or treatment of the disease, but merely to record the incidence of the condition in South Africa, as reflected in an analysis of racing over a period of 24 months in five centres, namely, Johannesburg, Pietermaritzburg, Durban, East London and Cape Town. Johannesburg is 300 miles from the coast and 5,400 feet above sea level; Pietermaritzburg is 50 miles from the coast and 2,000 feet above sea level. The other centres are on the coast.

I should add that I have no records of horses that bled (or died) during work, and that my figures do not include the few that may have bled during a race but were not reported.

The number of bleeders is small, and for that reason conclusions are perhaps not justified. The figures do, however, suggest that:

1. Approximately one in every 82 horses bleeds during a race, and bleeding occurs in one out of every 63 races.
2. Geldings are more liable to bleed than horses or mares.
3. The tendency to bleeding increases with age.
4. More horses bleed in sprints and races up to nine furlongs than in longer races.
5. The worst months for bleeding are August, September and October; the hottest time of the year produces fewest bleeders.
6. Horses are far more apt to break bloodvessels at sea level than in Johannesburg, which is 5,400 feet above sea level.

TABLE 1.  
*The Incidence of Bleeding.*

Number of races .....	3883
Number of horses .....	4015
Number of horses that bled in one race .....	37
Number of horses that bled in two races .....	11
Number of horses that bled in three races .....	1
Percentage of horses that bled in races .....	1.2
Total number of horses that bled in races .....	49
Total number of races in which bleeding occurred .....	62
Percentage of races in which bleeding occurred .....	1.6

There were 4015 horses, but each horse ran in several races; in the two years covered by this review the average number of races per horse was 19.5.

TABLE 2.  
*Sex Incidence of Bleeders.*

Sex of Runners.	Approximate Average Age of Runners (years).	Number of Runners.	Number of Bleeders.	Percentage Bleeders.
Horses (including colts)	4.5	1197	14	1.17
Geldings .....	5.5	1428	32	2.23
Mares (including fillies)	4.0	1390	16	1.17

Mares are retired from racing earlier than horses, and geldings are raced longer than horses. The average ages given above have not been accurately determined, but are my estimation, based on a sample analysis.

TABLE 3.  
*Age Incidence of Bleeders.*

Age (Years).	Number of Runners.	Number of Bleeders.	Percentage Bleeders.
2	1561	4	0.26
3	1878	11	0.58
4	1453	13	0.89
5	1054	13	1.23
6	671	7	1.04
7 and over	959	14	1.45

Two-year-olds run less frequently and over shorter distances than older horses. The horses that ran both years — and most of them did — are counted twice; for instance, as a three-year-old and again as a four-year-old.



TABLE 4(a).  
*Distance Incidence of Bleeders.*

Distance (furlongs).	Number of Races.	Approximate Aver- age Number of Runners per Race.	Number of Bleeders.
4	49	21	1
5	1041	15	17
6	910	14	14
7	432	14	6
8	540	14	9
9	252	10	9
10	378	11	3
11	28	16	1
12	187	10	2
Over 12	66	9	0

TABLE 4(b).  
*Distance Incidence of Bleeders.*

Distance.	Approximate Number of Runners.	Number of Bleeders.	Percentage Bleeders.
Up to 7 furlongs	2280	38	1.7
8 to 10 furlongs	1040	21	2.0
Over 10 furlongs	695	3	0.43

Horses are not restricted to one distance, but are usually kept within certain limits. A horse that habitually runs in races up to 7 furlongs may occasionally run in an 8-furlong race, and horses that concentrate on races of over 10 furlongs will quite frequently run in 8 to 10-furlong races. The figures in column two above are, therefore, very elastic.

TABLE 5.  
*Seasonal Incidence of Bleeders.*

Month.	Number of Races.	Number of Bleeders.	Percentage Bleeders.
January .....	363	3	0.82
February .....	382	2	0.71
March .....	302	5	1.65
April .....	333	7	2.10
May .....	382	6	1.57
June .....	289	2	0.72
July .....	326	4	1.22
August .....	334	9	2.69
September .....	285	7	2.45
October .....	369	10	2.71
November .....	304	5	1.65
December .....	314	2	0.63

The average number of runners per race varies very little throughout the year. The total number of horses, and the total number of runners, each month would be, therefore, directly proportional to the number of races run during the month.

TABLE 6.  
*Geographical Incidence.*

Centre.	Approximate Number of Races.	Number of Runners.	Number of Bleeders.	Percentage Bleeders.
Johannesburg .....	1727	1925	13	0.68
Pietermaritzburg .....	224	955	5	3.13
Durban .....	808		25	
East London .....	378	185	3	1.6
Cape Town .....	746	950	16	1.7

There is not very much movement of horses between the centres mentioned above—except between Durban and Pietermaritzburg. Visiting horses are in the top class, and are very unlikely to be bleeders. The number of runners given above is the number permanently located in that centre.

# FROM A 3RD YEAR STUDENT'S NOTEBOOK

## THE EFFECTS OF SOME SUBCUTANEOUSLY ADMINISTERED PURGATIVES ON HORSES.

The following demonstration was recently given to the Pharmacology class of 3rd year students. Four light horses, all in rather poor condition, were injected with arecoline, acaprin, prostigmine and carbachol respectively. The results are summarised in the table.

	Horse No. 1.	Horse No. 2	Horse No. 3	Horse No. 4	
Injection ...	Arecoline hydrobrom. 50 mgm.	Acaprin 3 cc.	Prostigmine (Roche) 8 cc. = 22.5 mgm.	Carbachol 2 cc. = 2 mgm.	Carbachol 2 cc. repeated after 30 minutes.
Salivation ...	Profuse. Started 3 minutes after injection and persisted for 30 minutes.	Very slight.	Very slight.	Profuse after 5 minutes.	Profuse.
Pulse Rate	Increased from 28 to 56 within 3 minutes.	Increased from 36 to 72 in 35 minutes.	No change.	Raised from 38 to 52 in 20 minutes.	Remained over 50.
Purgation ...	Watery faeces passed at 20 and 30 minutes.	Soft faeces starting at 20 minutes persisting 1 hour.	Started at 10 minutes, persisted for 1 hour. Slight colic.	None.	Soft faeces passed after 10 minutes.

## CONCLUSIONS

As will be seen, arecoline, acaprin and carbachol all caused marked acceleration of the pulse in spite of the fact that parasympathetic stimulation causes inhibition of the heart. This is due to the fact that the drugs also cause vasodilation and the increased heart rate is brought about by pressor reflexes in an endeavour to maintain the blood pressure. Acaprin caused the greatest disturbance in the circulatory system and its danger in animals anæmic from babesiosis is evident. Prostigmine, on the other hand, had no effect on the heart rate.

Salivation was profuse and even distressing with both arecoline and carbachol.

Purgation was obtained with all four drugs, but the most prompt and satisfactory action occurred in the horse which received prostigmine. This animal showed some signs of colic and had to be walked in order to prevent it rolling, but it must be remembered that it was the only one to receive a full dose. The complete absence of action on the heart is, therefore, all the more remarkable.

As a further demonstration, horse No. 4 was injected with 50 mgm. of atropine 25 minutes after it had received the second dose of carbachol. The pulse rate dropped from 50 to 34 and salivation ceased within three minutes.

## LETTERS TO THE EDITOR

### THE TREATMENT OF BOTULISM (LAMSIKTE).

In a recent outbreak of botulism in mules, I had the opportunity of trying the effects of carbachol and neostigmine on quite a number of cases. The results were so promising that I would like to suggest to my colleagues to include these two parasympathetic drugs in the course of their treatment of lamsiekte. I would like to stress the importance of small doses of these two remedies at short intervals rather than large doses at long intervals. Details of treatment on this line could be worked out by our colleagues who have to deal with outbreaks of lamsiekte. I suggested the use of neostigmine in conjunction with carbachol, as the former is a potent stimulant of voluntary muscle. Also neostigmine should be injected in small doses at short intervals. Experiments should also be conducted with picrotoxin which is a pronounced stimulant to all parts of the central nervous system. A preliminary intravenous dose of 10.0 cc. of a 0.3% solution of picrotoxin could be given to a full-grown beast or horse, and subsequent doses could be calculated on the reaction induced by the preliminary dose.

A point of importance in the treatment of botulism is the daily emptying of the large bowel by means of an oily enema.

I have treated quite a fair number of cases of botulism in mules, horses and cattle with strychnine, and in one outbreak in dairy cows I used picrotoxin with no apparent beneficial effects; it is for this reason that I would like to bring my last experience to the notice of our colleagues.

DOUW G. STEYN.

### AN ACUTE NUTRITIONAL DEFICIENCY OF EQUINES.

The object of this note is to place on record the clinical description of a malady affecting horses in the Fraserburg district of the Cape Province. Information received from farmers indicates that the disease apparently first made its appearance late in 1943. During the summer of 1944 a few more horses died. The disease was first investigated during January, 1945.

Fraserburg, essentially a sheep-raising area, has no acknowledged equine breeders. The horses affected are riding and cart horses. Geldings are chiefly affected, although a case was observed in a stallion and in a mare. The disease is only seen in horses put to work, veld horses apparently not being affected. Cases have been observed in the hot, dry summer months only. The mortality is almost 100%, although a single natural recovery was observed.

#### *Aetiology:*

The cause of the disease is as yet unknown. It is not infectious, and tetanus, botulism, Kimberley horse disease and dourine can be excluded. Because of the absence of the characteristic dark colour of the urine and the changes in the musculature and the kidneys, a diagnosis of azoturia

cannot be made, nor is there any evidence that plant poisoning plays a rôle. *Penzia incana*, one of the causes of Kimberley horse disease, is present in such negligible quantities that it is of no significance.

From 1940 to 1946 the district of Fraserburg experienced the severest drought in human memory. Except for wheaten chaff and lucerne irregularly obtained from small irrigation acres maintained by borehole water, nutritive fodder for working horses was at a premium. Because of the demands of war and also because of concurrent droughts in the rest of the country, maize and wheat were unobtainable. These animals, therefore, had to maintain their own body requirements and perform work on a ration, which, although bulky, contained comparatively little of nutritive value. It would appear that this lack of a balanced ration, fed to working animals over a long period, predisposed them to the train of symptoms set up.

#### *Course and Symptoms:*

The course is variable, ranging from a few days to a few weeks.

All affected animals show the first symptoms when put to work. In all such cases work has been jackal hunting or inspection of jackal-proof fences. The first symptom is twitching of the tail. The animal shows distress and refuses to move, but when forced to do so, muscular inco-ordination becomes apparent. The animal is both distressed and excited. The muscular weakness increases and the animal goes down. Frenzied, often unsuccessful, attempts are made to rise, the animal sometimes assuming a "dog-sitting" position. While attempting to rise, the animal sweats freely and groans as if in great pain and may have to be helped to regain its feet. In the cases investigated it has always been possible to lead the animals back to the homestead after such an initial attack, the distance, fortunately, having never been more than three or four miles. On the homeward journey a second attack may occur. This initial flare-up of the symptoms is often the only attack suffered by the animal, but a few cases showed a second attack. Subsequently, the only visible symptom is an extremely rapidly progressing muscular inco-ordination, which may be so severe that death supervenes within a few days. Should the case linger, emaciation and weakness follow. The animal invariably goes down and is unable to rise, presenting a pitiable picture. Decubitis sores are a natural sequel. Affected animals show no elevation of temperature, and, except that the conjunctiva is injected and slightly icteric, the usual clinical examination presents no unusual features other than those already described. Affected animals are able to eat and drink. There is no obstruction to the voidance of urine or the passage of fæces. Unless humanely destroyed, such animals may linger for two to three weeks before death.

#### *Post-mortem findings:*

These are entirely negative. Histo-pathological examination of specimens of the brain, spinal cord, liver, spleen, kidney and musculature by Research Officers at Onderstepoort revealed nothing of importance.

M. J. M. MEESER.

## BOOK REVIEWS

*DISEASES OF THE PIG*, by D. J. Anthony: 3rd edition; price 17s. 6d. Baillière, Tindall & Cox, London.

The first quarter of this book describes the breeds, feeding and management of pigs. The rest deals with their diseases. The information is generalised and detail is lacking. A number of subjects are described briefly. More space could be given to infectious diseases of pigs and less to conditions common to other animals. The author has attempted to cover too large a field in a small book. — D.G.S.

*ANIMAL DISEASES IN SOUTH AFRICA*, by Professor M. W. Henning: C.N.A., Johannesburg, 2nd Edition; price £3 5s. 0d.

This book deals with Bacterial, Protozoal and Virus and Rickettsial diseases in separate sections. Each disease is conveniently tabulated under sub-headings so that any particular aspect of the disease can be found easily. The author has taken pains to include as much information as possible and has given a list of references at the end of each chapter. There are a number of clear illustrations particularly of protozoal parasites in blood films.

The second edition is one volume. The section on poisonous plants which appeared in the first edition has been omitted. This book can be unreservedly recommended to all veterinary surgeons, particularly those in Africa. — D.G.S.

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### NOTICE.

A number of bound reprints of a series of articles by C. P. Bresler, M.A., LL.B., entitled "The Veterinarian and the Law" (about 25 pages), are still available to new members at 2s. 6d. per copy, post free.

## SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION.

*Minutes of Council Meeting held at 2.15 p.m. on April 20, 1950,  
in Prudential House, Pretoria.*

*Present:* J. H. Mason (President), G. D. Sutton (Hon. Treas.), S. W. J. van Rensburg, R. Clark (Editor), P. J. Snyman, G. Pfaff, J. G. Boswell, R. A. Alexander, W. D. Malherbe, A. D. Thomas, H. P. Steyn and M. de Lange (Hon. Sec.).

*Apologies:* P. J. du Toit, A. M. Diesel.

In his opening remarks the President referred to the passing of the late Director of Veterinary Services, Dr. J. I. Quin. The meeting stood for a few moments as a token of respect.

1. Minutes of meeting on February 2, 1950. Adopted.

2. Arising from these minutes:—

(a) *P.D.S.A. Cape Town:* Secretary reported that vacancy for part-time veterinarian had been circulated to all members and that two applications had been received. Owing to subsequent complications, no finality had yet been reached.

(b) *P.D.S.A., Johannesburg:* Dr Pfaff reported briefly on the latest developments regarding proposed veterinary assistance to this branch of the P.D.S.A. Negotiations with the Witwatersrand branch of the Association were proceeding.

(c) *Complaint of Member:* Secretary reported that he had been informed by Dr. Diesel that the dispute between the parties concerned had been settled.

(d) *Dog Licences in Transvaal:* Secretary was still corresponding with the Provincial Secretary, and would report to next Council meeting.

(e) *Financial Matters:*

i. *Assistance to Member:* Council approved assistance being given by Finance Committee to elderly member in straitened circumstances and supported suggestion that this member be granted life membership.

ii. *Assistance to Dependents:* Letter of appreciation from widow received and noted.

iii. *Student Loans:* Council approved action taken by Finance Committee in respect of non-repayment of loans.

iv. *Dr. Maud Bales Bursary:* Two applications were selected (from nine received) by Finance Committee. Messrs. I. R. Banks and C. H. van Niekerk were approved for recommendation to the Board of Trustees.

v. *Benevolent Fund:* In view of recently approved scale of fees to be charged by State Veterinarians for clinical work, the proposed scheme of collections on behalf of the Benevolent Fund to be left in abeyance, except in the case of donations received at Onderstepoort.

vi. *Book Fund Prize:* On recommendation of Faculty, the annual prize of £10 had been divided equally between Miss J. H. Verdurmen and Mr. P. G. Howell. Approved.



vii. *Life Members*: The following members had now qualified for life membership: F. A. Verney, W. P. Hamlyn, P. J. J. Fourie, W. Hay, D. D. Morton, J. Walker and G. T. Henderson. President to send letters of congratulation.

- (f) *Co-operative Employment of Veterinarians*: Letters from Registrar of Veterinarians read and discussed. Complainant to be informed that Council did not object to co-operative employment of veterinarians, or to Co-operative Societies advising their members as to veterinarians employed by them. In the letter concerned no specific charge was made, but if and when cases of unprofessional conduct were to occur in the above connection, the Veterinary Board was competent to deal with these.
- (g) *Amendments to the Veterinary Act*: Dr. Pfaff reported on the poor response to circular sent to members of Council, Veterinary Board, Faculty, Provincial Sub-directors and Branch Secretaries. President appealed to members of Council to speed up their replies. Secretary to write to Faculty.
- (h) *Appointment of Lecturer by Pretoria University*: Letter by President to secretary of the University Council read and approved. Letter received from Faculty read. Matter to be held over, pending reply of University Council.

### 3. *Correspondence*:

- (a) *International Congress Fund*: Contributions from Union required at a rate of one shilling per annum per veterinarian. Acting D.V.S. to approach Secretary for Agriculture with a view to obtaining a contribution from Treasury on a 50-50 basis.
- (b) *City of Bloemfontein—Consulting Veterinarian*: Letter from Town Clerk read. Secretary to make further inquiries.
- (c) *Letter from Member re sale of carbamylcholine to lay public* read. After discussion, decided to inform member that Council was fully aware of the position and that efforts to have the sale of this and other drugs restricted were continuing.
- (d) *Letter from Secretary, Australian Veterinary Association* read. Decided to request Dr. P. R. Viljoen, Union High Commissioner, to represent S.A.V.M.A. at Annual General Meeting.
- (e) *Letter Dr. L. W. van den Heever re salary scale Germiston Municipal Veterinarian* read. As post was soon being advertised at a higher scale, this matter to be left in abeyance.
- (f) *Letters, Secretary for Agriculture, re Poultry Diseases and Infectious Sterility* noted and receipt acknowledged.
- (g) *Letter Dr. Fourie re post-graduate course in Municipal Hygiene* read. Reply to express Council's approval.
- (h) *Letter Registrar R.C.V.S.* noted and receipt acknowledged.
- (i) *Letter Dr. Pullinger re National Health Council*. Dr. Snyman's report to be held over to next meeting.
- (j) *Lengthening of Veterinary Course*: Replies received from members were handed to Dr. Pfaff for report to next Council meeting.

### 4. *General*:

- (a) *Representative on Council of Natal Anti-T.B. Association*: Dr. Zwarenstein to continue as S.A.V.M.A. representative.

- (b) Dr. Alexander outlined departmental attitude on B.W.D. and C.A. field tests and on the proposed production of distemper vaccine. Also reported on possibility of a grant being received from Central Meat Co-op. Society to assist in the reorganisation of veterinary education, research and field services.
- (c) The Editor appealed to Council for articles for the *Journal*. Decided that Dr. Clark circularise an appeal to all branch secretaries. Dr. G. P. Bishop proposed to fill the vacancy on the Editorial Committee resulting from the death of Dr. Quin. Approved.
- (d) Date of General Meeting: To be held at Onderstepoort on September 5, 6 and 7, 1950. Proposal to have lapel cards printed for the meeting approved.

J. H. MASON,  
*President.*

M. DE LANGE,  
*Honorary Secretary.*

# THE PLACE OF THE VETERINARIAN IN MEAT PRODUCTION.

(Condensed from a Plenary Lecture given  
by Dr. C. Hopkirk, of New Zealand, at the International Veterinary  
Congress, London, 1949.)

Under the place of the veterinarian in meat production I include the breeding and feeding of animals, the prevention of disease, as well as the control of slaughtering, the inspection of meat and the control of its transportation. It may be disputed that the veterinarian has the right to claim an interest in all these phases, but he alone is competent to speak on all aspects and to work with other specialists on any of these lines.

## *Breeding.*

Each country, each people, each climate and each age has bred animals which have suited their temporary needs. In backward areas and before the present mechanised age, many animals have had to be of dual and treble purpose. But with the increase in machinery it seems likely that many dual-purpose breeds of animals may disappear, and give place to the single-purpose animal. As animal feeding becomes perfected the aim must be a healthy animal of optimum production, whether of meat, milk or wool.

Experimental work in breeding has led to many beneficial changes which may have a great influence on food production by the establishment of new breeds to suit the climate, as, for example, the crossing of zebu cattle with European types and the utilisation of mutants such as hornless types within a horned breed. Breed societies which insist on appearance rather than on production are doing a grave disservice. Horns in many cattle breeds should definitely be discouraged, for there is no greater harm done in the meat trade than the damage by horns in trucking and yarding of meat animals, while there is every reason to believe that hornlessness leads to docility and greater fattening propensities. Dehorning is work for the butcher rather than the veterinarian, who should be instilling prevention of horn growth into the minds of cattle owners.

With the introduction of artificial insemination, a great instrument for perfection or deterioration of breeding has been placed in our hands. It is a science which requires the greatest care in administration. Not only has it enabled us to improve breeding, but will go far to counteract the majority of venereal diseases which can be so devastating to a breeding programme. Its main use at present in European countries, unfortunately, is to prevent disease, and this must be its first aim, but its greatest advantage lies in using superlative semen carrying the factors we require for increasing production. It would appear, then,

that the duty of the veterinarian in breeding is to take charge of programmes of artificial insemination; to assist in experimental crossing; to see that no harmful factors appear; to recognise important new useful factors and to have that broad conception of where breeding on any experimental line is leading, so that immediate advantage can be taken of it.

### *Nutrition.*

The object of scientific nutrition should be uninterrupted and optimum growth. If the environment cannot produce that state, we must create it. Most of our meat supplies come from ruminant animals, and their first essential in the way of diet is a food which has *bulk* as well as a high food value. That means mixed pasture on highly fertile soil is the best food for production. Remember the Biblical text, "all flesh is grass". One must allow that, in over-populated areas where sufficient pasture cannot be produced, other feeds of high nutritional value such as grain and nuts may be required, but these are imported from other areas. Our agronomists have given us strains of grasses and legumes of very high protein value, but in many countries the farmer has not been encouraged or has not taken advantage of this new phase of agriculture. Each country must build up suitable strains of grasses for its own varied climate and soil conditions. Our contention is that only high quality pasture for stock should be developed, but this also demands increasing the fertility of the soil, which in turn demands increasing supplies of phosphate, nitrogen and potash.

The conservation of the grass crop for winter or drought feeding is also a need. Much depends on climate as to whether hay, ensilage, or kiln-dried grass is preferable. Too many of our nutritional disturbances in animals result from the lean winter and sudden change into conditions of flush feed. Here, the veterinarian with his increasing knowledge must advise and understand that production of milk and meat depends on the even flow of food to the animal during pregnancy and lactation.

From the nutritional angle, mineral deficiencies have probably been the main factors in retarding the production of stock, on otherwise suitable areas. They were first brought to our notice in South Africa when phosphate was found to be so necessary, and where its lack was found to interfere so seriously in breeding and in growth and also in precipitating many secondary disease conditions. Their further rôle was studied in the United States of America, Australia and New Zealand, where deficiencies in cobalt and copper were shown to impair animal production and breeding and even life, over huge areas of land. The benefits of cobalt and copper on otherwise sound land have been amazing. In small New Zealand, thousands of acres of virgin country which could not carry stock, have been turned into fertile land giving increasing yields of flesh and milk. In Australia hundreds of square miles of territory have been improved. This is a veterinary triumph. The story is not yet ended, and further research will, no doubt, reveal other trace elements, or combinations of elements, of importance,

### *Disease Control.*

The veterinarian must take full responsibility for the control of disease in all types of domestic animals. It is largely because of diseases which have been uncontrollable that the world must needs be short of meat. Were Africa free from rinderpest, pleuro-pneumonia, tick and fly-borne diseases, there would be an enormous reservoir of native cattle which could be used.\*

The results of the control of bovine contagious abortion by the vaccination of calves with Strain 19 has been extraordinarily successful in many countries. We hope to see extensive progress in the prevention of abortion in beef herds, as has occurred in dairy breeds, in the near future.

Where possible, tuberculosis can be and should be controlled by test and slaughter or where this is impossible, by vaccination.† Any country not seriously tackling this problem through its veterinary services is committing an offence to the human race.

One could enumerate many diseases against which effective control methods are already known — but one can also make a plea for intensified research on the lines of prevention of disease. Not only does one require the high-class veterinary research team, but also that buffer or interpreter between research and the farmer, the general practitioner, or field officer. And, finally, one needs enlightenment of the stock owner, which I contend is the responsibility of the Government. Extension services could go far to create a demand for disease-free animals. The more backward the country, the more necessary is it that a strong veterinary force exist, for it is in such countries that we often find the largest potential population of stock which could be utilised for meat.

### *Meat Processing and Distribution.*

There are many angles which can be considered in meat processing:—

- (a) Preparation for market;
- (b) Carriage to the abattoir;
- (c) Up-to-date slaughter houses and cleanliness of slaughter;
- (d) Inspection for disease;
- (e) Cooling, refrigeration and shipping;
- (f) Preparation in the butchers' shops for sale to the public.

We might consider these points in order.

(a) In many countries where beef is raised, it is the practice to run large herds on ranches for meat production. Special crops are grown, frequently alfalfa, which are used to give the already good-conditioned and well-grown beef that bloom and final fattening which the public demands. The quicker this process can be carried out, the

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\* (Soil erosion might well become as great a problem as disease. — Editor.)

† (To our knowledge, there is as yet no proved vaccine against tuberculosis. — Editor.)

better beef one gets; Argentine and Texan beef are produced by this method. Those countries with rich grasslands employ cattle for control of the pasture, both for dairy purposes and, more important still, for sheep farming. In New Zealand, the flush of growth in spring and autumn would make first-class sheep farming for fat lamb production almost impossible without proper control of the grass. Black Polled Angus or Galloway crosses are excellent hill scavengers and increase the mutton and lamb production by large amounts. It is the custom to take these cattle, as they reach the age of four years, to good pastures on fertile flats, where they are topped off on the best of grass and at once sent to the meat works for overseas trade. In older countries, where animals are bred and fed on grain for meat production only, topping off is not so necessary. The beef so produced is perhaps the best obtainable, but the cost of production is far too high.

There is also that big reservoir of native African cattle which have to rely only on native and poorly nutritious grasses which are rarely suitable for production of meat for European markets except at the end of the wet season. For meat production these countries will require not only control of disease, but improvement in pasture as well.

Much the same need of topping off is found in mutton and pig meat production. Lambs should be killed off the mother. Canterbury lamb, of world-wide fame, is produced on fertile pastures and killed before weaning. Where good pork is produced, the young pig is run on dairy pasture and fed skim milk and perhaps some grain, being shut up for two or three weeks before slaughter to give that required extra bloom. Baconers, however, to be first class, are quite differently fed. Countries with waste milk products do not as a rule produce the good bacon which comes from grain feeding. Poultry also requires extensive grain feeding, and speed of growth is essential.

(b) Animals ready for market, can so easily be ruined by inefficient droving or transport. Great spoilage of meat occurs in beef animals of horned breeds. There is always some loss in weight, and the sugar content of the muscles is greatly reduced. In heavy beef and pork animals considerable bacterial spoilage of the meat may occur, unless the animals are again extensively fed with carbohydrate food before slaughtering.

Bone taint is now considered to be a pre-killing nutritional fault which can be controlled after slaughter only by most efficient cleanliness and quick cooling. In those countries where animals are killed for meat export, an effort is made to have big slaughter houses placed at suitable intervals in heavily stocked country. The livestock trains are fast and as comfortable as possible. Overcrowding is often prevented by Government regulations, but even so there is a percentage of deaths which remains unavoidable. All that can be recommended with regard to travel is that the animal be got to the killing-centre as quickly as possible with as little loss of condition as the method used permits.

(c) Abattoirs should be up-to-date and built on a gravity system to lessen labour and the handling of offal. Provision should be made for everything to be utilised which is of use after slaughter. This

cannot be carried out in small abattoirs which kill a beast or a few sheep or pigs for local consumption. The unsanitary conditions and the loss involved in slaughtering on this small scale are appalling. The solution is that every country set up municipal abattoirs for the centralisation of slaughter with efficient transport of animals and meat. The result will be greater cleanliness, better inspection (for in the small abattoirs inspection is honoured in the breach), economic utilisation of offal, and a happier slaughtering-house staff.

(d) Inspection of meat scarcely needs stressing. It is usually the duty of a layman under veterinary supervision. Such laymen become highly specialised and their work is of great importance so that their training should be in the hands of a highly skilled veterinary panel. I do not believe that a veterinary surgeon should carry out actual inspection duties. He should, in his supervision of all phases of slaughter and processing, rely on his lay assistants but be prepared to take the responsibility for their decisions and make important decisions himself. His is the sole responsibility. With the shortage of meat the veterinary inspector can show a leniency in condemnation which would not be countenanced in time of plenty. He must do this with due regard to human health, saving a quarter here and another there, when the easiest course would always be to condemn the whole carcass. His is the responsibility for obtaining every pound of meat the carcass will give.

It is the custom of many countries to have laboratories attached to the larger slaughter houses, and this is to be strongly commended, for biochemical and bacteriological tests are important in those countries where a dead animal is salvaged. Where stock is plentiful, a dead animal is used for manure, but in closely populated areas the meat is too valuable and, if suitable, is used as public food. Again the discretion of the laboratory veterinary officer must be relied upon to protect public health.

Ante-mortem inspection has often been found particularly useful, especially in Foot and Mouth Disease outbreaks and Anthrax, but too frequently it is not carried out thoroughly. It should be a very serious part of the duties of the veterinary inspector.

(e) *Cooling, Refrigeration and Shipping.* The carriage of meat in refrigeration ships is not easy. It requires immense experience in precooling of the carcass, freezing in the slaughter houses, quick transfer to ships and careful regulation of temperatures over weeks of travel. The stowing of carcasses in a ship so that air may circulate between them is not a matter of guess work.

In some countries much of the post-slaughter damage to meat arises during the carriage from the slaughter house to the ship. Where the factory is at the water's edge no time is lost in packing meat into the hold by conveyor belt, but with factories many miles from the port the serious work of loading meat into trucks and rehandling for transfer to the ship is important. Frequently, the railway trucks used, although insulated, are not pre-cooled, and insufficient use is made of

CO<sub>2</sub> snow or other cooling methods for maintaining low temperatures in trucks while travelling in summer weather.

It may be claimed that refrigeration does not come under veterinary control. This is largely true, but if pre-cooling and refrigeration have not been perfect, then it is to the veterinarian that food authorities should turn to adjudicate on spoilage and salvage.

Finally, defrosting should be controlled. The appearance of the meat in the butcher's shop should be the pride of the veterinarian.

In conclusion, may I drive home several points upon which we should have action?

Firstly, breeding should be a major life interest for veterinarians. Too many of us think in terms of the uneconomic dog and cat instead of in terms of the betterment of economic breeds which mean our very life.

Secondly, too much land is wasted, often with uneconomical crops. This land should be used to create permanent pastures for meat production; pastures composed of known strains of high producing grasses suitable for the climate and the type of animal to be fed. That balance of crop and permanent animal pasturage which is the essence of good farming must be re-established. Veterinarians could stimulate thought in this direction in their sincere efforts to have good animals produced on the farm.

Thirdly, the enormous work of the control of epizootic disease is before you. Much has been accomplished, much more is required. Success can only be achieved through research and the practical application of existing knowledge.



# VETERINARY SERVICES IN AUSTRALIA AND NEW ZEALAND : A FEW IMPRESSIONS

GILLES DE KOCK,  
Cape Town.

## AUSTRALIA

The Commonwealth of Australia is a Federation of six States:—

Queensland .....	650,000	square miles
Victoria .....	87,884	” ”
New South Wales ..	309,433	” ”
South Australia .....	380,070	” ”
West Australia ...	975,920	” ”
Tasmania ...	26,215	” ”

The Northern Territory (523,620 square miles), larger than the Union of South Africa (472,550 square miles), the Australian Capital Territory (Canberra), and the Mandated Territory of New Guinea and Papua are administered by the Commonwealth. Australia, excluding the Mandated Territories, is almost as large as the United States, whereas it only has a population of slightly less than eight million. In spite of this, however, Australia is one of the leading agricultural countries of the world, and its products contribute a great deal of the food and clothing supplies of many overseas countries. Its wool production easily surpasses that of any other country, and it produces more than 50% of the world's Merino wool. According to the latest statistics, there are 115 million sheep in Australia and the woolgrowers' cheque (estimated in February, 1950) was £250 million, i.e. £55 million more than the previous year's record. The world's largest Merino stud farm in New South Wales covers 52,000 acres and grazes a maximum of 120,000 sheep. In 1949 there were nearly 5 million dairy cattle (the Jersey breed predominating) producing more than 1,206 million gallons of milk, 165,684 tons of butter and 43,318 tons of cheese. There are approximately 10 million beef cattle (mainly Aberdeen Angus, Hereford and Shorthorn) and some of the cattle freeholds in the Northern Territory cover thousands of square miles. The largest cattle station in this territory covers an area of approximately 10,000 square miles. It is maintained that this sparsely inhabited territory has great pastoral and agricultural potentialities. Much of the interior of Australia has, however, a rainfall so low that it does not carry any stock at all. Much of the central area is also deficient in water supplies, whereas in the rest of New South Wales, Victoria, South Australia and West Australia irrigation and water conservation have done much and will do more to bring about closer settlement. Under favourable conditions one acre can be expected to maintain at least one fat lamb and the ewe, whereas, at the other extreme where climatic and soil conditions are only suited

to sparse pastoral settlement, as many as 10 to 20 acres may be required to maintain one sheep throughout the year. The stock population is, therefore, not evenly distributed throughout the Commonwealth. The densest populations of sheep and cattle are to be found along the south-east of Victoria and along the east of New South Wales and Queensland. It is interesting to note that in certain areas beef cattle are maintained to control the growth of grass and keep the pastures well adapted to the requirements of sheep. Such cattle are known as "animal mowers."

#### DISEASES OF FOOD-PRODUCING STOCK

Pleuro-pneumonia (lungsickness of cattle) occurs, but there are no virus diseases of the food-producing stock of Australia. The only major tick problem is found in the extreme north of New South Wales and in Queensland. *Boophilus microplus*, the blue tick of Australia responsible for tick-fever, is found only in the northernmost district of Casino in New South Wales, and in the greater part of Queensland. At one time it had spread as far as the district of Grafton in New South Wales, but by 14-day dipping in arsenical preparations for a period of 15 months, under rigid control, this tick was been driven back. An arsenic-resistant blue tick has manifested itself in certain areas of Queensland. Blow-fly, buffalo-fly and the parasitic nematodes continue to be major problems in Australia.

Diseases such as *tuberculosis*, *brucellosis*, *mastitis*, etc., do not occur to the same extent as in the Union, neither are there so many poisonous plant problems. Major animal disease problems seem to be associated with nutrition and metabolic disturbances. Aphasphorosis seems to be widespread, less so cobalt and copper deficiencies. The latter is responsible for such diseases as *enzootic ataxia* (sway back) in sheep and falling disease in cattle. On the other hand, much publicity has been given to the occurrence of chronic copper poisoning in grazing sheep in Australia, and the whole copper problem is being very intensively and extensively studied by Dr. Bull and his team of research workers. The problem of infertility in sheep, due to the presence of a potent oestrogenic principle in subterranean clover, made its dramatic appearance in 1941 in Western Australia. Dr. Bennetts and his team at the Research Laboratory Nedlands, Perth, are giving this problem serious consideration. It would appear that a form of balanitis (or sheath-rot) in sheep, affecting as many as 5% on certain stations (e.g. Mt. Isa Station, Victoria) may be associated with a high protein diet. Rabbit eradication (control and research) looms largely in the foreground on account of the great damage caused. Increased infestation seems to occur in cycles and in some areas it is estimated that an average rabbit warren in a 100-acre paddock will take in the grazing of 15 sheep.

It is the intention to deal with the research aspect of some of these problems and the occurrence of similar conditions, under the South African environment, in a further paper.

In Australia there is a growing demand for veterinary guidance and help, not only for disease control and research, but for advisory

and extension work, both as regards animal health and animal husbandry. For this vast continent separate veterinary services are maintained by the Commonwealth and the Governments of the States.

There are a number of posts vacant, and establishments will undoubtedly be increased as additional veterinarians become available. The increases which have occurred have assisted veterinary control considerably, as the increased man-power has allowed of reduction in the size of districts over which official veterinarians have to operate.

In 1946 there were the following registered veterinarians in Australia :—

TABLE I.

	Holders of Degrees or Diplomas	Registered only
Queensland .....	36	74
New South Wales .....	112	116
Victoria .....	134	9
South Australia .....	13	8
West Australia .....	15	11 + 23*
Tasmania .....	11	few
Total .....	321	± 240

\* Permit holders.

In 1946 there were approximately 163 veterinarians in the service of the Commonwealth and of the States. It is, however, not clear how many of the some 240 registered veterinarians, not the holders of veterinary degrees or diplomas, were in the service of the States or the Commonwealth. Probably the majority of approximately 560 registered veterinarians were in private practice in 1946.

Veterinary services are also assisted by a large number of lay staff, such as inspectors of stock, meat inspectors, border crossing inspectors, etc. In New South Wales there were in 1946 approximately 700 lay staff operating under the Board of Tick Control.

Compare the above with the serious staff shortage position in the Union, where the stock industry is threatened by a number of serious infectious diseases.

#### THE COMMONWEALTH VETERINARY SERVICES

The Division of Veterinary Hygiene in the Department of Health at Canberra exercises control over the importation of animals and animal products into and the export from Australia. It also administers disease control and meat inspection of the Australian Capital Territory (Canberra). The Meat Inspection Branch of the Department of Commerce and Industry is responsible for the inspection of meat and other products intended for export, i.e. it administers the provisions of the

Commerce Act, whereas the Animal Industry Branch in the Department of the Interior is responsible for stock disease control and meat inspection in the Northern Territory.

The Chief Veterinary Officers of the different States are appointed by the Commonwealth as Chief Quarantine Officers and they use their state organisation for carrying out the provisions of the Quarantine Act.

The introduction of an infectious animal disease, not existent in Australia, empowers the Director of the Division of Veterinary Hygiene of the Commonwealth to convene a meeting of the interested Chiefs of Animal Health and Production of the States concerned, to review the situation and recommend the action that should be taken.

#### VACCINES, SERA, BIOLOGICAL PRODUCTS FOR VETERINARY USE.

These are manufactured by the Commonwealth Serum Laboratories at Royal Park, Melbourne.

#### RESEARCH IN ANIMAL HEALTH AND PRODUCTION

This is vested in the Division of Animal Health and Production, one of the Divisions operating under the Commonwealth Scientific and Industrial Research organisation. This new body under the Act of 1949 has similar powers and functions to the old C.S.I.R., which initiates and carries out scientific researches into Australian primary and secondary industries, for making grants in aid of pure scientific research, etc. Dr. I. Clunies Ross is the new full-time chairman of the Executive, with two full- and two part-time members.

The Chief of the Division of Animal Health and Production, Dr. L. B. Bull, is entrusted with the central organisation, with headquarters at the Animal Health Laboratories at Parkville N2, Melbourne.

The Division of Animal Health and Production controls and finances the following Research Laboratories and Field Stations:—

1. The Animal Health Laboratory at Parkville, Melbourne, with its field station at Werribee, Tooradin, and Baroogo. The following are a few of the problems which receive the attention of this organisation: Contagious bovine pleuro-pneumonia, toxæmic jaundice and chronic copper poisoning, mastitis in dairy cattle, sheep physiology, etc.

The main laboratory at Parkville is a well-equipped three-storey building, situated in the university grounds and wedged in between the City Hospital and the remains of the old Gilruth Veterinary School, part of which is utilised by the Division and part by the State of Victoria for diagnosis and investigation of stock diseases.

2. The McMaster Animal Health Laboratory at Sidney has a branch laboratory at the University College of Armadale, the McMaster Field Station at Badgery Creek, the National Field Station Gilruth Plains, Queensland, and the Fleece Analysis Laboratory at Villawood, New South Wales. The following are some of the most important problems receiving their attention: Helminthic physiology and therapeutics, wool biology, the bio-

chemistry of sheep diseases, mineral metabolism and diatetics, zebu hybridisation, etc.

The McMaster Laboratory at Sydney is wedged in between the buildings of the Veterinary School in the University grounds. The prospective development of the Veterinary School will almost preclude the McMaster from occupying further sites within its grounds. Plans have, however, been completed for the erection of a spacious field laboratory at Prospect.

The Division of Entomology of the C.S.I.R.O. has included entomological investigations in its research programme, whereas the Division of Biochemistry and General Nutrition at Adelaide has as one of its major activities research into basal problems of animal nutrition, particularly in sheep.

The C.S.I.R.O. Division of Animal Health and Production collaborates with State Departments of Agriculture in the case of certain problems. At the Queensland State Animal Health Station at Yeerongpilly, investigations on the cattle tick and buffalo-fly are being carried out.

In these research organisations many graduates besides veterinarians are employed, particularly chemists, biochemists, bacteriologists, statisticians, etc. It should be stressed here that the majority of these research workers under the jurisdiction of the C.S.I.R.O. are relieved of routine and administration. They are able to devote all their time to their respective research projects, and most of the problems are undertaken by teams of research workers. As a result of this organisation, great progress in fundamental research is being made, much more so than at present in the Division of Veterinary Services; Union of South Africa. Unfortunately at Onderstepoort, research is being smothered by routine as a result of the great shortage of trained professional staff and the very marked increase in activities. Furthermore, lack of appreciation and understanding on the part of the authorities of the magnitude of the problems in South Africa and of the economic importance of fundamental research, has added to this deterioration. Australia has the further great advantage in that problems dealing with animal health and production are not conducted in water-tight Divisions.

#### VETERINARY SERVICES OF THE STATES

Although acts differ in respect of title, subject matter, etc., much the same provisions exist in all the States for the control of disease; the machinery may, however, vary. Greater differences occur in regard to legislation concerning animal husbandry and meat inspection. In Queensland meat inspection, except for export, is undertaken as part of the State Veterinary Service, whereas in the other States it is administered by the State Department of Health. These Veterinary Divisions have different designations in the different States.

#### QUEENSLAND

The Division of Animal Industry of the Department of Agriculture and Stock, Brisbane, administers various acts, such as the Stock Act (which provides for the control of scheduled diseases of stock and the

prevention of introduction of disease from other States, etc.), Stallions Registration Act, Pig Industry Act, Poultry Industry Act, and the Brands Act. It also administers, through the Veterinary Surgeons' Board, the registration of veterinarians, and supervises the registration of veterinary medicines desired to be marketed in Queensland. Its Pest Destroyers Board controls all pest destroyers, including sheep and cattle dips. Research and diagnostics are conducted at the Animal Health Station at Yeeronpilly.

#### NEW SOUTH WALES

The Division of Animal Industry, Department of Agriculture, Sydney, administers more or less similar acts. Research for this Division is conducted at Glenfield (not far from Sydney) with a number of subsidiary stations. It is at present collaborating with the McMaster Institute in conducting a number of feeding investigations under drought conditions. Interesting results have so far been achieved.

A Board of Tick Control at Lismore, New South Wales, carries out a policy of tick eradication and control, as indicated by the Commonwealth Cattle Tick Control Commission. All dips (about one mile apart in the infested area) are government-owned and dipping is carried out by the Board.

#### VICTORIA

The Livestock Division of the Department of Agriculture, at Melbourne, administers more or less similar acts for the control of disease, improvement of stock, registration of veterinary surgeons and of stock remedies, etc. The diagnostic and research work of the Livestock Division of the State of Victoria is carried out at the University Veterinary Research Institute, Melbourne, the Department of Agriculture paying an annual grant to the university towards the expenses.

The Livestock Division of the Department of Agriculture, South Australia, the Stock Branch of the Department of Agriculture, Western Australia, and the Animal Health Services, Department of Agriculture, Tasmania, exercise more or less similar functions of control of diseases, etc., as the other States referred to above.

#### TRAINING OF VETERINARY SURGEONS IN AUSTRALIA

There are three Faculties of Veterinary Science respectively at the universities situated at Sydney, Melbourne and Brisbane, which grant degrees in veterinary science. At present only the Faculty at Sydney teaches the full five years' course. With the greatly increasing demand for graduates, the number of students had rapidly increased to 310 in 1946. Included in these are a number of New Zealand students, where there is no Faculty of Veterinary Science, and students from Malaya, etc. Full particulars of the curriculum and training will be found in the calendars of the Sydney University, a copy of which is in the Onderstepoort Library. In 1946 there were the following full-time staff and part-time lecturers:—

##### (a) Full-time Staff:

Dean of the Faculty and Professor of Veterinary Science.  
Reader in Veterinary Surgery and Obstetrics.

Senior Lecturer and two Teaching Fellows in Anatomy.

Lecturer in Zootechny.

Senior Lecturer, Lecturer, two Teaching Fellows and Demonstrator in Pathology and Bacteriology.

Lecturers (two) in Medicine.

Lecturer in Animal Husbandry (at McGarvie Smith Animal Farm).

Superintendent of the Veterinary Hospital.

Junior House Surgeon

Clinical Officer

Biochemist

Teaching Fellow in Animal Genetics

} At McGarvie Smith  
Animal Husbandry  
Farm.

(b) Part-time Lecturers :

Eight, giving instruction in Physiology, Principles of Nutrition, Veterinary Epidemiology, Veterinary Parasitology, Diseases of Poultry, Diseases of Sheep, Veterinary Jurisprudence and Meat Inspection.

Professors and Lecturers for first-year subjects are those of the relevant Departments of the Faculty of Science. The subjects of Physiology, Biochemistry and Histology are taught in the Faculty of Medicine, the major part of Physiology II being taught by a veterinary graduate within that faculty.

McGarvie Smith Animal Husbandry Farm: This farm of 400 acres, owned by the University and managed by the Faculty, is situated 30 miles from Sydney. Residential accommodation is provided for fifth-year students, who spend the whole of the academic year at this centre.

Clinical Training: This is provided at the Veterinary Clinical Hospital within the university grounds during the fourth year and at the university farm during the fifth year. In addition, students are required to spend at least six months during their course in general field work, of which ten to twelve weeks take the form of an extra-mural term between the fourth and fifth years, when the type of training experience is laid down by the Faculty.

Scholarships and Cadetships: Additional to certain scholarships granted by universities where instruction is given in veterinary science, State Governments and others employing veterinary graduates give cadetships (the equivalent of a scholarship) which provide for the tuition fees and residential expenses for a number of students. Cadetship holders may be called upon to join the service of the Department granting the cadetship and have provided a valuable means of recruitment for these services.

*(To be concluded in the next issue.)*

**NOTE:** The writer is most grateful to Dr. Wardle for his valuable notes, which were frequently quoted. He is also very much indebted to Drs. Bull, Turner, Gill, Bennetts, Edgar and other colleagues for their courtesy, valuable information and interesting discussions. He was also welcomed to Australia by Dr. Gregory, President of the Australian Veterinary Association.

# THE OCCURENCE OF THE " KIDNEY WORM " OF SWINE (STEPHANURUS DENTATUS, DIESING 1839) IN NATAL.

S. A. R. STEPHAN, S. G. TURNER and R. A. PAINTER.

This brief article is published with the object of recording the finding of *Stephanurus dentatus*, Diesing 1839, for the first time in the Union of South Africa. It was found by Turner, identified by Stephan and the identification kindly confirmed by Ortlepp of Onderstepoort. The authors are of the opinion that this worm is not confined to Natal but, owing to the prevailing methods of dressing of carcasses in abattoirs, it is very probable that it has been missed.

*History:* The attention of the second author was drawn to an extensive peritonitis in the lumbar region of a pig, and owing to this, the kidneys and perirenal fat had not been removed, as in all cases where the carcase appears normal. On cutting into the fat many cysts were encountered and, on sectioning these, the presence of the worms was established. Further cysts were found in the fat in the region of the ureters as well as numerous small abscesses in the vicinity of the walls of the tubes.

Similar lesions were found in four pigs out of a batch of five sent in by this owner from his farm in the Highflats area. This farm was visited by the third author, who found a few young pigs that were not thriving. The owner was persuaded to send these into the abattoir, and again *Stephanurus* was observed. Pigs sent from an adjoining farm showed that one was infected with *Stephanurus*. Both farmers were grazing some of their pigs over common damp grazing.

*Specimens Examined:* These consisted of the kidneys, ureters and associated perirenal fat of one affected pig. The lesions in the fat were confined for the most part to the vicinity of the ureters and consisted of numerous small abscesses containing dead parasites and pus and of cystlike lesions communicating by means of channels with the ureter. These small cystlike lesions contained up to four live worms.

The kidneys showed extensive pathological changes, although only two worms were found embedded in the cortex. There was atrophy of the medulla and a part of the cortex. The pelvis of the kidney and the ureter leading from it were very much dilated as a result of these cysts and abscesses, causing a partial stenosis of the ureters by pressure. The left kidney was more affected than the right. A large number of live and dead worms were removed. These worms vary in size from 20 mm. to 40 mm. and their outer covering appears to be somewhat transparent. They are easily seen.

*The worm — Stephanurus dentatus:* A description of this worm can be found by referring to any up-to-date textbook on Veterinary



Helminthology. Hutyra, Marek and Manninger (1938) state that the disease is very prevalent in America, Annam, Malaya, the Philippine Islands and in Australia. It is claimed that it is regarded as one of the most serious of swine diseases in the Southern States of North America, while in the exporting districts of Australia 62 to 84% of slaughtered swine are, as a result of the disease, unfit for export to England and 11% are altogether unfit for human consumption. It will thus be appreciated that if this worm should spread in the Union much loss is likely to result to the pig breeder. Mönnig (1934) states that infection takes place per os or through the skin. The life history is a complicated one, taking 100 or more days from the time of first entering the body to the time it reaches its real predilection sites, the perirenal fat, the ureter and the pelvis of the kidney.

*Symptoms:* Hutyra, Marek and Manninger (1938) state that as a rule no symptoms are shown in the case of mild or moderate infestations with this worm. In heavier infestations there may be loss of appetite, emaciation and slight inco-ordination in movements of a hind leg, or even an actual lameness up to paresis or paralysis of the hind-quarters. This paralysis is interesting, as so many cases of paralysis are seen in pigs.

Ante-mortem examination of the pigs from which worms were obtained was negative, the pigs appearing quite healthy. The young pigs, which were sent in after examination by the third author were definitely in poor condition, and were not thriving. Worms were obtained from them.

This farm is situated in the Highflats area of the Ixopo District of Natal, and the farmer runs about 130 pigs, made up of eleven breeding sows, one boar and the rest are baconers, weaners and young piglets. They are crosses between Tamworth, Large White, and Large Black pigs. There are 22 concrete styes of modern pattern with drinking and feed troughs. The styes are partly covered with thatched roofs. The floor in each sty is well sloped and drainage is adequate. The troughs are not removable but can be easily cleaned, and water is laid on to all the styes.

The management is as follows: The only pigs that are allowed out of the pens are the boar and the breeding sows. These latter animals graze in two low-lying areas, one of which has a stream running through it. There are numerous mud hollows where the pigs wallow. When a sow is close to farrowing she is brought back into the pens and allowed out again when her piglets have been weaned.

The only exception to this method was when the farmer was short of accommodation and was forced to allow six Large White piglets to run outside with their mothers. All these six were "bad doers" for no accountable reason, as their mother was a good sow.

As far as the previous disease history of the pigs goes there were two condemnations for tuberculosis last year and one for measles a few months ago.

The styes are washed out daily with water, using bass brooms. The bedding is changed once a week.

The history of the infected pigs is as follows: Five sows were

taken to the abattoir on or about 10th March. Four of the five harboured *Stephanurus dentatus*. With the exception of one pig they were all bred on the farmer's previous farm and their ages varied from 4-6 years. One of these sows, which had been bred at Rietvlei, had showed a temporary paresis in January after farrowing, but had recovered within a week.

Within the last year the farmer had introduced the following pigs on to his farm: from Johannesburg, one boar; Greytown, eight Large White sows; Southbroom (South Coast), seven Tamworth sows.

Generally speaking all pigs on the farm appeared to be quite healthy. There were isolated "bad doers" and these were pigs that had had access to the grazing lands.

The following prophylactic measures were suggested:—

- (1) All pigs should be removed from the lands for a period of seven months.
- (2) Daily washing out of the styes and bedding to be changed twice a week.

#### SUMMARY

The discovery of *Stephanurus dentatus*. Dying 1839 in the Union is recorded for the first time.

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## RESPIRATORY ACARIASIS OF CANARIES.

S. A. R. STEPHAN, V. R. KASCHULA and A. S. CANHAM,  
Allerton Laboratory, Pietermaritzburg.

In 1947 a well-known canary fancier brought some dead canaries to Allerton for examination. He stated that all of the dead canaries had shown respiratory distress and many other of his live birds in their cages were also affected in a lesser or greater degree. He was satisfied it was not ordinary cold to which canaries were susceptible in view of one special symptom, a peculiar sucking sound occasionally made by the affected birds.

The history was as follows: He had kept canaries at Cape Town from 1938 to 1946 when he was transferred to Pietermaritzburg. His birds were distributed among friends in Cape Town and Port Elizabeth until the aviaries in Pietermaritzburg were ready. The birds then travelled by train in open boxes, covered with wire netting, and the journey to Maritzburg took two days. On arrival, two were found dead and about six were obviously ill, showing respiratory symptoms. Odd losses continued during 1946, all of which showed these same symptoms, which were ascribed to colds.

After the first breeding season it was noticed that some of the progeny of these old birds had no voice. Some eventually recovered, while others simply wasted away and died. The infected adults bred normally.

Canaries were brought to Allerton during August, 1947, for post-mortem examination. On examining the unopened tracheæ minute black specks were seen in the lumen. These were found to be mites, the dark colour being due to ingested blood.

These mites were handed to Dr. R. F. Lawrence, the then Director of the Natal Museum, who decided this was a new species of *Sternosoma*, viz.: *Sternosoma tracheacolum* n. species grouped as follows: Parasitoidea. Family: Rhinoyssidae Trouessart. *Sternosoma* Berlese and Trouessart 1889. A full description was published in the Journal of Parasitology, October, 1948, Vol. 34, No. 5. To quote Lawrence: "No mites from the trachea or nasal cavities of canaries seem to have been previously recorded and the species in question probably has its closest relationships with *Sternosoma cryptorhynchum*, a mite described by Berlese and Trouessart (1899) from the nasal cavities of the European sparrow *Passer domesticus*. The canary mite has never been found in the nasal cavities.

From information received from various prominent canary breeders in the Union this condition has never been heard of or seen before. It is, however, suggested that this is not the first case of its kind that

has occurred in South Africa, the explanation being that these mites have never been looked for and consequently never been found.

That this parasite spreads by direct contact is borne out by the fact that the owner of the infected canaries gave another canary fancier some of his birds. In due course a number of the second fancier's canaries started to show similar symptoms, and in several that were post-mortemmed this same mite was found. It is also suggested that mother birds infect their young while feeding them with regurgitated food.

In the book "Canaries" by C. A. House (a world-famous expert, judge and journalist), the following paragraph appears:—

"The late Jacob Mackley was the first to bring to my notice the fact that parasites do kill canaries. It often happens that a canary, which is full of life and vivacity during the day, is continually coughing during the night. When this happens the bird should be caught, its beak opened and, by the aid of a strong magnifying glass, its throat should be carefully examined. It will then be seen that adhering to the sides of the throat are a number of parasites."

It is suggested that these parasites emerge from cracks in the cage at night and enter the nostrils of the canaries, but they cannot get further on account of the mucous present. The parasite is not named, but it is unlikely to have been *Sternosoma tracheacolum*, as this parasite has never been seen in the throat and never causes coughing.

Lawrence (1950), in a personal communication, states that endoparasites have been found in the nasal cavities of quite a number of wild and semi-domesticated birds, but not in the trachea or lungs. Mites are found in the lungs of seals, primates, snakes and procavias where they seem to do no harm. In all these the males are rare in comparison with females.

*Symptoms:* The symptoms shown are essentially respiratory in character. The affected bird appears to be "puffed up" and shows slight heavy breathing while sitting quietly on the perch in a sleeping position. As the intensity of the symptoms increases the bird shows a degree of irritation of the trachea, which is characterised by a peculiar sucking or smacking sound usually made twice in succession followed by quietness. The males refuse to sing. Eventually the bird squats on the floor and marked respiratory distress is shown by sneezing, frequent opening of the beak and repeated attempts to clear the throat, together with gasping respirations. The condition of the bird rapidly deteriorates. Towards the end, it selects a corner of the cage in which to die. There is a discharge from the mouth and nose and, after slight struggling, it falls over with its beak in the sawdust at the bottom of the cage. This is not the usual position in which dead birds are found—they usually lie on their sides. Post-mortem examinations reveal a variety of conditions:—

- (a) Pneumonia involving both lungs or one lung or a section of a lung. Mites may be found in the bronchioles and lung substance.
- (b) Aerocystitis varying from a slight reddening of the membrane to

a chronic inflammation with pus formation involving one or more of the air-sacs. Mites are frequently found in the air-sacs.

- (c) Tracheitis varying from a slight injection of blood vessels to an acutely reddened trachea with excessive amounts of mucus, which may occasionally be blood-stained with few or many mites. These may be seen with the naked eye through the unopened tube as small dark specks. They are frequently seen concentrated near the caudal larynx or syrinx. The mites found here are usually coated with a very tenacious mucus which is difficult to remove. From many post mortems carried out it was found that mites varied in number from one to sixty.

The habitat of the mite varies. They are very frequently found in the trachea where they move about freely and do not fix themselves to the membrane. They are usually but not invariably coated with mucus. They also move about freely in the air-sacs. The lungs are a further predilection site. If a portion of lung is flattened between two slides all stages from young females to mature female mites are found, and it was from here that the male mites were discovered. The female mites greatly outnumber the males. Some are found in the small bronchioles, but it appears that many are actually in the lung tissue. On one occasion they were found on the mesentery and it is assumed that, in the process of carrying out the post mortem, an air-sac was ruptured and the mites escaped on to the mesentery. On the other hand they may have made their way through the caudal larynx into the abdominal cavity. They have also been found on the surface of the liver. They have been identified from birds in two aviaries in one town and in a bird sent in from another town nearly 60 miles away. This condition may be much more prevalent in the Union and elsewhere than is at present realised.\* These mites are very active and can move rapidly in the air-sacs or on the surface of the liver. In spite of a most careful search, which was maintained for over a year, no male specimen of the mite could be discovered. Some females have shown a large egg in the abdominal cavity, some of which have contained a young larval form. How these leave the female is unknown unless it is by rupture of the body, followed by the death of the female.

From the last bird examined on 22nd February, 1950, a number of small reddish-coloured mites were obtained from the bronchioles of the lung which have been identified by Dr. Lawrence as males of the species and will be described in a further article by him. Lawrence (1948) remarks that "It is remarkable that the majority of *Rhinomyssid* species have been based on female species, the males being unknown." This is also the finding of Vitzthum (1935).

**Treatment:** The question of treatment was naturally of the utmost importance. Individual dosing of large numbers of small canaries is quite impracticable as is the placing of drugs in the drinking water. No known drug, dissolved in water, would have any effects on mites in the trachea and air-sacs. The only line of treatment that appeared to hold out any hope of success was by inhalation. The use of a finely powdered drug was suggested.

The first drug tried was sulphur dust. Four canaries were confined in a cage, the cage was covered with a cloth and sulphur dust was insufflated to form a fairly heavy fog, the exposure lasting three minutes. As a result one bird died of asphyxia, the sulphur dust having accumulated in the tracheal mucus and occluded the trachea. The remaining birds showed respiratory distress the following day and one died on the fourth day. Post mortem revealed an acute foreign body aerocystitis caused by the sulphur dust and the mites present were unaffected. The birds that remained alive after three weeks were visibly ill and miserable and suffered from diarrhoea. Post mortem revealed an aerocystitis caused by the sulphur and all mites remained unaffected. This treatment was consequently discarded.

The second substance used was Barium Antimonyl Tartrate — a drug advised for the treatment of Syngamus trachea in England. Three canaries were treated with this drug, but all died within two days. The method of application was the same as for the sulphur dust. On removing them from the cage after a three-minutes' exposure they showed respiratory distress and dribbled from the beak. Post mortem revealed the presence of live mites in the trachea.

Double Benhex Powder was then tried. This contains DDT and Benzine Hexachloride. Various methods of administration were applied with varying degrees of success. The method finally adopted was as follows:—

A show cage 6 ft. long by 1 ft. wide and  $1\frac{1}{2}$  ft. high was utilised. Twenty-five canaries were placed in it, and the front part of the cage was covered with a loose muslin cloth to prevent air currents inside. All water and food containers were removed. By means of an insufflator Double Benhex Powder was pumped into the cage to make a slight fog, and after a five-minutes' exposure to this powder the cloth covering the cage was removed. This same treatment was carried out the next day and then two further treatments at weekly intervals completed the administration.

Many of the birds treated showed almost immediate improvement and were drafted to other smaller cages. Those that only improved slightly were given further similar treatments at 14-day intervals. Improvement was judged by the cessation of the typical smacking or sucking sound made by infected birds, the commencement of chirping and singing and the return of normal respiration. Birds that succumbed were those showing marked symptoms, and post-mortem examination was carried out on all of them.

After the first week's treatment various canaries were sacrificed. Both dead and living mites were found in some cases. In others the dead mites became encapsulated by the mucous membrane, presumably as a result of a mild chronic inflammation and the production of new tissue. Other mites showed disintegration, while the remainder were dead but unaltered in appearance.

The main difficulty appears to be the fact that many of the mites

are coated in thick mucus, and it is impossible to bring the powder into direct contact with their bodies. Unfortunately neither of the ingredients of the dust is soluble.

Double Benhex Powder is toxic to canaries, but presumably only after the birds have been exposed to it for a considerable time. It should be regarded as a cumulative poison as, after the third treatment, traces of the powder can be found in the air-sacs. In birds undergoing their sixth or seventh treatment symptoms of poisoning are likely to occur. Diarrhoea appears first, followed by a peculiar nervous twitching of the body. Tonic spasms of the body also occur and the bird is unable to hold on to the perch. They sit on the floor and die within one to three days from the onset of symptoms.

The following breeds of canaries were involved: Clear white border, Yorkshires yellow, buff, cinnamon and green. Of all varieties the clear white border canaries were less resistant to both the mites and the treatment. A fair number of canaries were returned as cured to the owner.

It is not claimed that the Double Benhex treatment is a specific for the condition, but it assists greatly in keeping the mite under control. The owner claims improvement in his aviary since adopting this line of treatment.

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# A PRELIMINARY SURVEY OF BOVINE DISEASES AFFECTING THE QUALITY AND SAFETY OF A LARGE TOWN'S MILK SUPPLY.

L. W. v. d. HEEVER,  
Germiston.

With the appointment for the first time of a veterinarian to the local Municipal Health Department, this survey was undertaken with a view to obtaining some idea of the incidence of certain diseases in the herds supplying the town with milk for consumption in the raw state. Particular attention was paid to those two most important infectious diseases, tuberculosis and brucellosis, while streptococcic mastitis and mange, having direct bearing upon the quality of the milk, were also considered.

The procedure adopted was as follows:— The herd was always inspected at milking times. A brief history of the herd was obtained from the person in charge, after which it was examined. Special attention was paid to cows that showed any signs indicative of any of the above diseases, such cows being specially examined and the necessary specimens taken for further examination. At this stage it should be emphasised how important proper means of identification of animals are.

Bulk or group samples of milk were then taken, and every effort was made to obtain a sample representing the milk from every cow in milk at the time. To ensure this, a small amount of milk from every quarter of every cow was milked into a sterile container in the case of producer-distributors' herds, as most of this milk was bottled directly after coming off the cooler. In the case of producers, a 10 cc. sample was taken from every can after the contents had been well mixed. Sampling from individual cows had the advantage that one could examine each udder clinically, and furthermore the sample obtained was in most cases almost entirely free of contamination from external sources.

The samples were kept in the refrigerator until they were examined. The tests applied were those which would indicate the presence of one or more of the above diseases through serological reactions or pathological changes, or through the actual demonstration of the organisms. The following tests were applied:—

- (a) For Tuberculosis — Guinea-pig Biological Test.
- (b) For Brucellosis — (1) Ring Probe Test.  
(2) Guinea-pig Biological Test (inc. Agglutination of G.P. Blood).
- (c) For Mastitis — Test by microscopical examination.

In all cases the samples tested were of a composite nature. In the case of a producer-distributor the herd was divided into various groups



according to the arrangement of the cows in the stable, while one sample representing the whole herd was taken from that of a producer.

Briefly, the following laboratory routine was followed:— After thoroughly shaking the sample bottles, 1 cc. of milk was placed into a Ring test tube and 5 cc. into the Mastitis test tube by means of a sterile pipette. One drop of stained Antigen was added to the milk in the Ring test tube, and after a thorough mixing this tube was put into the incubator at 37°C for 50 minutes. The Mastitis test was plugged with sterile cotton wool and left in the incubator at 37°C for 24 hours, after which smears were made, stained and examined microscopically.

With the same pipette, 10 cc. of milk was put into each of six centrifuge tubes and spun at approximately 3,000 r.p.m. for 15 minutes. The deposit and the cream was mixed to form the inoculum for injection into guinea-pigs, two animals being used for each sample. These were killed after 6 - 8 weeks and post-mortem examinations were held. Blood samples were collected and despatched for the Agglutination Test, and organs were sent for histo-pathological examination where necessary. Organ smears were made where indicated, and sub-inoculation of material was also resorted to.

The following is a summary of the results obtained:—

No. of samples examined .....	98
No. of samples with a positive Ring test .....	71
No. of samples with a positive T.B. Biol. Test .....	3
No. of samples showing a positive Mastitis Test .....	18
No. of samples showing a Brucellosis confirmed by Biological Test .....	27

At this stage a few brief observations on the various tests and the results may be permitted.

(1) *Ring Probe Test*: Van Drimmelen (1948) described this test and mentioned its wide use in Scandinavian countries, its value in surveys and in milk control generally. The test was found to be simple to perform, and the results seem to be reliable, as of the 27 samples where *Brucella* organisms were demonstrated in the milk, only one showed a negative Ring test. The herd from which this sample was taken perhaps warrants a special note:

Ever since the occurrence of abortions due to Brucellosis three years before, the owner had been inoculating all his calves and every cow, 3 - 4 weeks after parturition, with strain 19 vaccine obtained from Onderstepoort. The cows were done after every calving, whether they had been inoculated as calves or after previous calvings or not. Van Drimmelen (1949) has shown that after 2 - 4 months, inoculated bovines will only show a positive Ring test when the milk is undiluted, but that it will not continue to do so when the milk is diluted with known negative milk. A negative bulk sample test from this herd may therefore possibly be explained as having been due to dilution, whereas the positive Biological test result may have been due to strain 19 *Brucella* organisms having been voided in the milk, although no details of this can be given.

(2) *Biological Test:* This was a combined test to detect the presence of Tubercle bacilli and Brucella organisms in the milk.

(a) *Tuberculosis:* Three samples were positive, and acid-fast bacilli were demonstrated in organ smears. The organ lesions were typical. Blood samples were negative to the Brucella agglutination test.

This constitutes almost a 3% infection, and which approximates to Pullinger's (1942) findings of 2.5%. It may be of interest to note that in the first herd (one of 100 odd, adult cows), no cases of udder tuberculosis could be found, and one cow with an extremely enlarged tuberculous precaval lymph node was detected clinically. Tuberculin tests (arranged for by the owner) showed a total of seven reactors, and all showed lesions of tuberculosis at post mortem. The one cow which was eventually found to be voiding the bacilli (by biological tests on the milk) showed no macroscopic lesions in the udder or the supra-mammary gland, but only a primary parotid abscess which was very small (see Pullinger, 1941). All the reactors were in excellent condition.

In the two other groups of cows, mastitis of various types and causes was commonplace and a number of cows had udders which were grossly indurated. These cows were sold between the time that the bulk sample was taken and the date on which the tests were read. Repeated subsequent tests failed to find any tubercle bacilli in the milk, so that the responsible animals were probably disposed of. The owner informed the author that some of these cows had been condemned at an abattoir for tuberculosis, but this could not be confirmed.

It is therefore of interest to note that in the first herd, clinical examination failed to reveal the offending animal, due to no detectable udder or gland lesions being present. According to Francis (1947) tuberculous cows showing no udder lesions can at times shed bacilli in their milk, periodically or continuously. The possibility of an extremely early lesion being present but undetected should, however, not be ignored. In the other two groups of cows the extensive damage to the udders by various types of mastitis made the clinical detection of the tuberculous type difficult or almost impossible.

The above facts therefore indicate that clinical examination alone cannot safeguard the milk supply as far as infection with tubercle bacilli is concerned. The value of regular biological testing is apparent, but the value of tuberculin testing when bulk samples are found positive cannot be over-estimated.

(b) *Brucellosis:* Twenty-one guinea-pigs showed signs at post mortem of having been infected with brucella organisms, but agglutination tests confirmed this in only 18 samples. Whether some of the guinea-pigs showing an enlarged spleen and enlarged, slightly reddened lymph glands but with negative Brucellosis agglutination tests were actually early tuberculosis, is an open question. Although Topley and Wilson (1946) state that the two infections will develop independently if present in the same inoculum, Van Drimmelen (1948) and Pullinger (1936) had other views on the subject. Histologically, differentiation between lesions of brucellosis and tuberculosis in guinea-pig organs is almost impossible, unless bacteria are found in the sections, which is rare.

Roughly 50% of the guinea-pigs used for these tests were males, and yet only once was a typical Strauss reaction seen in the testicles and epididymises. In this instance a purulent epididymitis was present in the one epididymis. In one other guinea-pig, an extensive purulent condition of the seminal vesicles and other accessory glands was observed. Both these animals showed positive brucella agglutination tests.

Twenty guinea-pigs showed lesions at post mortem which were strongly suspicious of brucellosis, but in only 9 instances was this confirmed by positive agglutination tests. A further 12 samples of blood gave positive agglutination even though no post-mortem lesions could be found. *Brucella abortus bovis* does not survive indefinitely in milk, as shown by Van Drimmelen (1948), so that it is possible that these 9 milk samples contained *Brucella* organisms that were not fully viable, but were capable of antibody production in the guinea-pigs.

Stitt, Clough and Clough, however, state that "agglutination of *Br. Abortus* by the serum of the guinea-pig is sufficient proof that the milk was infected." For the purpose of this survey, only those guinea-pigs showing organ lesions and a positive agglutination test were taken as being positive.

During this survey two guinea-pigs caged together developed salmonellosis through natural infection. This was confusing at post mortem, but it was detected histologically. The blood samples, however, gave positive agglutination tests for brucellosis.

To summarize, in 98 groups of cows, there were 71 (or 72.5%) which contained one or more cows with contagious abortion, and that of these 71 groups, 27 (or 38%) contained cows that were voiding viable brucella organisms in their milk. This indicates to what extent the disease is present in the urban and rural areas of the Highveld, and to what extent milk supplied to the consumer is infected with disease-producing organisms. The urgent need for a national disease-eradicating campaign and for pasteurisation of all uncertified milk is obvious.

(3) *Mastitis*: The microscopical examination of stained smears made from incubated milk samples according to the method described by Van Rensburg (1941) was applied. In the majority of cases, milk taken from cans was found to have such a growth of contaminants after 24 hours in the incubator that except for the presence of leucocytes, no reliable information on the presence of mastitis milk in the sample could be obtained. Where the samples were taken directly from the cows the test was more reliable, and the presence of leucocytes and so-called typical long chained streptococci was taken as positive.

Of the 98 samples examined, 52 were from producer-distributors, and of these 16 (or 31%) were positive. Of the 46 producer samples, only 2 (4%) were definitely positive. This gives a total incidence of 18%, but this figure is considered unreliable because of the reasons given above. Probably the figure of 31% as given for the producer-distributors alone is more accurate, and this corresponds more closely

to the figures given by Van Rensburg (1942), Pullinger (1944) and Meara and Mowat (1947).

(4) *Mange*: Of the 98 groups examined, only 26 showed no signs of infestation. The survey extended through various seasons, so that the well-known influence of summer and winter on the course of this disease should be taken into consideration. Well cared-for herds in good condition were rarely affected, while the reverse was equally true. More often than not the bull was more severely affected than the cows in milk.

Often the disease was localised to the area immediately before and behind the udder, and in these cases it is quite impossible for the milker to properly wash and cleanse the udder prior to milking, with the result that there is the ever-present danger of bacteria-laden crusts and hairs falling into the bucket during the actual milking operation. Moreover, due to the irritation produced by these mites, no cow with this infestation can produce the maximum amount of milk, and is invariably in poor condition. Both Sarcptic and Psoroptic mange were encountered.

Many owners on the highveld have come to take this disease as a normal condition in dairy stock, and consider it incurable, except for the temporary improvement in summer. A great deal of education and propaganda will have to be done before an improvement will be seen, but that such an improvement in the position will be to the dairy farmer's advantage, there is no doubt, as it has a decidedly adverse effect on the cow's production and general condition.

### CONCLUSIONS

(1) From the public health point of view, the incidence of diseases such as tuberculosis, brucellosis, mastitis and mange is alarming, and pasteurisation of all uncertified milk, in conjunction with a national scheme for the eradication of these diseases, is urgently necessary.

(2) Clinical inspection of dairy stock, without regular testing of milk samples, cannot guarantee a safe milk supply and can only give a false sense of security.

(3) For agricultural and economic reasons there is a great need for concerted action to eliminate these diseases from dairy stock. Their unhindered spread can only lead to disaster.

### SUMMARY

Details are given of a survey to determine the incidence of tuberculosis, brucellosis, mastitis and sarcptic mange in dairy herds supplying milk to a large town, with some pertinent observations on the tests applied and the results obtained.

## ACKNOWLEDGMENT

The author is indebted to Dr. J. H. Rauch, Medical Officer of Health, for making this survey possible and for permission to publish the figures, as well as to the Research Officers in the Bacteriology and Pathology sections at Onderstepoort for assistance and guidance.

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# SKIN NECROSIS IN DOGS CAUSED BY THE BONTPOOT TICK (*HYALOMMA AEGYPTIUM*).

A. D. THOMAS.

## *Bontpoot Tick Bites in Other Animals:*

The fact that bites of the bontpoot tick in cattle often lead to painful inflamed swellings and suppurating sores is only too well known in the Bushveld areas of the Transvaal. Compared with other animals cattle are well protected by a relatively thick and tough skin and yet the bite of this particular tick (which has not been incriminated in the transmission of disease) can in itself produce very troublesome lesions.

It is not uncommon to see cattle suffering from a carrying-leg lameness as a result of a tick bite in the interdigital space. (Not to be confused with the paralysis tick *Ixodes rubicundus*, the bite of which may cause complete paralysis in sheep and cattle.) These ticks appear to prefer those parts of the skin less densely clothed with hair such as the perineum, udder, sheath, axilla, etc. Although cattle may not show pain to any great extent, it is more than likely that they suffer appreciably, especially as these bites in cattle tend to become infected by bacteria or infested with the maggots of the screw worm fly, *Chrysomya bezziana*.

No one to my knowledge has ever attempted to assess the damage to udders, genitals, etc., occasioned to cattle by these ticks, but it must certainly be considerable.

Persons who have been bitten by the bontpoot tick would also, I am sure, testify to its being a most disagreeable experience. If it were not that the bite is usually discovered early on account of the intolerable itch which develops, and the offending tick removed or killed at once, the lesions produced might well be much worse.

As regards other animals, apparently little is known of the effects of such tick bites. It is for this reason that the few observations below are recorded, in the hopes that some additional light may thereby be thrown on this subject.

## *Bontpoot Tick Lesions in the Dog:*

Two years ago I was greatly puzzled by the occurrence on several dogs presented for treatment, of peculiar punctures through the skin of the back and sides, as if these had been neatly punched out, leaving a deep, clear-cut ulcer ranging in diameter from 1.5 to 3 cm. and extending down to the subcutis. Only much later did I realize the cause of these perforations.

A collie dog was brought in with a very bad lameness of the hind leg. The limb was not swollen, but was very tender to the touch, especially in the region of the hamstring. On close examination an oval

reddish area was found on the *tendo achillis*, and this definitely appeared to be the centre of the tender area. The owner then volunteered the information that he had removed a bontpoot tick from that spot the day before. He would not believe that a single tick could cause such intense pain and lameness. Fortunately the animal was kept under observation while undergoing treatment. Hot fomentations were applied twice a day during the next two days. The reddish blotch became purplish, then brownish as the central patch of dead skin separated and finally came off leaving a typical neatly circumscribed ulcer in the skin. By then the pain and lameness had decreased. Healing took place by granulation and in less than three weeks all that was left was a small hairless scar.

An opportunity was afforded to observe the development of similar lesions in two other cases, and particularly to note the excruciating pain which can accompany the pre-necrotic stage.

The one was a ridgeback type of mongrel brought in by its owner in great concern, as the dog was stiff and could not move or be touched without emitting loud and repeated yelps of pain. On careful palpation it was established that the neck was the most sensitive part and, after clipping the hair, a round reddish area about the size of a shilling was located. Hot fomentations were applied three times a day. It is significant that the animal did not lie down for the whole of the first day and night, and merely sat on his haunches with head and neck extended, hardly moving at all. The pain gradually decreased and disappeared on the third day of observation, i.e. as soon as the necrotic disc of skin had sequestered and was ready to drop off. Healing by granulation was uneventful and rapid.

The other was a full-grown Irish Setter dog brought in on account of a carrying-leg lameness of the left fore. On the light-coloured skin of the axilla extending down over the left elbow was a slight puffiness and a characteristic reddish area. In the centre was a dark spot or depression with a small scab where one bontpoot tick was still actually attached. Surrounding this was a greyish-brown to purple zone separated from healthy skin by a reddish-pink ridge. After three days of fomentation with hypochlorite solution and urea the necrotic skin came off leaving an ulcer rather larger than had been seen hitherto. The ulcer was irregular in shape, jagged in outline and 5 cm. across. When sufficiently clean the edges of the ulcer were freshened up, brought together and sutured. This greatly shortened the period of healing, as the loss of tissue was considerable. Perfect union resulted in five days without leaving an ugly, puckered scar which would have resulted if left to heal by granulation.

Several other cases have since been seen, one in the groin, one in the axilla and another in the interdigital space which exposed the phalangeal bone. In all these three cases a single bontpoot tick was still attached to the necrotic skin. When sloughing has occurred there is, of course, no evidence of the tick.

As will be noted these lesions require no specific treatment other

than that based on general principles. Of greater consequence in my opinion is the desirability of recognising such lesions as separate pathological entities and in relation to their true cause.

#### SUMMARY AND CONCLUSIONS

1. In small animals, the Bontpoot tick (*Hyalomma aegyptium*) secretes and injects into its victim, at the site of the attachment, a substance or venom capable of causing intense local pain and a circumscribed necrotic lesion probably accompanied by mild and transient general malaise.

2. In microscopic section of the skin of an ox cut through the mouth parts of an attached *Hyalomma* picked at random some years ago, it was possible to establish that within a radius of .5 cm. from the hypostome there was absolute karyolytic and coagulative necrosis of all cells including the tough and generally resistant connective tissue cells of the dermis. This would indicate that the substance responsible was very potent and at least as toxic as that of *B. necrophorus*.

3. Complications due to secondary infection with bacteria or infestation with screw worm (*Chrysomya bezziana*) seem to play a more important rôle in cattle than in the dog.

4. Mainly from an academic point of view, one wonders whether this venomous substance could be isolated and identified and if so, whether cattle could be immunized to protect them against the effects of this particular tick bite.



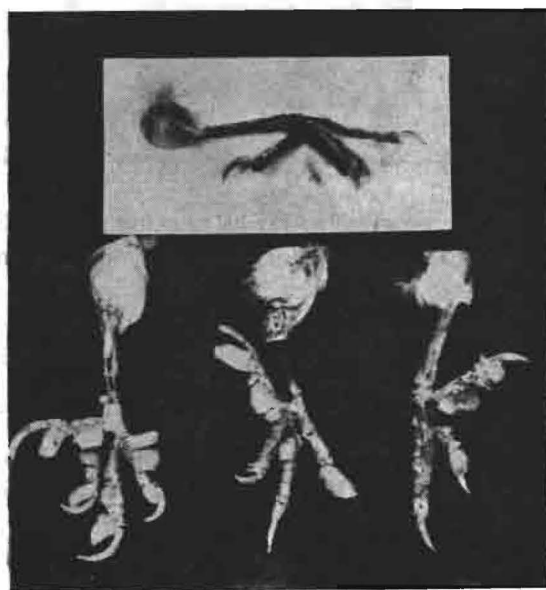


## "SCALY-LEG" OF THE CANARY [SERINUS CANARIA (LINN)].

V. R. KASCHULA,  
Pietermaritzburg.

During the course of the writer's diagnostic experience he has frequently been called upon to give an opinion on bird diseases. In this service he has encountered several mite diseases of the canary, namely tracheal acariasis caused by *Sternostomum tracheacolum* n.s.p. described by Lawrence (1948) and further discussed by Stephan *et alia* (1950)\*. He has also quite often encountered a condition which he prefers to call "Tassle-foot," but owing to its similarity in ætiology and pathology to "scaly-leg" of the fowl, it is being described under the latter name.

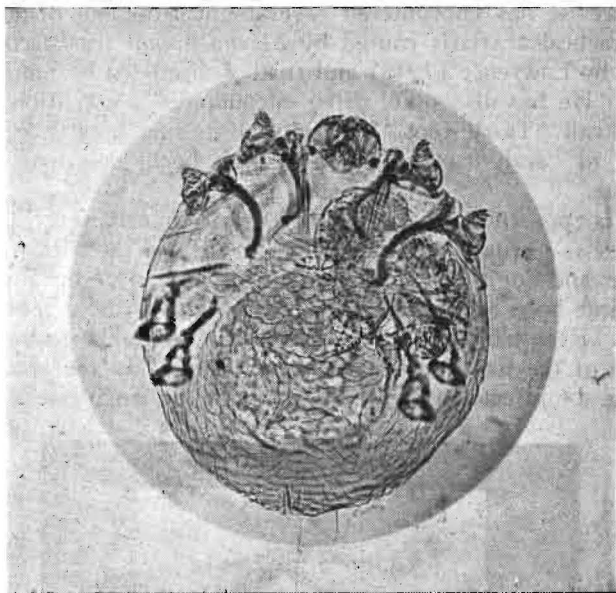
This is apparently quite a common disease in canaries. The disease commences as a crust which forms on the pads of the feet. The crusts get thicker and due to the great amount of flexion of the joints of the feet, the scab becomes divided up corresponding to the several segments of a digit. Eventually they develop into long tassle-like scabs extending the length of the toes and a half an inch thick. In the accompanying photo (fig. 1) infected feet are shown with a normal one in the inset



\* He has also encountered a feather-mite occurring on the plumage (1947). The "quill" mite has been found on one occasion, affecting the quill as does *Syringophilus bipectinatus* of the domestic fowl.

for comparison. These cases were not very advanced. In no instance though did the lesion extend up the leg. This is, of course, different from scaly leg in the fowl, where the lesion is confined to the skin of the shank and the pads of the feet are not infected.

In each case investigated a *Cnemidocoptes* was found present, often in large numbers. The mite resembles *C. mutans*, the cause of scaly-leg in the fowl, but the parasite looks smaller. A photo (fig. 2) shows a pregnant female. The detailed morphology was, however, not studied.



The disease may progress so far as to cause marked lameness and inability to walk and to perch comfortably, but no deaths can be ascribed directly to it.

In the differential diagnosis one may consider canary-pox, though this disease in its known forms is a very virulent infectious disease taking a systemic course.

The treatments used with good success have been removal of the "tassles" with a pair of scissors and the dipping of the feet twice weekly in paraffin (kerosene) or crude oil. The perches should also be treated. Sulphur ointments have not been successful.

It is believed that this is the first time that this condition has been described.

#### ACKNOWLEDGMENTS

Thanks are due to Dr. A. S. Canham for the references and to Mr. L. Hill for the photography.

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## VETERINARIAN HONOURED

On the occasion of the King's birthday, the list of honours included the high award of Companion of the Order of St. Michael and St. George to Dr. J. Carmichael, D.Sc., M.R.C.V.S., in his capacity as member of the Colonial Advisory Committee on Agriculture, Animal Health and Forestry.

Dr. Carmichael is head of the Veterinary Research Division of May & Baker, Ltd. The Pharmaceutical Society recently appointed him chairman of the Action and Uses Sub-committee in connection with the compilation of a British Veterinary Codex.

## GUIDE FOR THE PREPARATION OF SYNOPSES

(Being the guide drawn up by the Royal Society in 1949 and kindly supplied by the C.S.I.R. Library and Information Division.)

### 1. *Purpose.*

It is desirable that each paper be accompanied by a synopsis preferably appearing at the beginning. This synopsis is not part of the paper; it is intended to convey briefly the content of the paper, to draw attention to all new information and to the main conclusions. It should be factual.

### 2. *Style of Writing.*

The synopsis should be written concisely and in normal rather than abbreviated English. It is preferable to use the third person. Where possible use standard rather than proprietary terms, and avoid unnecessary contracting.

It should be presumed that the reader has some knowledge of the subject but has not read the paper. The synopsis should therefore be intelligible in itself without reference to the paper, for example it should not cite sections or illustrations by their numerical references in the text.

### 3. *Content.*

The title of the paper is usually read as part of the synopsis. The opening sentence should be framed accordingly and repetition of the title avoided. If the title is insufficiently comprehensive the opening should indicate the subjects covered. Usually the beginning of a synopsis should state the objective of the investigation.

It is sometimes valuable to indicate the treatment of the subject by such words as: brief, exhaustive, theoretical, etc.

The synopsis should indicate newly observed facts, conclusions of an experiment or argument and, if possible, the essential parts of any new theory, treatment, apparatus, technique, etc.

It should contain the names of any new compound, mineral species, etc., and any new numerical data, such as physical constants; if this is not possible it should draw attention to them. It is important to refer to new items and observations, even though some are incidental to the main purpose of the paper; such information may otherwise be hidden though it is often very useful.

When giving experimental results the synopsis should indicate the methods used; for new methods the basic principle, range of operation and degree of accuracy should be given.

### 4. *Detail of layout.*

It is impossible to recommend a standard length for a synopsis. It should, however, be concise and should not normally exceed 200 words.

If it is necessary to refer to earlier work in the summary, the reference should always be given in the same manner as in the text. Otherwise references should be left out.

When a synopsis is completed, the author is urged to revise it carefully, removing redundant words, clarifying obscurities and rectifying errors in copying from the paper. Particular attention should be paid by him to scientific and proper names, numerical data and chemical and mathematical formulæ.

**NOTE.**—It is suggested that this journal adopt the system of giving synopses at the beginning of articles.—Editor.

## BOOK REVIEWS

*TEXTBOOK OF MEAT INSPECTION*, by H. Thornton, B.V.Sc., M.R.C.V.S., D.V.M.; pp. xi + 659, with 257 illustrations. Published by Baillière, Tindall and Cox, London. Price 50s. net.

This book is undoubtedly a valuable addition to the library of veterinarians and others interested in abattoir work.

Most of the conditions are very fully and adequately dealt with, but I must draw attention to the following omissions.

1. Where tuberculosis is diagnosed in the routine examination of the bovine carcass, the possible examination of such inaccessible glands as the popliteals is referred to, but no mention is made of the ischiatic glands, which can be easily examined in the split carcass.

2. It is surprising that the very much better method of staining blood smears for anthrax with Giemsa is not advocated.

3. The fact that ripe proglotids of *Tænia saginata* may leave the human host spontaneously and not necessarily during defæcation is not stated, a fact which is of great significance in the control of the condition.

4. Oesophagostomiasis is very inadequately dealt with. A true understanding of the pathology and pathogenesis of this condition is of the greatest importance in making an evaluation.

5. Preisz-Nocord infection is not considered in making a routine examination of the sheep carcass, although admittedly this is a disease of special importance in countries like Australia and South Africa.

6. The more suitable designation for the condition described as "Osteohemachromatosis" probably is "congenital porphyria or porphyria-rinuria".

7. A chemical aid to diagnosis such as the simple phase test is much more practical than other elaborate tests to differentiate between bile and plant pigmentation.

I read the chapter on the bacteriology of meat with great interest and appreciation. This should urge those abattoir authorities who have not already done so, to organise a laboratory as an active unit in the abattoir organisation.

The author could perhaps have profitably included a discussion on the necessity of a histological examination of organs in certain cases. This is not only helpful in making an evaluation on good scientific grounds, but also stimulates research into a vast field for which ample material should be available at abattoirs.

The chapters on poultry and fish are valuable contributions and should stimulate veterinarians to take greater interest in these aspects of public health work.

P.J.J.F.

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*NUTRITION OF THE DOG*, by C. M. McCoy; pp. xiv — 338 with 47 figures. Second edition. Price 30s. net. Baillière, Tindall and Cox, London.

The second edition of this valuable book contains 197 pages more than the first edition published in 1946. A wealth of scientific data is given in eight new chapters. The literature cited is most comprehensive and would be appreciated by those who wish to gain more information.

Our knowledge of the nutritional requirements of dogs has largely been incidental, due to the fact that dogs were so commonly used as experimental animals for physiology. Professor McCoy is to be congratulated therefore for having collected the scattered data, which, with his own research and practical experience, is ably presented in this well-illustrated book.

It is felt that more space should have been given to applied nutrition such as the artificial feeding of puppies and other problems with which the veterinarian is often confronted. On the other hand the advice given for the carrying out of numerous feeding trials to demonstrate deficiencies might well have been omitted. Such work would be done to best advantage by students under trained direction.

This work will be a most useful reference book to veterinarians.

J.W.G.

## SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION

*Minutes of Council meeting held at 2.15 p.m. on June 8, 1950,  
in Prudential House, Pretoria.*

*Present:* J. H. Mason (President), A. M. Diesel (Vice-President), G. D. Sutton (Hon. Treasurer), R. Clark (Editor), S. W. J. van Rensburg, G. Pfaff, A. C. Kirkpatrick (Hon. Life Vice-President), H. P. Steyn, R. A. Alexander, C. J. van Heerden (Hon. Life Vice-President), W. D. Malherbe and M. de Lange (Honorary Secretary).

*Apologies:* P. J. du Toit, P. S. Snyman and A. D. Thomas.

1. *Minutes of meeting* held on April 20, 1950, read and adopted after some amendments.

2. *Arising from these minutes:*—

- (a) *P.D.S.A., Cape Town:* Letters addressed to a member and to P.D.S.A. read and approved by Council. After some discussion decided that Secretary obtain further information.
- (b) *P.D.S.A., Johannesburg:* President and Dr. Pfaff reported on negotiations with the Society to date. After considerable discussion decided that President and Dr. Pfaff were to have a further interview with the committee of this Society to explain Council's attitude on the employment of a veterinarian.
- (c) *Dog licences in the Province of Transvaal:* Letter from Provincial Secretary read. Council was in full agreement with the amended legislation.
- (d) *Amendments to Veterinary Act:* Dr. Pfaff reported on progress. Decided to hold a special meeting on Monday, 26th June, 1950, at 2.15 p.m. to discuss the proposed amendments. Secretary to circularise Dr. Pfaff's summary of replies received to members of Council, also to Dr. Parkin and some other interested members of the Association.
- (e) *Appointment of Lecturer by Pretoria University:* In spite of reminder sent no reply had been received from University Council. Secretary to send further reminder at end of June.
- (f) *Letter Dr. Pullinger re National Health Council:* In view of Dr. Snyman's absence matter held over for next meeting.
- (g) *Lengthening of B.V.Sc. Course:* Dr. Pfaff summarised the replies received to circular. After discussion decided that Council was not in agreement with extension of the length of the course for the purpose of teaching of Zootechnics, Dietetics and Animal Management. Council did feel that the course could be improved by making provision for six months' clinical experience away from Onderstepoort prior to graduation, e.g. with a private practitioner. Secretary to inform Faculty accordingly and to submit Dr. Pfaff's summary and all replies received to Faculty.
- (h) *International Veterinary Congress Fund:* Dr. Alexander reported on negotiations with Secretary for Agriculture.

3. *New Members:* The following have applied for membership:—  
L. Abrams, P. H. Bunton, D. J. de Waal, M. S. Dison, R. Every, J. M.



Huyser, G. J. Lay, A. Littlejohn, A. C. Maree, J. J. Oberholster, J. Robinson, R. A. Solomon, J. H. D. Snyman, K. M. van Heerden, C. J. Muller, W. L. S. Macintosh, H. C. Watson. Decided to recommend these candidates for election by next General Meeting.

4. *Resignations*: J. W. Rainey, G.V.O., Derby, Tasmania, tendered his resignation as he was now settled in Tasmania. Decided to recommend acceptance to General Meeting.

5. *General Meeting*: Decided that meetings should commence at 8.45 a.m. every morning. The business meeting to be held the last day from 11 a.m. to 1.0 p.m. and to include resolutions, if any.

The last afternoon to be left for continuation of business meeting, if necessary, and/or to enable visitors to consult the research staff in their respective laboratories.

6. *Refresher Courses*: Dr. Clark reported on poor response to date. Secretary reported that courses were again being publicised by means of a circular.

Dr. Alexander to approach Secretary for Agriculture on the question of leave for field officers to enable them to attend the refresher courses.

7. *Correspondence*:

- (a) *Invitation from S. Rhodesia Veterinary Association* to attend their Annual Meeting in Salisbury: Decided that a delegate could not be sent, and that S.A.V.M.A. should reciprocate by inviting Rhodesian colleagues to attend our General Meeting.
- (b) *G. Thompson, Cape Town*: Secretary reported on case of advertisement of "canine specialist". Correspondence between Registrar and Mr. Thompson read. Decided to request Secretary of Cape Western Branch to interview Mr. Thompson.
- (c) *Scales of Fees*: Lists submitted by Witwatersrand, Pretoria and Natal branches respectively to be passed on to Veterinary Board.
- (d) *Annual General Meeting, Natal Branch*: Agenda brought to notice of Council. Decided to ask Secretary, Natal branch, for summary of discussion on "Rôle of Private Practitioner in Control of Scheduled Diseases".

8. *General*:

(a) *Finance*:

- (i) Treasurer reported that £300 had been invested in Union Loan Certificates.
- (ii) Estate late Mrs. Footner: £220 had been received as final payment out of this estate and Treasurer proposed investing this money plus a further £80 (i.e. £300) in Union Loan Certificates. Approved.
- (iii) Dr. B. J. Brummer had qualified for life membership. President to send letter of congratulation.
- (b) Dr. Alexander reported on progress of negotiations with Central Meat Co-op. Society and S.A. Agricultural Union regarding proposed grant for reorganisation of veterinary education, research and field services.
- (c) P.D.S.A., Johannesburg: Decided to leave this matter in abeyance

until question of employment of veterinarian had been finally settled.

- (d) Delay in filling of vacancies of the posts of Director of Veterinary Services and Chairman of Veterinary Board: After discussion decided that President write to Minister, giving Council's views on the matter.

The meeting closed at 5.15 p.m.

J. H. MASON,  
*President, S.A.V.M.A.*

M. DE LANGE,  
*Hon. Secretary, S.A.V.M.A.*

## SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION

*Minutes of Council Meeting held at 2.15 p.m. on 10th August, 1950,  
in Prudential House, Pretoria.*

*Present:* J. H. Mason (President), A. M. Diesel (Vice-President), R. Clark (Editor), P. S. Snyman, G. D. Sutton (Hon. Treasurer), A. C. Kirkpatrick (Hon. Life Vice-President), J. G. Boswell, R. A. Alexander, P. J. du Toit (Hon. Life Vice-President), S. W. J. van Rensburg, W. D. Malherbe, A. D. Thomas, H. P. Steyn, M. de Lange (Hon. Secretary).

*Apology:* G. Pfaff.

1. Minutes of meeting held on June 8, 1950. Adopted.
2. *Arising from these minutes:—*
  - (a) *P.D.S.A., Cape Town:* Letter from Messrs. Ward & Boyes, attorneys, to P.D.S.A., and Secretary's reply read. As a member had evidently accepted the post, this matter could be regarded as closed. The other two applicants had been duly informed.
  - (b) *P.D.S.A., Johannesburg:* President reported that the P.D.S.A. committee was not prepared to meet members of this Council in the absence of the Superintendent.
  - (c) *Dog Licences:* Dr. Clark reported that he had received information that the licence fee for bitches in Transvaal, whether spayed or not, was to be £1-0-0 as from 1951. Secretary to make inquiries and to report to next meeting.
  - (d) *Amendments to Veterinary Act:* Minutes of Special meeting held on 26th June, 1950, to be circularised before next meeting of Council.
  - (e) *Appointment of Lecturer by Pretoria University:* Secretary reported on telephone conversation with Secretary of University Council. Matter to be held over for next meeting.
  - (f) *Letter Dr. Pullinger re National Health Council:* Dr. Snyman reported on contents of Dr. Pullinger's letter. Decided that Dr. Alexander discuss matters with Dr. Pullinger before the annual meeting of the National Health Council.
  - (g) *International Veterinary Congresses Fund:* Secretary reported on reply received from Secretary for Agriculture. Decided that the Association would contribute the full amount required for the year ending August, 1950, viz., £12-10-0.
  - (h) *Refresher Courses:* Dr. Alexander reported that the matter of leave for State veterinarians to attend the refresher courses had been satisfactorily settled with the Secretary for Agriculture.
  - (i) *S. Rhodesia Veterinary Association:* Decided to extend an invitation to any and all members of the above Association to attend the Annual Conference. Special invitation to be extended to Dr. D. A. Lawrence to contribute to the discussion on Infectious Sterility in Bovines.
  - (j) *Natal Branch:* Discussion on the "Rôle of the Private Prac-

tioner in the Control of Scheduled Diseases". Secretary to ask Natal Branch Secretary for a summary if and when this scheme has been formulated.

- (k) *Delay in Filling of Posts of D.V.S. and Chairman of Veterinary Board*: Correspondence between President and Minister read. Matter held over for next meeting.
- (l) *New Member*: Dr. H. Halenke has been proposed for membership by Dr. J. S. Watt. Decided to recommend the candidate for election by next General Meeting.

### 3. *Financial Matters*:

- (a) *Book Fund Prize*: Finance Committee recommended that the Prize Fund be increased from £250 to £500 by transfer from the reserve account, thereby increasing the annual student's prize from £10 to £20. Agreed.

Also recommended that Faculty be requested to regard only third and fourth-year students as eligible for the award. Agreed.

- (b) *Insurance*: Recommended that all insurance premiums in future be paid by stop-order, to reduce administrative work. Agreed.
- (c) *Subscriptions*: Recommended that, in view of increased costs of administration, etc., annual subscription be increased to £3-3-0 and life membership subscription to thirty-five guineas. Notice of motion of alterations to Rule 3(b) and 7(a) of Constitution to be sent out with agenda of General Meeting to all members.
- (d) *Student Loans*: Honorary Treasurer reported on progress with recovery of loans.
- (e) *Honorarium — Auditor*: Recommended by Finance Committee that honorarium of twenty-five guineas be paid to auditor for examination of books for the financial year 1949/50. Agreed.
- (f) *Presentation to former Registrar of Veterinarians*: Several members paid tribute to Mr. N. J. Cloete for the services he rendered to the veterinary profession during his period of office as Registrar. Decided to present Mr. Cloete with a cheque for twenty guineas on some suitable occasion.

### 4. *Correspondence*:

- (a) *Resignation*: Letter of resignation by S. Jackson read. Decided that Secretary write to Mr. Jackson, requesting him to reconsider his resignation, and to report to next Council meeting.
- (b) *Letter Secretary for Agriculture*, re expiry of term of office of Dr. A. M. Diesel as member of Veterinary Board: Secretary to call for nominations and to arrange ballot for new member.
- (c) *Letter Australian Veterinary Association* re their annual general meeting read.
- (d) *Letter S.P.C.A., Durban*, re appointment of part-time veterinarian read. Decided to refer to the Natal Branch and ask for details re past arrangements, etc. Report back to Council.
- (e) *Letter Dr. Frean*, re loss of Government property stolen from motor car read. After discussion decided to place the matter before the Pro-technical Section Committee of the Public Servants' Association.

(f) *Letter Director of Abattoirs, Cape Town*, re alteration of date of General Meeting. As all arrangements for the meeting have to be made considerably in advance, the date cannot be changed at this stage.

5. *General:*

- (a) *Guests to Dinner and Dance:* Decided to invite Dr. and Mrs. R. Bigalke and Prof. and Mrs. F. N. Bonsma to attend.  
(b) Next meeting of Council to be held on 24th August, 1950.

The meeting closed at 4.40 p.m.

J. H. MASON,  
*President, S.A.V.M.A.*

M. DE LANGE,  
*Hon. Secretary, S.A.V.M.A.*

## SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION

*Minutes of Special Council Meeting held at 2.15 p.m. on June 26, 1950,  
at Prudential House, Pretoria.*

*Present:* J. H. Mason (President), G. D. Sutton (Hon. Treasurer), A. M. Diesel (Vice-President), G. Pfaff, P. S. Snyman, R. Clark (Editor), W. D. Malherbe, S. W. J. van Rensburg, A. D. Thomas, H. P. Steyn, M. de Lange (Hon. Secretary), R. A. Alexander, C. J. van Heerden (Hon. Life Vice-President).

*Representatives Faculty of Veterinary Science:* B. S. Parkin, P. J. J. Fourie, T. F. Adelaar.

*Apology:* P. J. du Toit.

The President welcomed Faculty representatives and asked Dr. Pfaff to open discussion on the proposed amendments to the Veterinary Act. The following amendments were agreed to:—

### Section 1 (a): *Add:*

"If the Chairman is absent from any meeting, the members of the Board shall choose one of their number to act as Chairman.

If the Chairman has been granted leave of absence the members of the Board shall choose one of their number to act as Chairman until the Chairman resumes duty".

*Add:*

"If the Chairmanship falls vacant through the death or resignation of the Chairman or for any other reason, the remaining members shall elect a Chairman to act until the new Chairman is appointed in terms of Rule 1 (a)".

### Section 1 (c):

Five (instead of three) persons to be appointed by the S.A.V.M.A.

### Section 6 (i):

Amend so that the ordinary meetings of the Board shall be held at intervals of not more than six months, and special meetings whenever the Chairman of his own motion or upon the direction of three members of the Board, convenes a meeting.

Delete "with the consent of the Minister".

*Add:*

"All acts of the Board shall be decided by a majority of the votes of the members present at any meeting. The Chairman, in addition to his vote as a member, shall in the case of an equality of votes, have a casting vote".

### Section 8:

In the second line for "after considering any" substitute "on the".

In the fourth line between "after" and "examination" insert "a full course and".

### Section 9 (i) (a) and (b): *Add:*

"or South West Africa".

### Section 11 (ii):

For "Minister" substitute "Veterinary Board".

Section 16:

Delete "or under the Medical, Dental and Pharmacy Act, 1928, as a Medical Practitioner."

Section 17 (i) :

For the present sub-section, substitute the following:— "Any person not registered under this Act as a veterinarian, who pretends or by any means whatever holds himself out to be a veterinarian (whether or not purporting to be so registered) or uses the name of veterinarian or veterinary surgeon or any name, title, description or symbol indicating or calculated to lead persons to infer that he possesses a degree, diploma or other qualification as a veterinarian or veterinary surgeon or that he is registered under this Act, or who practises for gain as a veterinarian or veterinary surgeon or performs an act specially pertaining to the calling of a veterinarian or veterinary surgeon, shall be guilty of an offence and liable on conviction to a fine not exceeding fifty pounds; provided that nothing in this sub-section shall prohibit any person from performing any operations on or applying any treatment to his own animals or prohibit any bona fide farmer from castrating, dehorning, injecting or vaccinating any animal or docking any lamb."

Section 19:

Clarify this Section.

New Clauses:

Make provision to enable the Veterinary Board to specify by regulations or otherwise what is an operation or other work which may be performed by a person not registered under the Act.

Make provision to prevent any person [other than one mentioned in the amended Section 17 (i)], society, institution or other body, whether charitable or otherwise, to engage in veterinary activities except under the supervision of a veterinarian registered under this Act.

This Act shall be binding upon the Government of the Union of South Africa; and if necessary amend Section 11 (iii) by the deletion of the last six lines from "provided that such registration" to "shall lapse".

Make it a condition of registration that the applicant is resident in South Africa at the time of registration.

Give the Veterinary Board powers somewhat similar to those held by the Medical Council (Sections 25 and 26 of the Medical, Dental and Pharmacy Act, 1928) in connection with the registration of veterinary students, and veterinary education and examination in the Union. Rather than write the details into the Act it would be preferable to provide enabling authority by Regulations to govern this point if the occasion arises.

Provide for the appointment of Inspectors under the Act.

Apply the Act to South West Africa.

By definition or otherwise, restrict the use of the word "veterinary", e.g. prevent chemists terming themselves "veterinary chemists".

J. H. MASON,  
*President, S.A.V.M.A.*

M. DE LANGE,  
*Hon. Secretary, S.A.V.M.A.*

## WANTED URGENTLY

The Association requires copies of the following issues of the Journal: Vol. I: 3, Vol. V: 1, 2, 3 and 4, Vol. VII: 4, Vol. X: 2, Vol. XI: 2 and 4, Vol. XII: 1 and 3, Vol. XV: 1 and 3, Vol. XVI: 4, Vol. XVII: 3 and 4, Vol. XVIII: 1, Vol. XX: 1 and 2, Vol. XXI: 1.

Can any members oblige?

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## THE PRICE OF THE JOURNAL

Owing to increased costs the subscription price of this journal will be raised as from Volume XXII (1951). The new rate will be 15s. (S.A.) per annum. Back numbers will be obtainable at 5s. per number.



## OUR DUTY TO THE PROFESSION.

In his Presidential Address, Dr. Mason pointed out that the scope of modern veterinary science was exceedingly broad. It embraced all the activities having to do with the breeding, feeding and care of animals, with research work on the normal and on the sick animal, with methods of preventing and curing disease, with administration leading to the prevention or eradication of disease and with the production of clean milk and sound meat. No one of us can be expert in all these aspects of our profession, but we should all take some interest in all of them and each of us should be keenly interested in at least one of them.

Dr. Mason appealed for a practical approach to our problems and warned against the dangers of being side-tracked into purely academic investigation.

The goal of the veterinary profession is the building up of a healthy and productive animal population. Each one of us can best assist by becoming as proficient as possible in his own sphere and by assisting his profession as a whole.

Dr. Mason likened the attainments of veterinary science to a building which is ever being enlarged and improved, but the foundations of which remain the solid achievements of the past. The South African Veterinary Medical Association is the caretaker of the edifice and every member must assist to his utmost in that task.

# THE EPIZOOTIOLOGY OF NEWCASTLE DISEASE AND ITS CONTROL BY VACCINATION.

V. R. KASCHULA,

Regional Veterinary Laboratory, Cape Town.

(Paper read at the 45th Annual General Meeting.)

Although Newcastle Disease has received considerable attention from scientists in both the veterinary and medical fields its epizootiology still presents many unsolved problems.

In the short time I have at my disposal I regret that I am unable to review the fairly extensive literature on its epizootiology. As the subject is too large to cover completely I will limit myself largely to the possible rôle of wild birds in the dissemination of the disease. I will also give an account of some experiments we have been doing in this respect.

During the past year more than 250 outbreaks of Newcastle Disease involving a quarter of a million birds have been reported in the Western Province. Most of the bona fide poultry farmers conscientiously adopted all the recommended measures to avoid Newcastle Disease. When the Fort Dodge killed Vaccine became available, they used it readily, but there were some who were under the impression that if they were careful enough their birds would not get the disease and hence they did not vaccinate. Some of those owners consequently suffered disastrous losses, when their flocks became infected. It soon became evident that although it was necessary to continue all the hygienic precautions as part and parcel of good management, these alone were not sufficient to prevent the entry of Newcastle Disease. Of all the control measures, vaccination was the surest and the most direct safeguard.

The movement of all poultry was under the control of the Sub-Director of Veterinary Services, Cape West. In very few outbreaks could a satisfactory explanation for the introduction of the disease into the flock be found. The important question thus is: how did the disease get into these flocks?

In England<sup>1)</sup> and on the Continent the spread is mainly ascribed to the use of infected swill or traffic in stock and carcasses. De Lay and co-workers<sup>2)</sup> have shown in California that the virus can be borne on dust. This no doubt plays an important rôle once the disease gains entrance to a flock. Asplin has presented evidence that ducks, geese and rats<sup>3)</sup> and pigs<sup>4)</sup> may be inapparent carriers of the disease for short periods. In India, a pigeon has been shown to have contracted the disease naturally.<sup>4)</sup> Several human cases of conjunctivitis have been demonstrated by virus isolation.<sup>5, 6)</sup> All the gallinaceous birds (fowls, turkeys, guineafowls, quail and pheasants) are naturally susceptible, but a number of wild birds have been shown to develop the disease naturally. In the United States, Gillespie *et al*<sup>6)</sup> have found a natural case of Newcastle Disease in a nestling European starling (*Sturnus vulgaris*), a bird which is very common in the Union. Wilson<sup>7)</sup> was

able to recover the virus from the bone-marrow of a dead Gannet (*Sula bassana*) in the Orkneys. Similar birds are present along our coast. In the outbreak in Natal in 1945 we incriminated the movement of infected fowls as the means of spread.<sup>8)</sup> To-day we are convinced that there are important routes of dissemination other than the movement of infected poultry.

In the Cape many large poultry farmers considered themselves safe from the disease if the strictest precautions were taken. On one farm of 7,000 birds the disease broke out in a run right in the centre of the plant, the one most distant from the nearest neighbour. In another plant of 13,000 laying hens kept in batteries the disease started on the side farthest from the gate and spread very rapidly. Numerous red-mites were present in the thatched roof of the batteries and sparrows fed freely in the food troughs. On another farm of 2,000 hens the disease first broke out in the run farthest away from the house. The possibility of movements of infected stock on to these farms was eliminated. These and many other instances supported the idea that wild birds might have introduced the disease.

On a number of occasions the disease seems to have jumped great distances, appearing suddenly in an area distantly removed from known outbreaks and with no history of any known connection with other outbreaks. It was therefore decided to investigate the possible rôle that wild birds could play in the spread of the disease. In the Western Cape the open range system of husbandry is almost exclusively practised, largely because of the suitable soil and climatic conditions. Wild free flying birds are present on all these farms. The grain-eating birds, sparrows and doves, are often attracted in large numbers to the farms by the food put out for the fowls.

A number of these birds were caught and tamed for a month. They were then tested for susceptibility by injecting 0.1 cc. of chick embryo allantoic fluid infected with a local virulent Cape strain intramuscularly with the following results:

Species	No. Tested	Died	Course
1. Cape pheasant <i>Francolinus capensis</i>	2	2	4 and 7 days
2. Turtle dove (tortelduif, Ringdove) <i>Streptopelia Capicola</i>	6	0	apparently resistant
3. Laughing dove (Klein tortelduif, Lemoenduif) <i>Stigmatopelia senegalensis</i>	6	5	6, 6, 7, 7, 9 days. One survived
4. Cape Sparrow (Mossie) <i>Passer melanurus</i>	4	4	6, 9, 10, 17 days

Paralysis, especially of the legs, was seen in all birds that died.

The cause of death was established as being due to Newcastle Disease by the isolation and identification of the virus from the organs.

In the case of the doves and sparrows controls remained unaffected. No controls were available for the pheasants.

Having established that these birds were susceptible, it was decided to dose them *per os* and to study their excretions and their ability to carry the infection mechanically on their feet.

The feet of two sparrows were "tramped" forcibly into the naturally infected fowl faeces and virus was recovered from their feet one hour later. No doubt this period would have been considerably longer if further investigation had been done.

Groups of four each of the three species most intimately associated with fowls were then tested for ability to shed the virus in their excretions.

Sparrows (*Passer melanurus*), turtle doves (*Streptopelia capicola*) and laughing doves (*Stigmatopelia senegalensis*) were tested. Each bird received 0.1 cc. of chick embryo Allantoic fluid infected with the local virulent Cape strain *per os* and each group was placed in a previously sterilised cage. Each day they were transferred to another cage with a fresh supply of food and water. Saline suspensions of the pooled faeces samples were well mixed and centrifuged, and after treatment with penicillin and streptomycin according to Beaudette's<sup>9)</sup> recommendation, 0.2 cc. was injected into the allantoic sac of 8-12-day-old embryos. Six eggs were used for each test. Swabs were also taken from the mouth and throat of the doves. The saliva so collected was washed off in saline and treated with the antibiotics, but not centrifuged, and likewise injected into eggs.

The antibiotic technique worked well, very few non-specific deaths being experienced. A slide hæmagglutination test was done with the amnio-allantoic fluid of each embryo that died, and in many cases a full tube test of the alpha procedure of the U.S.B.A.I.<sup>10)</sup> was done, testing the amnio allantoic fluid against known positive and negative serums for specific diagnosis, especially in the case of the virus isolated from the organs of the birds.

The results of the faeces examination was as follows:—

Species	Days after dosage of recovery of N.D. virus from pooled faeces.																			
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
Turtle dove	—	—	—	—	—	—	+	—	—	—	—	—	—	—	—					
Laughing dove	+	+	+	—	+	+	—	—	—	—	—	—	—	—	—	—				
Sparrow	+	+	+	+	+	—	—	—	+	+	—	—	+	+	+	—	—	—	—	—
Days	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45	46	
	+	+	+	+	+	—	—	+	—	+	—	—	—	—	—	—	+	+	+	—

The result of the saliva examination was as follows:—

Species	Days after dosage of recovery of N.D. virus from pooled saliva.																	
	0-8 days								9	10	11	12	13	14	15	16	17	18
Turtle dove	No tests taken								-	-	-	-	-	-	-	-	-	-
Laughing dove									+	+	+	+	-	-	-	-	-	-
Sparrow	Not tested																	

+ Denotes positive test, — Negative test, o Bird died.

The sparrow and laughing dove which died on the 6th and 7th days respectively, were obviously ill for 24 hours before dying. These birds were listless and sat with ruffled feathers. The sparrow which died on the 15th day developed a progressive muscular inco-ordination from the 11th day onwards. This bird had a good appetite, but eventually was unable to stand, exhibiting giddiness. Its vent feathers were soiled by a greenish diarrhoea. The sparrow which died on the 19th day was listless and sat with ruffled feathers for three days prior to its death. It also had a diarrhoea. The fourth sparrow's faeces could unfortunately not be tested on the 21st to 27th days. On the 22nd day it developed a slight muscular inco-ordination. This became progressively worse and ataxia developed as well as a diarrhoea. The bird appeared stupid and abnormal, yet fed very well. Large quantities of virus were being passed in its faeces. The drunken state was at its worst on the 27th to 32nd days, while Newcastle virus was being shed in quantity. It lost condition, and died on the 46th day. The control birds remained in perfect health. In all the birds mentioned above that died, Newcastle Disease virus was isolated from the heart's blood, spleen and liver without any difficulty. There is no doubt that these birds developed Newcastle Disease from the oral dosage.

The three surviving laughing doves were later challenged with virulent virus and proved immune. Two controls injected at the same time both died. The turtle doves, together with controls, were also injected with virulent virus, but again none of them was affected.

Recently Kraneveld and Mansjoer<sup>11</sup> have done similar work in the Dutch East Indies—using two species of sparrows and two of doves. They dosed these birds with food contaminated with infected droppings. Although no active disease developed in the test birds their faeces contained the virus for three to five days as proved by dosing it to susceptible chickens.

Hofstad<sup>12</sup> has demonstrated that the mites (*Liponyssus sylviarum*) which had fed on infected fowls may harbour the virus. It is known that birds may spread these mites and I suggest that this is a further possible indirect means by which birds may spread the disease.

I would like to devote the remainder of my time to a discussion on results of vaccination in the Cape.

The imported Fort Dodge Killed Vaccine, of which more than  $\frac{3}{4}$  million doses have thus far been used in the Western Province, has had a very rigid field trial. The results on the whole have been very satisfactory. Several vaccinated flocks involving several thousand birds were affected by the disease and the lowered mortality was very striking. Normally the mortality was 60 - 90%, but in vaccinated flocks it was often less than 1%. There was, however, a severe drop in egg production lasting, in some cases, up to nine weeks. The vaccine is effective, but is not practical on a large scale over a long period, as the cost is prohibitive. The results of the large-scale field trial with live virus in Natal are now becoming available. From experimental evidence and reports from overseas, the immunity should be much more solid and durable and therefore this method will be far more suitable and practical.

Since May, 1950, we have been conducting experiments with locally prepared Beaudette's Roakin strain live virus on Robben Island. We first used it on a small field scale on the island, where 1,300 fowls in 49 flocks are kept. Newcastle disease had never occurred on the island. Half the fowls on the island were vaccinated in order to determine whether the other half would suffer from the use of live vaccine in the neighbourhood. After four months no ill effects have been reported and hæmagglutination inhibition tests done on a cross-section of these control birds proved negative. In one small flock of 28 fowls, 8 fowls were vaccinated and 20 kept as controls. They were all run together and after a month were tested by the H.I. test, when only the vaccinated birds showed titres. In yet another flock of 80 fowls there were two hens each with 15 one-week-old chicks. All the adult fowls were vaccinated, but not the two mothers nor the chicks. These had direct contact with the vaccinated fowls. Nothing happened to the chicks in spite of their being highly susceptible.

The only reaction of significance was the marked drop in egg production which started a week after vaccination, falling to as low as 20% of the original production. There was a gradual recovery, however, usually complete in four weeks. Six cases of inco-ordination and hypersensitivity were noticed, but these all recovered. The eight birds that died during the three weeks following vaccination were regarded as having died from other causes, but the vaccination may have hastened or contributed to their deaths.

Fowls vaccinated with the Roakin strain applied by the stick method have taken very ill, but in birds on which killed vaccine had previously been used there were no takes even  $3\frac{1}{2}$  months after vaccination, according to H.I. tests. However, when the virus was instilled intranasally, intraocularly or intratracheally good takes were recorded on such vaccinated birds.

Fowls vaccinated with Roakin strain and challenged with virulent Cape and Natal strains withstood the challenge completely.

## CONCLUSIONS

The virus of Newcastle Disease is essentially an avian one. Therefore it is not surprising to find that it affects a wide range of birds.

From an epizootiological point of view, I believe it is of significance that wild free-flying birds, which are so intimately associated with fowls, not only harbour and transport the virus on their feet, but that they can propagate and discharge the virus in quantity in their saliva and faeces over a long period. One sparrow was shown to be discharging the virus for 46 days after oral dosage. These birds may develop inapparent or subclinical infections, but a number will actually become frank cases. They may die, but some may eventually recover. It may be asked: If these birds are so susceptible, why are they not seen dead or sick in numbers? In Nature all birds must eventually die from natural or other causes, yet one seldom sees a dead bird. I have discussed this matter with ornithologists and they assure me that when birds feel "off colour" they go to cover and thus die in secluded places. The bodies of dead birds are soon disposed of in Nature, small rodents and ants playing an important rôle.

If these birds can contract the disease naturally as easily as they do in laboratory experiments, then they should be regarded as potentially extremely dangerous disseminators of the disease. Grain-eating birds are attracted by the food put out in the poultry runs, where they can sometimes be found in large numbers. It is often suggested that servants and visitors are likely to be important carriers. But if this is so then birds must be much more important, because they can be seen walking on the food or in the water and indeed even bathe in the drinking-water. Their droppings may be charged with virus. These birds have a motion every 10 to 15 minutes, which may easily be dropped on the food or in the water. Their flight from farm to farm is unrestricted. I feel that they should be regarded as far more dangerous than human beings. As far as I know the disease has so far broken out only under back-yard conditions, or on farms where the open range system was practised, but it did occur on two large battery plants where wild birds had direct access to the food containers. I therefore suggest that this be regarded as support for the belief that birds play an important rôle in the dissemination of the disease.

I believe that the solution to the problem is that farmers must practise an intensive system of management, not only for Newcastle Disease control, but for other poultry diseases as well. The chickens, however, must be allowed on the open range for the growing period of adolescence. As pointed out earlier, of all control measures, vaccination offers the surest safeguard against the disease. I therefore recommend that the young stock be vaccinated with live virus, before being put out onto open range, to ensure a solid and durable immunity. The quality of the immunity conferred by live virus vaccines is superior to that conferred by killed vaccines, though there is no doubt that these killed vaccines give a satisfactory protection. The Beaudette Roakin strain live virus has proved very successful and its use is to be highly recommended. It has proved as satisfactory or even more so under our conditions than it has in America. The danger of its becoming virulent appears to be non-existent, though recently in Natal it has shown ability to spread to an immediately adjoining pen,<sup>14)</sup> but this property has not been noticed in the fairly extensive observations

made in other field tests. It has also given more severe reactions in chickens suffering from some other disease such as coccidiosis. In general, however, it has proved safe to use and very satisfactory.

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#### DISCUSSION

During the ensuing discussion it was emphasized that no evidence was obtained during the 1945 outbreak in Natal or the recent outbreak on the Witwatersrand to indicate that wild birds played any appreciable part in spreading Newcastle Disease. Indeed, it was often observed on the Rand that the well-cared-for flocks belonging to Europeans escaped infection even when the younger birds were running on open range, while the fowls owned by Natives only a few hundred yards away were dying in large numbers.

During the outbreaks in Australia and Great Britain it would appear that birds were also not held responsible for any significant spread of the disease.

For some unknown reason birds of all ages injected in South Africa with locally produced vaccine seem to have reacted less severely to the American vaccine virus than similar fowls injected in the United States with vaccine prepared there.



## SOME VETERINARY PROBLEMS OF THE STATE VETERINARIAN IN NATAL.

L. L. DALY,  
Pietermaritzburg.

*(Paper read at the 45th Annual General Meeting.)*

The province of Natal, lapped by the Indian Ocean along its entire eastern boundary, is bounded in the north by the Usutu River, which forms the boundary with Portuguese East Africa, in the south by the Umtamvuma River and further inland by the Umzimkulu River, which mark the boundary with East Griqualand. The Drakensbergen in the west and north-west form the boundary between the province and Basutoland, Orange Free State and the Transvaal and due north the Ubombo Mountains form a common boundary with Swaziland from the Pongola River to the Usutu. Natal, including Zululand, covers an area of 33,650 sq. miles, a comparatively small area compared with the other provinces. Indeed, it is smaller than the area of the State Veterinarian of Calvinia! Physically, the land varies from the sandy coastal belt from which it rises rapidly in a confused succession of hills and ridges to form the greater part of the province which is largely middle veld, to the high-lying, almost highveld areas adjoining Basutoland, the Orange Free State and the Transvaal. The whole of the province lies in the high summer rainfall area and is probably the most closely settled and intensively farmed province in the Union.

Whatever Natal may lack in size, it certainly makes up for in the number of veterinary personnel employed there. The professional establishment, falling under the Sub-Director of the province, is composed of 12 State Veterinarians in 11 Veterinary divisions. One division requires the services of two veterinarians, viz. Vryheid. The lay establishment consists of 4 Supervision Stock Inspectors, 51 Stock Inspectors, 353 Assistant Stock Inspectors, 2 Meat Inspectors, 33 Native Constables and 713 Native Tank Assistants. This staff is larger than the combined staffs of all the other provinces. The Native tank assistants assist with the dipping in Native areas. They are employed by the Native Affairs Department, but are controlled by our Division. They afford an excellent example of a joint effort by the two Departments for the control of disease.

Veterinary Research is represented in the province by the long-established Allerton Laboratory, Pietermaritzburg, which deals mainly with poultry diseases at present, and Nagana Research Station at Masimba, which has been engaged so successfully for many years in tsetse fly research.

The reason for this large staff is not far to seek. As horse-sickness research was the principal factor in the establishment of

Onderstepoort, so East Coast Fever was responsible for the growth of the present veterinary field service. The danger of this disease, first introduced into the Union in 1902/03 and into Natal in 1906, where it spread rapidly as a result of the Zulu rebellion of that year, demanded close control, involving a permit system. Experience led to still closer control, until to-day we have an organisation capable of dealing with all epizootics except those which are air-borne. This was fully proved when foot and mouth disease was successfully checked after appearing in the Eastern Transvaal in 1937/39 and again in 1945 and at Nqutu in 1938. The latter outbreak, appearing suddenly in almost the centre of the province in a heavily populated area, was not allowed to spread further than the limit of the cattle movements which had taken place. Only the organisation which existed for controlling East Coast Fever could have made possible the successful outcome of those particular campaigns. Indeed, in my opinion, East Coast Fever experience and administration form the basis of a successful career of the veterinarian in the State Service. There were notifiable diseases prior to the advent of East Coast Fever, but none of them demanded the same amount of close control and attention to detail. The list of notifiable diseases has steadily grown in South Africa, and in Natal we have our fair share of such diseases to contend with. To deal with all the veterinary problems encountered in Natal in the space of this short paper would be an impossibility and I will therefore restrict myself to the most important.

#### 1. *East Coast Fever.*

The fact that this disease, first introduced into the province in 1906, is still with us, albeit with a much reduced incidence, is no reflection on the close control referred to above but rather, in my opinion, which is shared by other experienced East Coast Fever officers, to an unknown aetiological factor in this disease. Unfortunately, for many years the presence of Kochs bodies in a smear was taken to be East Coast Fever and the outbreak treated as such, but it is only from the late thirties that a differentiation was made and the relationship of Kochs bodies to Tzaneen disease understood. Thus prior to 1940 our statistics indicate a number of East Coast Fever outbreaks which, in the light of our present knowledge, were not entirely East Coast Fever *per se*, but that disease plus Tzaneen disease. This lack of differentiation may explain to a large extent the reason why many outbreaks were cleared up entirely by dipping and quarantine measures whereas in many others the disease recurred repeatedly despite the most exacting quarantine control and short-interval dipping. In far too many recurrences after a period of four to six years were there cattle from the previous outbreak to shake off the uneasy suspicion that more cattle, particularly calves, recover from this disease than we realise and that usually, after a period of four to six years, the immunity is broken down and a recurrence of the disease occurs. The fact that our research workers, under laboratory conditions, have not succeeded in breaking down the immunity in East Coast Fever does not convince me that this does not take place under natural conditions. Numerous instances of

repeated recurrences in the same areas in Natal can be cited, but two will suffice:—

- (a) In tank area No. 394 along the Umkomaas River in the Polela district, records show repeated outbreaks going back to 1916 and recurrences every four to six years up to 1943, when a very heavy mortality occurred. The mortality was stopped by short-interval dipping and rigid quarantine restrictions. The quarantine was not raised after the usual period but was maintained energetically, but despite this a further recurrence occurred five years later. That in spite of the fact that no cattle were allowed into or through the area.
- (b) In the Pietermaritzburg district a bad outbreak of the disease occurred on a farm called Binchester Grange in 1943. There was a heavy mortality, but, eventually, with short-interval dipping, it ceased. The farm was opened, after the usual precautions, in 1945. The farm changed hands and the new owner held a dispersal sale. The following outbreaks of East Coast Fever occurred in the Pietermaritzburg district: Allandale 10.3.47, Tyrone 4.5.48, Notuli 24.2.49 and Springvalley May 1949—all farms some distance apart and between which no cattle movements had occurred. There was, however, one common factor. Each of these farms had cattle which came from Binchester Grange and which had survived the heavy infection of 1943. It is noteworthy that up to the present no recurrence has occurred on Binchester Grange nor are there any cattle from the previous outbreak of 1943 on that farm. Time does not allow of further examples being cited, but from an experience of 26 years in the Transvaal and Natal, I am convinced that the recovered animal is a danger and that, under certain conditions, possibly in association with an intermediary animal, can set up the disease on that farm. Only research can settle that point. It is on account of this danger of recurrence of East Coast Fever that the slaughter-out policy is favoured to-day.

It would be entirely incorrect to say that dipping and strict quarantine measures have not been attended with success. From the widespreak incidence of the disease after the Zulu rebellion in 1906, when practically every district in Natal was infected, to the beginning of 1930, a great deal must have been accomplished and at this time 29 districts were free of the disease. A comparison of the position at the end of the Annual Report years 1929, 1939 and 1949 is interesting:—

	1929	1939	1949
No. of outbreaks .....	58	11	10
No. of districts infected .....	16	5	2

For the period 1940 to 1949 outbreaks involved 23 districts out of the 45 magisterial districts in Natal. At the end of 1949 only two districts in the whole of Natal were infected, viz. Vryheid and Pietermaritzburg. At 30th of June, 1950, two further districts had become

infected, viz. Babanango and Polela, both as a result of recurrences on previously infected areas.

To attain this highly satisfactory position the slaughter policy has played a very important part and, in the last remaining foci it appears to be the most effective and quickest method of finally ridding this province of the disease. This policy is not new, having been employed spasmodically as far back as 1928 in Natal. We all know that the Transvaal was speedily cleared by this method, and as the policy appears to have been adopted here on a voluntary basis so far, it is earnestly hoped that East Coast Fever will be completely eradicated within the next three years. At the present time, all infected farms in Natal, with the exception of a recently infected farm (one case so far) in Pietermaritzburg district, have been cleared by slaughter, and only one tank area in a Native location remains infected. It is noteworthy that this tank area is the only one in the whole of the Native territories in Natal still to have the disease.

The following are a few examples of the successful results obtained by applying the slaughter policy:—

<i>District.</i>	<i>No. of outbreaks slaughtered.</i>	<i>Date of slaughter.</i>	<i>Result.</i>
Kranskop . . . . .	5	1928—1929	One further outbreak in 1940 which was slaughtered out. Since no further outbreak.
Lions River . . . . .	5	1935	One further outbreak with one death only in another part of the district. Since all clear.
Richmond . . . . .	5	1929—1931	No further outbreaks in area. One further outbreak in another part of district in 1938. This was slaughtered. Since which no further outbreak.
Vryheid . . . . .	23	1942—1943	E.C.F. recently re- curred on four of these 23 farms, but in each case it was re-intro- duced.

At the 1949 Annual Congress of the Natal Agricultural Union the following resolution was unanimously adopted:—

“This Congress, in view of the fact that after 40 years of treatment East Coast Fever has not been eradicated, urgently requests the Government to investigate the possibility of effecting total eradication by means of a slaughter policy such as has been successfully demonstrated in Portuguese East Africa and the Transvaal Lowveld.”

So far the policy has been on a voluntary basis and it is hoped that the Minister of Agriculture will make it compulsory. The owner

is allowed to select one of the following methods of compensation:—

- (a) The *gross* realisation of his cattle at the abattoirs plus the values allowed under the schedule of the Diseases of Stock Act for East Coast Fever; railage, commission and abattoir charges being borne by the Department;
- (b) an agreed valuation on the farm. This requires the State Veterinarian to estimate the slaughter value of the herd, using the values allowed under the Act, to come to an agreed valuation with the owner. This is the method usually preferred;
- (c) the owner may accept the valuation of the State Veterinarian or elect to have a valuation made by a Board in terms of paragraph 2 of Government Notice 752 of 8.5.31. This procedure was only once adopted in 1949 in Natal and only because the herd was a high-grade dairy herd.

The application of the slaughter policy to 13 farms, nine in Vryheid and four in Pietermaritzburg, in 1948/49 resulted in the removal of 3,376 head of cattle to abattoirs at Durban and the cost to the State amounted to £14,932 14s. 6d., which includes rail charges to an amount of £2,504 15s. 11d., i.e. an average of £4 9s. 1d. per head.

Some interesting figures are revealed in the clearing of the nine farms in the Vryheid district in 1949/50. The cattle were removed by road motor service to Vryheid and thence by rail to Durban abattoirs, after temperaturing at dawn, dipping, hand-dressing and branding. It may be mentioned that, in all cases, loading banks were constructed or improvised on the infected farms, roads repaired to bear these heavy 10-ton vehicles and in some cases temporary roads had to be constructed.

No. of farms cleared .....	9
No. of cattle evacuated .....	3056
Average number of cattle per load .....	22.24 head
Average distance of farms to station .....	34.33 miles
Total compensation paid to owners .....	£23,223 8 0
Average compensation per animal .....	£7 12 0
Total cost to State .....	£7,078 0 10
Average cost to State per beast .....	£2 6 4
Total railage costs .....	£1,706 16 8
Total road motor transport costs .....	£1,041 4 1
Average cost per mile per beast by R.M.T. ....	2.38 pence
Railage per beast averaged .....	£0 11 2
R.M.T. costs per beast averaged .....	£0 6 10

The cattle population of Natal at June, 1950, was approximately 2,676,945, 1,229,973 being in Native areas and 1,446,972 in non-Native areas. There were 374,202 recorded deaths during the year, for which 368,866 smears were submitted and examined. Of these, 32,120 or 8.8% were undiagnosable compared with 33,864 or 8.7% in the

previous year. The number of smears examined during the past nine years averaged 406,611 with a total of 3,659,710, as follows:—

1941	.....	426,530
1942	.....	387,363
1943	.....	431,069
1944	.....	396,578
1945	.....	415,052
1946	.....	478,099
1947	.....	367,904
1948	.....	388,248
1949	.....	368,866

The number of privately owned dipping tanks at June, 1950, amounted to 6,701 and Native area tanks to 679, a total of 7,380 or roughly a dipping tank to every  $4\frac{1}{2}$  sq. miles. When it is remembered that the great majority of the cattle in Natal are dipped and checked under supervision every 14 days and smears collected and submitted expeditiously, it will be realised why the large lay staff, mentioned at the beginning of this paper, is so necessary.

The smear examination is carried out by the field division. Each State Veterinarian has his own smear examining centre. The routine examination is done at each centre by two lay smear examiners with the veterinary officer performing frequent surprise check examinations. In addition frequent cross checks are carried out between smear centres. Selected cartons of smears, with the diagnoses are sent by one veterinary officer to another in exchange for a similar batch from the latter's station and a comparison made of the respective diagnoses by the veterinary officers concerned. It is surprising how efficient the majority of these lay examiners have proved themselves to be. They are not only responsible for the staining and examination of smears, but also the marking, recording and despatch of diagnoses to the parties concerned. Each station deals with an average of 3,000 to 3,500 smears per month.

The legal machinery enabling the officers of this division to carry out the duties assigned to them is contained in the Diseases of Stock Act No. 14 of 1911 together with Government Notice No. 638 of 1915 and sundry other Government Notices. The most important Government Notice is 1782 of 1941. This notice requires an owner to keep a register of his stock and record all births and deaths, cattle movements, etc., and to collect his stock on an appointed day at a given time. I firmly believe that this notice has been a great factor in the successful campaign against East Coast Fever. Before leaving East Coast Fever, I must pay tribute to two of my predecessors in Natal, viz., Col. C. J. van Heerden and Dr. A. M. Diesel. Both these officers laid the foundations and established our present organisation against organised opposition and amidst bitter feelings, but they stood their ground and gradually won over the opposition, and it is in recent years that their labours have borne fruit and resulted in the happy position we find in Natal to-day.

## 2. *Theileriosis.*

(a) *Small Piro.* In Natal particular attention is paid to smears containing small piroplasms by themselves or associated with *P. bigem*, *B. bovis* or anaplasms. Usually the farms from which these smears come are *not* quarantined, but kept under close supervision and observation. These cases are generally referred to as Group 1. For the year ending June, 1950, there were 22 cases of small piroplasms, 36 cases associated with piroplasmosis and three cases associated with anaplasmosis; a total of 6- cases in various districts throughout the province.

(b) *Kochs Bodies or Tzaneen Disease.* This condition is fairly prevalent in Natal and is usually referred to as Group II. It occurs in various districts and seems to have no relationship to old or recent East Coast Fever outbreaks. It often occurs in districts which have been free of the latter disease for as many as 30 years. In some years the incidence is greater in some districts than in others, e.g., there were numerous cases in Eshowe in 1946/47 and at present there is a heavy incidence in the Port Shepstone and Greytown divisions. For the year ending June, 1950, 29 districts were affected involving 130 areas with 197 cases, the greatest incidence at present being in the Umvoti, New Hanover and Port Shepstone districts. The condition is usually found in young calves, which have died as a result of some debilitating factor, e.g. scours, paratyphoid, etc., or in cattle slaughtered at abattoirs. *Theileria mutans* appears to be ubiquitous in Natal and, in my opinion, all local cattle acquire a natural immunity. The cases we find on smear examination are merely coincidental.

However, these cases do occasion a great deal of work in that, on account of the possibility of East Coast Fever, each case is thoroughly investigated and the farm is quarantined for a period of one to three months depending on the particular circumstances, including the smear history.

(c) *Suspected East Coast Fever.* Very occasionally a smear may reveal Koch's bodies — macro- end micro-schizonts — associated with small piro, and it is impossible to differentiate from East Coast Fever. The case may occur in an area remote from any known East Coast Fever outbreak, the history of the case may not support an East Coast Fever diagnosis nor cattle movements indicate that possibility. In such cases, the smear history of that farm is closely considered and a quarantine of six to nine months imposed, depending on all factors, and that farm kept under a very close observation pending developments.

Koch's body cases appear to be on the increase in the province and this increase may be due to the substitution of B.H.C. dips for arsenical dips.

## 3. *Redwater and Gallsickness.*

The number of cases of these two diseases seems to remain fairly consistent, as the following figures indicate:—

<i>Year.</i>	<i>Redwater and Gallsickness cases.</i>
1941/42 .....	6307
1942/43 .....	6516
1943/44 .....	5571
1944/45 .....	7330
1945/46 .....	6408
1946/47 .....	5770
1947/48 .....	5022
1948/49 .....	5709
1949/50 .....	7486

The spread of the arsenic-resistant tick does not appear to have influenced the incidence of these diseases to any extent. The cases for the year 1949/50 reported by Veterinary Divisions reveal an interesting position:—

<i>Division.</i>	<i>Redwater.</i>	<i>Gallsickness</i>
Dundee .....	173	97
Durban .....	156	96
Estcourt .....	253	281
Greytown .....	408	327
Ixopo .....	205	435
Ladysmith .....	115	152
Nongoma .....	102	51
Maritzburg .....	117	170
Port Shepstone .....	179	391
Vryheid .....	189	33
Eshowe .....	3280	276

The heavy incidence in the Eshowe Division is difficult to explain. The figures for that Division for the previous year were 1,544 Redwater cases and 278 Gallsickness cases. The State Veterinarian at Eshowe reports as follows:—

“There appears to be a definite correlation between Redwater and very wet spells. Oxen especially are affected, although their condition might not be bad.

On one farm Native boys were put with the cattle, and animals showing the slightest signs of being sick were treated with Pirevan. On this farm only about 25% of treated animals recovered. The total losses on this farm amounted to 126 head from Redwater, starting in the wet months and reaching a peak in February. Most cases were *peracute*, the animals were sick for only 4-8 hours, very often they were not even suspected of being sick.”

Personally, I consider that this problem merits special investigation by the Research Division and, on the whole question of these two diseases, would appeal to the Director of Veterinary Services to make the combined Redwater and Gallsickness vaccine available again.

#### 4. *Nagana.*

Dr. Rene du Toit is to give you an address on the present position in regard to this disease, and when you have heard this, I think you



will agree with me that the Division of Veterinary Services might well be proud of the success which has been attained in Zululand. The campaign has been spectacular and unique in that this is the first occasion that D.D.T. spraying from aeroplanes plus dipping of cattle in D.D.T. have been used in Africa in an attempt to eradicate the disease. The Research section has played the major rôle in this campaign. Paradoxically, that section carried out one of the biggest field operations of recent years as research. The field section was associated in this work by seconding Dr. Kluge from the field together with one stock inspector and eight assistant stock inspectors for full-time work on Nagana and placing the veterinary officers of Vryheid, Nongoma and Eshowe and their staffs at the disposal of the officer-in-charge of operations.

Although the chief vector in the spread of epizootic Nagana, viz. *Glossina pallidipes*, has been virtually eradicated, the complete eradication of Nagana from Zululand is, under present conditions, impossible. *Glossina austeni*, occurring in the thick bush along False Bay, the Mkuzi and Pongola Rivers, is unassailable on account of the impenetrable bush, and *Glossina brevipalpis*, even if vulnerable, would always occur along the upper reaches of the Pongola River due to infiltration from the southern area of Portuguese East Africa in the vicinity of the Usutu River and its junction with the Pongola. This means, in effect, that Nagana in Zululand must, in future, be looked upon purely as a local disease such as Redwater, Heartwater, etc. To avoid the disease, stock owners must avoid the thick bush known to harbour *Glossina austeni* or *Glossina brevipalpis*, but should they be unfortunate, they have recourse to treatment with M. & B. 1553, which proved so successful during the heavy incidence of Nagana, or with Antricyde. In other words, it *can* be prevented but, failing this, is *can* be *cured*.

It is impossible to give accurate figures, but it is estimated that 60,000 to 70,000 head of cattle died as a result of Nagana during the epizootic of 1946/1947. No wonder the matter became one of national importance and a million pounds voted to combat the disease. That money has been well spent and Zululand is to-day the rancher's paradise. Whether the removal of nature's sentinel in that area, viz., the tsetse fly, is to be a good thing for Zululand will depend entirely on good farming practice and veld management. My own fear is that the favourable conditions in that country will lead to overstocking with all its deleterious effects.

The following were confirmed cases of Nagana for the year ending June, 1950:—

<i>District.</i>	<i>Cases.</i>	<i>Last Case.</i>
Ingwavuma ... ..	9	26. 8.49
Hlabisa ... ..	12	28.10.49
Mahlabatini ... ..	1	9.12.49
Ngotshe ... ..	23	12. 5.50
Nongoma ... ..	6	28.10.49
Ubonbo ... ..	2	12. 8.49

—  
53  
—

Dipping in D.D.T. emulsion was carried out in the whole of the Nagana area and community dipping arranged for in 47 dipping tanks in Zululand and 39 in the Ngotshe district at a cost of £25,000 per annum. It is impossible to assess the value of this dipping in the eradication of *Glossina pallidipes*, but it must have been a contributory factor as it controlled other blood-sucking flies in unsprayed areas. The Nagana position has improved so materially that dipping in D.D.T. dip supplied by the State is now being carried out only in the tanks surrounding the Hluhluwe Game Reserve, pending the complete eradication of *Glossina pallidipes* and *Glossina brevipalpis* from that area.

#### 5. *Anthrax*.

For the year ending June, 1950, there were 23 outbreaks of this disease involving 13 districts. There were 29 deaths. The previous year there were 43 outbreaks with 137 deaths.

The block inoculation of cattle in Zululand has not been carried out for some years, on grounds of economy, but despite this there were only two outbreaks of Anthrax with two deaths this year and two outbreaks with three deaths last year.

#### 6. *Sheep Scab*.

After a lapse of many years, dating back to the simultaneous compulsory dipping of 1929, Northern Natal became infected with this disease in 1948 and again in 1949. Dipping in B.H.C. in most cases was used for the first time to control the outbreaks and two dippings proved very effective.

The following outbreaks occurred:—

<i>District.</i>	<i>1948/49.</i>	<i>1949/50.</i>
Newcastle .....	1	—
Paulpietersburg .....	5	2
Utrecht .....	18	4
Vryheid .....	3	—
Helpmekaar .....	—	2
	27	8

At present the area is free from the disease.

#### 7. *Mange*.

This disease was encountered to the following extent during the past year:—

One outbreak in cattle, four in equines, four in goats and one in pigs. B.H.C. dipping proved effective in all cases.

#### 8. *Infectious Equine Anaemia*.

Two cases of infectious equine anæmia in mules were confirmed by Onderstepoort during the year. They occurred on the Natal Estates at Mt. Edgecombe in the Inanda district. The origin could not be

traced, but as the sugar estates generally obtain their mules from outside the province, it is likely that a reservoir of infection may be found in the mule-breeding areas of the Western Free State or North-Western Cape.

#### 9. *Dourine.*

Up to the end of 1948, Natal was considered free from Dourine, but in February, 1949, a suspicious case in a stallion was confirmed in the Utrecht district. Two tests on this farm revealed 22 positive out of 52 equines. Two nearby farms were also found to be infected. More recently a case was discovered in the Greytown district in an expensive polo pony mare intended for export to Rhodesia. At present Natal appears to be relatively free of this disease, but I think that compulsory testing will reveal many more cases, particularly as a large number of horses are being brought into the Province from outside for trading with Natives.

#### 10. *Tuberculosis.*

Very little progress in the eradication of T.B. can be reported. At present there are only 14 accredited herds in Natal involving 3,081 cattle. Five of these herds are Government owned, one Provincial administration and eight private owners. The largest herd is the Baynesfield Estate with 1,162 head. Under the interim scheme very few tests have been conducted and, as yet, no herds have become accredited. Until a premium is put on milk from a T.B. herd, I am afraid very little progress will be made in the control of this disease.

#### 11. *Horesickness and Bluetongue.*

The past summer produced a very heavy mortality in respect of these two diseases. Statistics in the case of Bluetongue are not available, but in Horesickness there were 136 reported deaths in immunised equines and 1,804 reported deaths in unimmunised equines. The figures for the preceding year were 40 and 479 respectively. Two outbreaks of Foot and Mouth Disease were reported, which on investigation proved to be Bluetongue in cattle moved from the drought-stricken Orange Free State to Northern Natal following good rains in the latter area.

#### 12. *Infectious Sterility.*

This condition presents a major problem in Natal at present. An intensive survey has revealed four foci of infection and one highly suspicious focus. Of these, two are gradually being slaughtered out, but the others are carrying on. The baffling problem on one of these farms is the non-development of clinical symptoms in the bulls, although the parent herd in the Transvaal was found to be heavily infected and the cows on this farm showed typical symptoms. On one of the others, the two infected bulls were destroyed and replaced by two healthy bulls. After a period of roughly 10 months these bulls still show no clinical symptoms. The suspicious outbreak cannot be confirmed, because the bull shows no lesions. The calving rate on these three farms appears to be normal. All these farms are in quarantine and the question is

how long can they be kept in quarantine? Finality in regard to a policy must be arrived at.

Infectious sterility has focused attention on another problem which is probably more widespread in Natal than we imagine and which is possibly as serious as Epivag. I refer to *Trichomoniasis*. Recently this condition has been diagnosed in two herds in Natal and I am convinced that an intensive survey throughout the dairy herds here will reveal many more cases. In the case of one herd the owner estimates a sterility rate of roughly 20%. The economic loss in this herd over a period of nine years is put at £5,000. Whether these figures will be borne out on close scrutiny only further research will tell.

### 13. *Newcastle Disease.*

Dr. Kaschula has dealt very fully with the epizootology of this disease and this has evoked a very interesting discussion, so I will not weary you by giving you more than a brief history of the outbreaks on the Natal coast. The disease appeared for the first time in South Africa in the Durban Division in May, 1945. We tried to combat the infection by quarantine measures and a permit system. Whether by good luck or good management, the disease ended when the last focus at Lamontville was slaughtered out on 7.1.46. All remained quiet and no further signs of the disease were seen until the State Veterinarian diagnosed it again at Mt. Edgecombe in the Inanda district, in December, 1949. The outbreak was soon surrounded and all fowls penned and we thought we had isolated it. So slaughter-out was resorted to in May, 1950. Fourteen days after this was completed, the disease made its appearance in the Springvale-Avoca areas of the same district, followed soon after by outbreaks at Verulam. It appeared at Riverside in the Durban district north of the Umgeni on 20.6.50 and at Chakas Kraal in the Lower Tugela district in August, 1950. Widespread outbreaks followed during this month of August south of the Umgeni and at present this disease extends from the Umgeni River southwards through the Durban Borough and Greater Durban, including Clairwood, Wentworth, Jacobs down to Umzinto and as far south as Sezela.

The Inanda district offered an excellent opportunity of experimenting with the live virus, as the outbreaks there occurred in pockets, surrounded by sugar cane, and, therefore, practically isolated. The decision to do so was made and the vaccination campaign commenced on 11.7.50 and was completed by the 12.8.50. This experiment will form the subject of an article in the *Journal* giving all details. Suffice to say, here, that 90,245 birds were vaccinated from day-old chicks to adult birds. It was not possible to get mortality figures for the whole area, but the figures available, based on 45,129 birds, gave percentage mortality figures as follows:—

*A. Class:* Four months to adult, .84%; *B. Class:* One month to four months, 1.6%; *C. Class:* Day-old to one month, 5.6%. Average percentage mortality over 45,129 birds = 1.86%.

These figures are sufficiently reassuring to allow of the use of live vaccine, if not generally, then at least in those districts endangered

by the disease. At present a large-scale vaccination campaign is being carried out from the Umgeni River southwards through Durban, Umlazi, Pinetown, Umzinto districts down to Sezela.

I would like to place on record my sincere appreciation of the excellent work done by Dr. Zwarenstein and his staff in the Inanda experiment, for his meticulous recording of data and congratulate him on the successful outcome of that experiment.

#### 14. *Miscellaneous.*

Time does not permit of any detailed discussion of the following conditions and I merely want to draw attention to the fact that they do occur in Natal:—

##### *In Sheep.*

- (a) Domsiekte has been reported from the Vryheid district.
- (b) Enterotoxaemia is fairly prevalent in the sheep areas of the province. A suggestion has been made by certain veterinarians that this condition has followed dosing of sheep with Phenothiazine. In some cases this appears to be borne out, but the condition is also found where Phenothiazine has not been used.
- (c) Lumpy wool has been encountered in the areas where sheep were dipper for scab in 1948 and 1949. Whether this condition was caused by the use of Benzine Hexachloride or not will be determined by experiment.

##### *In Pigs.*

- (a) Oedema of the Bowel — This condition has been diagnosed in the Natal Midlands and Southern Natal and appears to be on the increase. The ætiology is obscure, but the indications are that it is dietetic in origin.
- (b) Spirochaetosis — is prevalent throughout the province and while the mortality is negligible, the nuisance value is great.
- (c) A new worm has been identified — *Stephanurus dentatus* — from two farms in the Highflats area. It is known as the kidney worm and occurs in the perirenal fat, pelvis of kidney and walls of the ureters and rarely in other organs.

The pig industry is important, not only in Natal, and at the last meeting of the Natal Branch of the S.A.V.M.A. a resolution was passed asking the Director to go into the question of more research in pig diseases. I trust that this will receive his attention.

##### *In Poultry.*

In the Durban district two poultry plants were found to be infected with gape worm — *Syngamus trachea*. This is, as far as I am aware, the first occasion this worm has been found in South Africa.

Fowl typhoid is fairly prevalent. Allerton supplied 148,000 doses in Natal last year.

##### *In Cattle.*

- (a) Paratyphoid occurs generally. 30,000 doses of vaccine were supplied in Natal last year.

- (b) *Epidural abscesses*. This condition appears from time to time especially in Southern Natal. Material and cases have been sent to Onderstepoort, but we do not seem to have made much progress in establishing the ætiology of the disease.
- (c) *Heartwater*. Occurs in the Port Shepstone division, the thornveld of Klip River and, on a much larger scale, in Zululand and portions of the Vryheid Veterinary Division. The incidence of this disease rose steeply in Zululand when thousands of cattle were introduced there after Nagana had subsided.
- (d) *Sweating Sickness* in calves is very prevalent in Zululand.
- (e) *Verminosis* in cattle, particularly calves, is a common condition throughout the province and deserves more propaganda amongst the farmers by our veterinarians.

In conclusion, I must say that the lesson I have learnt in Natal is that the better the farming practice the less disease one encounters. This would appear obvious, but it goes further than that. It throws a responsibility on the shoulders of every one of us to do all in our power to persuade farmers to study veld management and to establish pastures. As guardians of the health and well-being of stock, we are vitally concerned with soil fertility and what that soil produces for the feeding of stock. Just as the youth in the city slums is susceptible to all kinds of diseases through malnutrition and bad housing, so our stock cannot reach the optimum under conditions which lead to malnutrition, i.e., overstocking and maintenance on poor, deficient grasses. Ill-nourished stock must be susceptible to disease. For those who are interested in this aspect, I cannot do better than recommend two books by Louis Bromfield, viz., *Pleasant Valley* and *Malabor Farm*.

# THE TREATMENT OF "GID" IN SHEEP.

S. W. DE VILLIERS,  
De Aar.

*(Paper read at the 45th Annual General Meeting.)*

I am pleased to be able to record my experience and observations in connection with the treatment of "Gid" or *Coenurus Cerebralis* in sheep.

Whereas in the past we have had to deal with individual cases as a rule — and I take it that this has been the experience of most veterinarians — at the Smartt Syndicate, Britstown district, the mortality figures for this disease have risen to over 200 so far, and fresh cases are still being encountered almost daily.

*History:* The Smartt Syndicate consists of eight farms comprising some 40,000 morgen of ground, on which there are approximately 15,000 sheep. There are 15 camps each holding about 1,000 sheep. In addition there are numerous camps in which lucerne is grown. During the drought, i.e. during the months of October, November and December of 1949 and January of 1950, approximately 3,000 ewes and lambs grazed on these "lucerne lands," and it is amongst these sheep that the outbreak took place.

*Source of Infection:* This must primarily be attributed to the large number of dogs on the syndicate. During the past two months or so, 45 dogs were shot of which ownership was unclaimed. In addition there are 20 belonging to members of the European staff and approximately 50 belonging to the labourers on the farm. Some 130 sheep are slaughtered monthly at the Syndicate, and the heads and entrails of these sheep are sold to the labourers. If these sheep harbour the bladder worm cysts and the dogs have access to them, one may well imagine how easily the segments and eggs of *T. multiceps* may be spread. In addition, the „draaijakkals" may also have been a contributory factor, as no less than 22 were shot on the farm during the past two months. There are 16 labourers watering the lucerne lands, and as each has a dog or two, access to these lands by the dogs is of daily occurrence.

*Treatment:* As we all know, the skull undergoes pressure atrophy even to the extent of perforation, and on examination the affected part can be localised, as it yields to pressure. In other words, there is a thinning of the frontal or parietal bones, usually the latter. This occurs at the site of the bladder worm cysts. Small oval or rounded cavities in the bone are formed covered by a thin, almost transparent, membrane. Here it is of importance to note that this occurs only in sheep from 12 months old or less up to 18 months old. So far I have not been able to detect this phenomenon in sheep of 4-tooth or over.

The wool over the region of the frontal and parietal bones is clipped as short as possible. The bones are palpated and a spot is located which easily yields to pressure. Absolute alcohol is applied to the site and a sterilised needle is inserted. It requires but very little pressure, as a thin membranous covering only has to be pierced. As soon as the needle penetrates the cyst, the clear fluid contained in the sac spurts out. This fluid may now be aspirated by means of a syringe, but it has certain disadvantages. A vacuum forms and causes discomfort and struggling on the part of the sheep and displacement of the needle during the aspiration process. It is better to allow the fluid to be evacuated by gravitation. The sheep remains motionless and no injury to the brain is likely to be caused. Evacuation is facilitated if the sheep is placed on a table and the head turned so that the cranium faces downwards. At first the fluid runs freely, but when drop by drop exudes, the head is revolved so that the head of the needle faces directly upwards. As soon as the head is turned and the head of the needle faces directly downwards, the fluid again runs freely. This is repeated until froth or bubbles are observed in the last remaining drops. The drainage is then complete. The needle is withdrawn and the site again disinfected.

Of the 63 sheep treated so far, 39 (31 merinos and 8 karakuls) have apparently fully recovered. They were operated upon during the period 5th to 13th July and were examined by me on the 17th October. A period of three months has elapsed and during that time they have been grazing normally and would appear to be perfectly normal in every respect. Of the remainder, 12 have died and a further 12 are still under observation. Of these latter, symptoms of Gid have recurred and further cysts had to be drained.

On the 16th September, six sheep were treated and, after drainage, 1 cc. of a solution of 1/1000 acriflavine was injected into the cyst cavity. To date none has shown any ill-effects.

The following observations are of interest:—

1. Amount of fluid extracted:

<i>No. of Sheep.</i>	<i>Amount extracted in cc.</i>
3	4 – 10
11	11 – 20
16	21 – 30
18	31 – 40
8	41 – 50
4	51 – 60
2	61 – 65
1	67

2. Sheep that have one cyst only and from which perfectly clear fluid is drained invariably recover.

3. If one or other of the small arteries or veins are struck and the fluid which emerges is slightly bloodstained, such sheep do not recover.



4. Sheep, although drained twice from separate cysts, stand a good chance of recovery if the fluid in both cases is perfectly clear as it emerges.

5. Of the sheep treated by forcible introduction of the needle, i.e. 4-tooth and over, none has recovered. Hæmorrhages of the brain have been observed on post mortem.

6. Sheep that recover commence to graze normally after about the second day.

7. Sheep that were prostrate or down for some considerable time, regain their feet immediately after the operation and remain on their feet.

8. For every one sheep of over 4-tooth that was found to be infected, approximately 20 of 2-tooth and under were involved.

As it was of considerable interest to determine the fate of the lining of the cyst and the scolices after drainage, specimens of two brains of treated sheep were submitted to Onderstepoort for examination.

*Specimen No. 1:* Sheep treated 7.7.1950. Selected as apparently normal and slaughtered 26.9.1950 — period 2 $\frac{3}{4}$  months.

The report was as follows:

"The cyst assumed by me to have been drained was partially collapsed and contained jelly-like material (it may have been fluid and set after the formalin fixation). The cyst wall was thickened and wrinkled. The scolices were all everted and appeared healthy according to a report by Dr. Ortlepp after a study under the dissecting microscope."

*Specimen No. 2:* Sheep treated 5.7.1950. Selected as apparently normal, and slaughtered 17.10.1950 — period 3 $\frac{1}{2}$  months.

Report:

"The space occupied by the cyst was filled with greenish caseous pus, in the centre of which remnants of the cyst wall and scoleces could be identified macroscopically. In both cases there was evidence of an overlying localised encephalitis and meningitis. The drained cysts have not yet been examined histopathologically."

From the above, I think it may be concluded that once the cyst is collapsed, it does not refill again and although there is a localised encephalitis and meningitis, it apparently does not affect the health of the sheep.

I wish to express my indebtedness to Drs. de Boom and Ortlepp for examination of the specimens of brains submitted.

# THE MANAGEMENT OF DRUGFAST BILIARY FEVER IN DOGS.

W. D. MALHERBE,  
Onderstepoort.

(*Resumé of paper presented at 45th General Meeting of the S.A.V.M.A., 1950.*)

Drug treatment of *B. canis* infection in dogs is usually very satisfactory. This paper, however, offers some observations on the problem of drugfastness as encountered from time to time in practice.

The literature of biliary fever contains very few references to this aspect. Lourie and Yorke<sup>1)</sup> and Fulton and Yorke<sup>2)</sup>, working with stilbamidine, produced drugfastness in a single pup merely by underdosage, and found that serial passage through 42 dogs and puppies left the parasite still unresponsive to stilbamidine. Dickson<sup>3)</sup> in this country has recorded his observations on drugfastness and tried the antimalarial drug "paludrine" with inconclusive results.

Experience at Onderstepoort has shown that in the course of time with intensive treatment of biliary fever in a circumscribed area, a degree of resistance has developed to any drug constantly in use. Trypan blue, highly efficient from about 1914, gave in the third decade of the century a relapse and drugresistance rate of up to 50%.<sup>4)</sup> Acaprin in 1935 was hailed as a great improvement, but in about four years relapses and resistance became increasingly troublesome<sup>4)</sup> even in dosage of up to 4½ times the recommended figure. Phenamidine was introduced in about 1939 and has at Onderstepoort been found to be very satisfactory, perhaps because underdosage is consciously avoided by weighing all dogs before treatment and using doses slightly higher than those recommended by the manufacturers. Cases of persistence of parasites after treatment do, however, give trouble periodically. In the treatment of such cases the following principles are observed:—

1. *Kennelling*, which is considered highly desirable in order to restrict movement (thus avoiding all exertion) and to facilitate complete supervision.

2. *Thorough clinical examination*, for the purpose of eliminating where possible concurrent infections (e.g. rickettsiosis or ankylostomiasis) and organic disease (e.g. constipation or nephritis).

3. The use of *high dosage of phenamidine*, which sometimes suffices to bring about recovery.

4. The use of *trypan blue and the flavines* as reserve drugs.

5. The use of *arsenicals* as supportive treatment. These have been found to be exceedingly useful even though they are not regarded as specifics. Courses of novarsenobillon (N.A.B.) intravenously are used as a routine in these cases with good results.

6. *Avoidance of drug therapy.* Cases, even still showing fever and parasites but preserving good habitus, are, subject to daily or twice daily checking, left without drug treatment. This is a useful procedure when other methods have failed to clear the parasites, but a degree of somatic resistance has been built up.

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# OFFICIAL APPROVAL FOR THE TESTING OF STOCK AND ITS CERTIFICATION OF FREEDOM FROM SCHEDULED DISEASES.

A. M. DIESEL,

Deputy Director of Veterinary Services (Field Services).

*(Paper read at the 45th Annual General Meeting.)*

## INTRODUCTION

The testing of stock, which are susceptible to the proclaimed diseases, has frequently to be undertaken for reasons such as the following:—

- (1) To fulfil the requirements of importing countries.
- (2) To satisfy buyers, owners and custodians of stock within the Union.
- (3) To arrive at a diagnosis in respect of stock in ill-health.
- (4) To give effect to control and eradication measures conducted or sponsored by the Department of Agriculture.

For the purpose of this paper, testing means the application of approved methods of detecting any scheduled disease, including the use of diagnostic agents, the application of accepted laboratory tests and of recognised methods of clinical examinations.

The paper includes a general reference to the certification of certain related products of animal origin.

The examination of animal products and of associated foodstuffs is a consideration on its own. It is hoped that it will form the subject of an early address by one of our members, particularly as the trade concerned with the handling and preparation of these products is rapidly expanding.

## THE LEGAL BACKGROUND

- (a) *The relative Sections of the Diseases of Stock Act (Act No. 14 of 1911) as amended.*

The Department through its Veterinary Division is responsible for the administration of this Act. In doing so it must naturally remain within the provisions and spirit of the Act, and the Regulations, Proclamations, Prohibitions and Orders made under it.

According to the provisions of Section 9(1) of the principal Act, as amended by Act No. 5 of 1930, the removal of stock infected with a proclaimed disease can only be effected in accordance with the permissions laid down by the Act, its Regulations and Orders.

Under Section 22 of the Principal Act, as amended by Act No. 5 of 1930, it is not sufficient for a person charged with failing to report

the existence of a notifiable disease, to prove that he had no knowledge of the disease unless he proves in addition that he took all reasonable steps to discover its presence.

These two Sections assume that when an owner is in doubt as to the existence of a scheduled disease among his stock, that he will consult a veterinarian in order that he may remain within the provision of the law. Section 9(2) (Act 5 of 1930) requires him to report any fact or suspicion of disease to an officer of the Department or a member of the South African Police. These officers in turn are required to transmit their reports to the Minister. The Minister, if satisfied that a notifiable disease exists, then causes the area to be declared infected by a written notice served on the owner of the land by the State Veterinarian.

The responsibility of reporting the existence of scheduled diseases thus rests with the owner of the stock.

The State Veterinarian alone is concerned with the testing of stock by the following provisions:—

- (1) The Principal Veterinary Officer under Sections 4 and 5 of the Act (as amended by Act No. 25 of 1916) has the right to cause cattle to be tested for tuberculosis and stock generally for the presence of suspected diseases, at the ports of entry where they are presented for the purpose of being introduced into the Union. This he may do before or at the time he grants a permit for its introduction.
- (2) By Section 10 of the Act (as amended by Act No. 31 of 1923) the State Veterinarian is authorised to destroy stock visibly infected with Glanders, Tuberculosis and Lungsickness, and to test the contact stock for the presence of these diseases.
- (3) Section 17 of the Act (as amended by Act No. 17 of 1940) gives power of entry onto any land, premises, vessel or vehicle by persons generally or specially authorised by the Minister, so that they may test stock for the existence of scheduled diseases. These persons are generally officers of the Department.

When the State is conducting large-scale campaigns against serious epidemics, it must be in the best interests of all concerned, for the State Veterinarians alone to be responsible for the final diagnosis of these diseases. The Division of Veterinary Services engages itself particularly in this regard against East Coast Fever, Sheep Scab, Foot and Mouth Disease, Nagana, Anthrax, Lumpy Skin Disease, Newcastle Disease, Infectious Epididymitis and Vaginitis of Cattle.

This does not mean that Private or Municipal Veterinarians are necessarily precluded, in the ordinary course of their veterinary work, from making a diagnosis.

It becomes embarrassing, however, when the diagnosis of the disease by the State is in conflict with that made by another Veterinarian, and accordingly, communicated to the owner.

Thus it is, in respect of the two diseases, Glanders and Tuberculosis, which can be diagnosed through the use of diagnostic agents, that under the regulations promulgated by Government Notice No. 638

of 1915, control over these diagnostic agents and to some extent over tests themselves is given to the Principal Veterinary Officer.

It is to be appreciated, too, that the Diseases of Stock Act often arranges for compensation to be paid to owners in respect of diseased stock destroyed under its provisions.

Where the Division of Veterinary Services sponsors voluntary schemes for the control of notifiable diseases, the diagnosis would necessarily be undertaken by any Veterinarian recognised under the scheme. The Division has voluntary schemes for the control of Bovine Tuberculosis and of B.W.D. and Fowl Typhoid. A scheme for the control of Dourine existed before the last war, but has hitherto not been re-instituted. A scheme for the control of Contagious Abortion is receiving consideration at present.

(b) *Connecting links under the Public Health Act (Act No. 36 of 1919) as amended.*

The following Sections of this Act link up the control between the Departments of Health and Agriculture:—

(1) *Section 4. (Act No. 57 of 1935).*

Permits the Director of Veterinary Services to be a member of the Council of Public Health.

(2) *Section 6.*

The Minister of Health may require local authorities to furnish reports and returns regarding any matter relating to public health. He may, therefore, require local authorities to report cases of animal diseases discovered by Municipal Health Officers, if such animal diseases are capable of affecting the health of humans.

(3) *Section 18.*

Anthrax, Glanders and Rabies are among the diseases declared notifiable by this Section. Under it the Minister may declare any disease to be notifiable.

(4) *Section 36.*

According to this Section the Minister of Health may make regulations for preventing the spread from any animal or animal carcase or product to man, of Rabies, Glanders, Anthrax, Tuberculosis or any other disease communicable by any animals or animal carcasses or products, to man. He can, therefore, make a regulation requiring local authorities to report the discovery of any such diseases.

(5) *Section 39.*

Under this Section any person who becomes aware of any unusual sickness or mortality among rats, mice, cats, dogs or other animals susceptible to plague or other formidable epidemics, must immediately report the fact to the local authority.

(6) *Section 114.*

By this Section local authorities must report the discovery of

Tuberculosis-infected milk and dairy products, to the Chief Health Officer and the Principal Veterinary Officer. (Incidentally under the Tuberculosis Regulations of the Diseases of Stock Act, promulgated under Government Notice No. 638 of 1915, anyone who discovers the presence of tubercle bacilli in milk, must report this fact without delay to the Principal Veterinary Officer.)

(7) *Section 115.*

Under this Section the Minister of Health may make regulations for the inspection, examination, detention, slaughtering, etc., of diseased animals and articles of food for human consumption.

The Meat Inspection Regulations promulgated under Government Notice No. 2118 of 1924, as amended, are made under this authority. The "approved" Veterinarian recognised in this authority is defined as one approved by the Principal Veterinary Officer. The Principal Veterinary Officer (Director of Veterinary Services) must thus approve all Veterinarians employed on full-time or part-time meat inspection duty.

It is well here to remind part-time Municipally employed Veterinarians who have not yet been given this authority that they should apply for it to the office of the Director of Veterinary Services, Box 806, Pretoria.

(8) *Section 137.*

Under this Section reciprocal notification and consultation is arranged between the Departments of Health and Agriculture, in respect of diseases affecting both man and animals.

- (9) Lastly may I, in passing, direct attention to the provisions of Government Notice No. 739 of 14th May, 1937, whereby the importation and manufacture of Rabies vaccine is prohibited except with the approval of the Minister of Health.

#### THE TESTING OF STOCK FOR EXPORT

All countries concerned with preventing the introduction of formidable stock diseases lay down strict requirements in regard to the introduction of stock and products from other countries.

Many countries demand official certificates from the appropriate authorities of the country of origin, indicating freedom of that country or set portions of it, in respect of particular periods of time, from certain named diseases, such as Foot and Mouth Disease, Contagious Bovine Pleuro-pneumonia, Rinderpest, Rabies, Sheep Pox, etc. Most countries require certificates indicating that the stock itself, which is to be imported, has been examined and found free from disease.

Some countries will accept health certificates from persons approved by the authority of the country of origin. Many require the names and specimen signatures of all the persons so authorised.

Other countries prefer to issue Import Permits in advance and to stipulate thereon the certificates which are to accompany the stock.

Some countries even prefer to transmit such permits to the exporter, via the office of the Director of Veterinary Services.

It would take too long in a paper of this kind to give a complete set of requirements of all the countries to which stock (and pets) proceed from the Union. The office of the Director of Veterinary Services, Box 806, Pretoria, and of the Provincial Sub-Directors is in possession of much of this information and it can be had on application.

I may just mention that cattle proceeding to Southern Rhodesia will henceforth be consigned to Bulawayo in the first instance. There they will on arrival be tested for T.B. and otherwise examined. Any owners who wish the application of the tuberculin test for their own edification prior to export, must arrange for this to be done at least 30 days before the cattle are due to arrive at Bulawayo. Should cattle for some reason or another have been tested within 30 days this fact must be disclosed on the Certificate of Health.

The Director of Veterinary Services will consider applications for the return of the cattle to the Union, the introduction of which has been refused at Bulawayo. A standard form of certificate U.A.D. 932 for completion by Veterinarians generally, and countersignature by the State Veterinarian or Provincial Sub-Director of Veterinary Services, is available for use.

#### CERTIFICATION OF PRODUCTS OF ANIMAL ORIGIN FOR PURPOSES OF EXPORT

These certificates are invariably required by the importing country, from the State organization of the country of origin. So it is with countries like Canada, Egypt, the United Kingdom, the United States of America, Australia and others.

In regard to hides, skins and wool the certificates are required to state that the country of origin is free from Foot and Mouth Disease, Rinderpest and other serious epizootics; that Anthrax is not prevalent and that the hides, skins and wool originated in the country from which they are exported.

Certificates in respect of meat and meat food products, including canned foods, must indicate that they were derived from animals which received ante- and post-mortem Veterinary inspection, are sound and wholesome, fit for human consumption and have been handled in a sanitary manner.

The abattoirs at which animals are slaughtered in the Union, the meat products of which are to be exported, are largely under Municipal Veterinary control. It is natural that the Directors of these Municipal Abattoirs should wish to sign the required certificate. Counter-signature by the Director of Veterinary Services is indicated in order to give official recognition and satisfaction, particularly regarding the scheduled diseases.

The whole question of examination and certification of meat and meat food products for export is one which is presently receiving the attention of the Department and of the Livestock and Meat Industries Control Board. A meeting of interested persons is due to take place shortly.



PERMISSION TO APPLY *the* MALLEIN AND DOURINE  
AND THE TUBERCULIN TESTS

Written permission is given by the Director of Veterinary Services, Box 806, Pretoria, to each Veterinarian who is to be authorised by him to undertake these tests. The letter of authority sets out the position in full and explains the reasons for the authorization. Attached to the letter of authority are standard forms for use in regard to the application of the Mallein and Dourine tests to equines intended for export. Specimen forms of the indemnity concerned with the destruction of equines which have reacted positively to the Dourine test and concerning the branding of reactors to the Tuberculin test are also attached.

Veterinarians who are not in receipt of this letter of authority from the Principal Veterinary Officer should apply for it to the Director of Veterinary Services, Box 806, Pretoria, in order to ensure that Mallein, Tuberculin and the Dourine test results will be available to them when they make their requests to the Director of Veterinary Services, Onderstepoort. You will note, too, that serum sent to Onderstepoort for the Contagious Abortion and Dourine tests is now to be paid for at the rate of 6d. per test.

# SCIENCE AND THE CONSERVATION OF WILD LIFE IN SOUTH AFRICA.

R. BIGALKE,

Director of the National Zoological Gardens.

*(Paper read at the 45th Annual General Meeting.)*

Before proceeding with the subject of this paper, I wish to thank the President cordially for his invitation to address this conference of veterinarians.

The topic that I have selected may appear to be a strange one to some of you. You may feel that it is a case of talking to the converted, when an assembly of veterinarians is addressed on the necessity of applying science to the vast field of wild life conservation. But an association of more than twenty years with conservation work in the Union has repeatedly shown me that the biologist's standpoint is by no means taken for granted. Although it is axiomatic to us that wild life conservation must fail if it is divorced from scientific research, many people in the Union are still opposed or indifferent to the rôle of science in this important work. In South Africa it is still widely believed that the only people with a knowledge of the fauna are those who have repeatedly shot everything ranging from a sparrow to an elephant.

To be able to discuss this topic, it is necessary to know how wild life conservation is organised in the Union.

Section 85(x) of the South Africa Act empowers Provincial Councils to make ordinances for fish and game preservation, and by Union Government Proclamation No. 212 of 1935 these limited powers were extended to "the preservation of flora and fauna". But wild life conservation is also undertaken by the Central Government through the National Parks Act (No. 56 of 1926), and Divisional Councils and Municipalities also play their part.

Contrary to the general belief, restrictions on the shooting of game animals were imposed at an early date in the history of South Africa. In 1656, that is to say only four years after Van Riebeeck landed at the Cape, the Governor placed restrictions on the shooting of game. Two official hunters were engaged by the East India Company in order to provide the garrison at the Cape with venison. Other persons could only shoot such animals for which bounties were paid, that is to say animals like lions, leopards and hyenas.<sup>8)</sup> The rewards paid in the year 1656 were 25s. for a lion, 16s. 8d. for a hyena and 12s. 6d. for a leopard. In the following year these rewards were increased to 34s. 9d., 27s. 9d. and 13s. 10d. respectively.<sup>8)</sup> It is interesting to note that at present a reward of £5 is paid for a leopard by the Cape Provincial Administration.

During Commander Borghorst's administration in the year 1669,

licences were first issued to the burgers to shoot game wherever they wished to do so.<sup>8)</sup>

In 1742 the shooting of zebras was prohibited subject to a fine of £10.<sup>7)</sup> In the year 1753 further restrictions were imposed when the shooting of big game, except carnivorous animals, was prohibited.<sup>7)</sup> It must be pointed out, however, that the preservation of game animals in South Africa has always suffered from the difficulty of effective enforcement of the laws owing to the great distances. It is still widely believed in the Union that game is nobody's property and that it may, therefore, be destroyed at any time and place.

For about two and a half centuries after the founding of the settlement at the Cape, the preservation of the fauna was attempted by the negative method of trying to curb its destruction. But towards the end of the nineteenth century it began to be realised that protective legislation was not sufficient and that special areas would have to be set aside in which the fauna (and flora) could enjoy complete protection.

Conservation of wild life implies a great deal more than the merely negative preservation from destruction that is implied by protection. Conservation necessitates the deliberate control of wild life and its environment, where necessary, for the "wise use of man," but there should always be as little human interference as possible. To achieve the aims of conservation, be it in the field of wild life conservation or of other natural resources, it is essential to enlist the aid of both research and education. In the United States the necessity for using science as a tool in the service of conservation was realised as far back as 1910. There it is now generally accepted that protective legislation alone, even when effectively enforced, can achieve no more than to retard the forces of destruction.<sup>3)</sup>

The first game reserve established in South Africa appears to have been the Pongola Game Reserve, which came into existence on the 13th June, 1894, and was abolished on the 12th January, 1921. This Transvaal game reserve lay in the narrow strip of territory formed by the Pongola River, the southern border of Swaziland, and the Lebombo Mountains. It was followed by the establishment on the 22nd April, 1897, of four game reserves in Zululand, two of which, the Hluhluwe Game Reserve and the Umfolosi Game Reserve, are still in existence. On the 30th March, 1898, President Kruger established the Sabi Game Reserve, which now is the southern part of the Kruger National Park in the eastern Transvaal.

Apart from 61 Forest Nature Reserves with a total area of 12,906.7 acres, the following national parks and nature reserves exist in the Union :—

National Parks (inclusive of two "Provincial"	
national parks in Natal) .....	
	7
Game Reserves {	Provincial Game Reserves .....
	16
	Municipal Game Reserves .....
	6
	Nature Reserves .....
	6
Divisional Council Nature Reserves .....	3
<hr/>	
Total .....	38
<hr/>	

As a nation we have, therefore, no reason to be ashamed of the provisions we have made in the form of national parks and nature reserves for the fauna and flora of our country. But in the sphere of the administration of those areas, a great deal remains to be done in two respects, namely the scientific and educational aspects of conservation. Only in cases in which the presence of wild life has clashed with the interests of farmers in the Union has the aid of science hitherto been invoked. Thus diseases like nagana, snotsiekte, swine fever and foot and mouth disease have met with considerable attention, because they are considered to be threats to the farmer's herds of domesticated animals. We must not lose sight of the fact, however, that it is by no means always the case that wild animals are the sources of infection of stock in the case of diseases common to both. There is evidence to show that frequently the converse is true. It is only necessary to remind you of the incidence of tuberculosis in kudus of the Grahamstown bushveld. In this case it is almost certain that the infection came from cattle.<sup>9)</sup>

It is, of course, the veterinarian's principal task to provide for the welfare of the farmer's stock, but I feel that sufficient cognisance has not always been taken of the zoologist's standpoint, and that research from the zoological aspect of the problems has frequently been almost entirely lacking. On the other hand, I realise that research into these difficult problems takes time. When the politicians press for quick results, the veterinarian is in a quandary and may be forced to resort to immediate measures of which he himself does not approve. All biologists hope that the steps that are now being taken in Zululand will solve the nagana problem in Natal for all time. In cases in which the interests of the country's wild life clash with those of domesticated animals, I would like to urge strongly that in future zoologists, botanists and ecologists should have a proper place in the organisations set up to deal with the problems.

The national parks concept originated in the United States of America, where the first national park, namely the Yellowstone National Park, was established on the 1st March, 1872. It is not surprising, therefore, that there is no part of the world in which all aspects of national administration, particularly the scientific and educational aspects, have met with so much attention as in the United States. A special service to administer the American national parks, namely the National Park Service, was created as far back as 1916. Canada has naturally been influenced by the United States in its methods of dealing with national parks.

Britain is one of the most recent converts to the national parks concept. During the past four years much attention has been paid to this matter in Britain, both by interested organisations and by Government Commissions and Committees. This has culminated in the passing of the "National Parks and Access to the Countryside Act, 1949". Britain is a highly industrialised and highly developed country "where almost every acre of land is used in some degree for the economic needs of man and has its place in the complex design of agricultural, industrial and residential use".<sup>5)</sup> In Britain there are no

vast expanses of virgin territory such as we find in Africa. For these reasons the need for national parks in Britain is urgent, and for these reasons, also, it has been decided in Britain that wild life research shall play its part from the very inception of national parks. In a report entitled "Conservation of Nature in England and Wales" we read that "the essential foundations of an effective conservation policy are research and education. The starting-point must be found in a competent Biological Service working on a sound long-term programme of research into the fundamental factors affecting wild life". "It is often forgotten," the report says, "that neither the special provisions nor the general measures can be effectively put into operation without the most detailed and expert scientific action. There is abundant evidence (i.e. in Britain) to demonstrate the ill-effects on wild life of inexpert and unco-ordinated local action, however well intentioned." "Scientific conservation," the report says, "is animated by an outlook completely divorced from the unthinking sentiment which so frequently brings about the destruction of the very organisms it seeks to protect. A conservation policy directed to maintaining any particular biological equilibrium entails constant vigilance and a fine-scale 'management' of a kind comparable to the most highly developed farming".<sup>4)</sup> In the British national parks established farming use will be maintained.

A Nature Conservancy has now been constituted in Britain with the following functions:—

- (a) To provide scientific advice on the conservation and control of the natural flora and fauna of Great Britain.
- (b) To establish, maintain and manage nature reserves in Great Britain, including the maintenance of physical features of scientific interest.
- (c) To organise and develop the research and scientific services related thereto.

The wild life problems in South Africa are legion, and this field of research is almost entirely untouched. There is not a single national park or reserve in the Union of South Africa in which the most elementary requirements, namely taxonomic surveys of the fauna and flora, have met with attention. Such surveys are essential preliminaries to the investigation of other scientific problems and are indispensable prerequisites for the educational work in such parks.<sup>2)</sup> They are also necessary in order to determine the natural areas and boundaries of national parks and nature reserves. The National Parks Board of Trustees and other controlling bodies can hardly be expected to engage taxonomists for this work, but it should be possible to make mutually satisfactory arrangements with some of the country's museums and the Department of Botany for the required surveys. Surveys of the geology, the history and the pre-history of our national parks and reserves are also required, but these are not so urgent.

It has already been stated that human interference in areas where wild life is preserved should be limited to the absolute minimum. But control measures may become necessary from time to time. It is essential, however, that no control should be exercised until a detailed

investigation by biologists has shown that such a measure is necessary.

In South Africa biological considerations have seldom played any part in determining the boundaries of national parks and reserves — in fact the fixing of these boundaries has generally been a matter of expediency. The result is that most of these will have to be provided with artificial barriers at some time. The late Dr. Austin Roberts drew attention to this as far back as 1935.<sup>2, 6)</sup>

The introduction of exotic species of animals and plants into a national park or a reserve is anathema to biologists.<sup>2)</sup> Yet instances are not lacking in South Africa in which proposals have been made to stock national parks and reserves with non-indigenous species. Scientific control of our national parks and reserves is the best guarantee against the adulteration of the flora and fauna of such institutions. Reintroductions are only justifiable in the case of species that were indigenous to the area concerned during some time of its history.<sup>2)</sup>

A source of friction along the boundaries of national parks and reserves, particularly in cases where adequate range is not available, is the damage done to domestic animals and crops by carnivorous and other animals that wander out of the protected areas. Farmers are apt to describe the areas as breeding places of vermin and noxious weeds. Within the protected areas vermin does not exist, since there can be no natural biotic community without its complement of carnivorous animals. The latter are not vermin until they begin to help themselves to the domesticated animals or crops of the neighbouring farmers. A careful study of this problem awaits investigation along the western and the southern boundary of the Kruger National Park and on the boundaries of the Addo Elephants National Park. Less frequently damage is also done by rhinoceroses that wander out of some of the Zululand reserves.

In 1939<sup>1)</sup> I drew attention to the fact that the efficacy of salt and bone-meal licks in relation to the seasonal migration of ungulates should be thoroughly investigated in the Kruger National Park. If there are mineral deficiencies at certain times of the year, it may be possible to influence the movements of the animals by means of licks. The Conservator of Fauna and Flora, Dr. T. G. Nel, informs me that on a recent visit to the Nairobi Game Reserve he saw not only Thomson's Gazelles but a Giraffe taking a mineral lick off the ground.

Other important conservation problems that await investigation in our national parks and reserves, particularly the Kruger National Park, are the following:—<sup>2)</sup>

- (a) Factors affecting the densities of wild life populations.
- (b) The interrelations between carnivorous and herbivorous species.
- (c) The diseases and epidemics to which wild animals are subject.
- (d) The relationship of supplies of food and water to the animal populations and methods of improving the position where necessary.
- (e) Deficiencies in the food supplies.
- (f) The effects of veld burning and other factors on the annual migrations of ungulates.
- (g) The carrying capacities of the areas concerned.

- (h) Difficulties arising from the simultaneous occupation of national parks by man and wild life.

Let us now see what recent progress has been made on the scientific front of wild life conservation in the Union.

In the Cape Province a full-time biologist was first appointed in 1942 to take charge of the Jonkershoek Inland Fish Hatchery. It is anticipated that the scope of this biologist's work will be extended, so as to place him in charge of the flora and fauna of the Cape Province on a similar basis to that of the Conservator of Fauna and Flora in the Transvaal.

On the 28th March, 1945, the Transvaal Provincial Administration appointed a commission to inquire into and report upon the problem of game preservation in Transvaal. This commission's report (1945) written by Mr. S. Lombard, the Provincial Secretary of Transvaal, was described by me at the time of its appearance as the *magna charta* of game preservation in Transvaal. The most important recommendation in that report is undoubtedly the one in which the establishment of a game department under two professional officers is proposed. In 1947 this recommendation was put into effect by the appointment of a Conservator of Fauna and Flora and a Director of Inland Fisheries. This was the first occasion in the history of South Africa on which the welfare of the wild life of a province, other than that of freshwater fish, was entrusted to biologists. A Fauna and Flora Advisory Board has also been established by the Provincial Administration of Transvaal.

In Natal the Administrator appointed a Research Committee of four members on the 1st December, 1947, to deal with research in the Natal reserves.

In the Orange Free State, where there are as yet no national parks, no provision for scientific wild life conservation has yet been made. It is probable, however, that the Provincial Council of the Orange Free State will follow the example set by the Provincial Council of Transvaal.

In addition to progress made in some of the Provinces, the Minister of Lands appointed a Scientific Advisory Council for National Parks and Nature Reserves on the 1st January, 1949. This body consists of two zoologists, two botanists, one agriculturist, one geologist and one veterinary surgeon. It advises the Minister of Lands on wild life problems, and it is significant that scientific advice will now also be sought when the establishment of new national parks is to be undertaken.

The most recent advance is the appointment of a biologist to the staff of the National Parks Board of Trustees. I have been pressing for the creation of such a post for more than ten years. In the United States professional personnel is employed by the National Park Service "to study problems concerning the maintenance, protection, preservation and interpretation of the scenic and scientific values in national park areas and to submit recommendations pertaining thereto".<sup>10)</sup> The object of research work in the national parks of the United States of America is summarised as follows:— "To procure a

constant flow of essential facts relating to the natural features, the interrelations of life forms and their interpretation to visitors or to administrative policies. Basic data are frequently obtained through the co-operation of outside agencies because most parks do not have adequate funds and personnel to carry on a sustained research programme".<sup>10)</sup>

The British Wild Life Conservation Special Committee<sup>4)</sup> emphasises the necessity for centralisation of research in the following words:— "If a co-ordinated scientific policy is to be applied and a staff with the necessary attributes recruited and held, the management of the reserves must be brought within a wider scientific service concerned with the basic problems of nature conservation and control throughout the country. Without some control direction by highly skilled full-time officers, individual wardens working in isolated reserves could do little towards achieving the principal objects of these proposals". Centralisation of wild life research in the Union must also be the ultimate goal, but in view of the fact that we were heading for disaster because wild life research has been completely ignored ever since the National Parks Board came into existence 24 years ago, the appointment of one biologist at this juncture is fully justified. It is perfectly clear, however, that the research activities in connection with wild life conservation in the Union will have to be modelled on the pattern of the American or British systems. At present there is little prospect of obtaining the funds required for this purpose. The important thing is that a beginning is being made. Expansion will follow in due course.

With regard to the future of wild life conservation in South Africa, progress will be principally in two directions, namely scientific control based on a study of the scientific problems, and the provision of educational facilities for the thousands of tourists that visit our national parks and reserves, particularly the Kruger National Park, every year. Educational work is, in fact, complementary to the scientific work, for the investigation of the scientific problems will furnish a constant and reliable stream of facts that will form the basis of the educational work.

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# THE USE OF ANTI-FROTHING AGENTS IN THE TREATMENT OF ACUTE BLOAT.

R. CLARK,  
Onderstepoort.

*(Paper and Demonstration given at the 45th Annual General Meeting.)*

## SUMMARY

Experiments are reported in which carbon dioxide was introduced into the rumen of fistula sheep and the intraruminal pressure recorded. Tracings are presented, showing that the administration of anti-frothing agents into the rumen greatly facilitated belching, with a consequent drop in pressure despite a constant inflow of gas.

## INTRODUCTION

In 1948 Clark<sup>1)</sup> showed that turpentine and certain phenol preparations, commonly used as anti-fermentatives in the treatment of acute bloat, did not reduce the amount of gas formed. It was suggested that the undoubted beneficial effect of such remedies lay in their surface tension action, thereby breaking the foam.

In 1949 Quin, Austin and Ratliff<sup>2)</sup> reported successful clinical trials in America with a proprietary anti-frothing agent.

The object of this paper is to report on the results of experiments on induced bloat and its treatment with anti-frothing agents.

## ARTIFICIAL INTRODUCTION OF GAS INTO THE RUMEN

Owing to the extreme difficulty encountered in producing bloat, it was decided to introduce the gas into the rumen artificially. Carbon dioxide was run from a cylinder into 20-litre aspirator bottles. The gas was then displaced by water from the mains and forced into the rumen. Sheep with permanent ruminal fistulae were used and three tubes were inserted through the cork in the fistula as follows:—

- (1) The gas inlet about 9 inches long tipped with a perforated rubber tube to spread the gas.
- (2) A tube connecting a small rubber balloon inside the rumen to the water manometer with a float and style writing on a moving kymograph.
- (3) A short tube, fitted with a piece of rubber tubing and a clamp, through which the remedy could be injected without loss of intraruminal pressure.

## EXPERIMENTAL RESULTS

Despite every effort made to reproduce conditions conducive to bloat, it was found very difficult to cause retention of gas within the rumen. The great majority of sheep started belching when the intra-ruminal pressure reached 2 to 3 centimetres of water and then maintained that tension, without any signs of distress, despite an inflow of up to two litres of gas per minute.

The experiments were finally conducted as follows:— The team of six fistula sheep were starved for 15 hours and then fed an abundance of fresh green lucerne. Preliminary trials, consisting of the introduction of one litre of gas per minute were then made and only those animals which retained the gas and developed a pressure of 5 cms. of water were selected. The vast majority of trials was unsuccessful and the sudden appearance of a "bloater" could not be explained. It was noticed, however, that one particular animal reacted on several occasions. These findings are in conformity with the general experience of natural bloat where individuals of a herd suddenly develop the condition for no accountable reason.

In those instances where belching did not take place, it was noted that visible distension of the abdomen and signs of distress became apparent at a pressure of approximately 5 cms. water. The animals became restless with frequent urination and defæcation. At this stage 5 cc. of a proprietary anti-frothing agent was introduced into the rumen, the introduction of gas and recording of pressure being continuous throughout.

The tracings taken during two such trials are shown in figures 1 and 2.

### INDUCED BLOAT BY NATURAL FERMENTATION

Figure 3 indicates the effect of turpentine on the intra-ruminal pressure of a sheep which developed moderate bloat after being dosed 100 gm. of sucrose and 2 gm. saponin.

### BELCHING AFTER ANTI-FROTHING AGENTS

During the experiments an observer was stationed at the sheep's head to note the occurrence of belching. Profuse regurgitation of the gas invariably followed the introduction of the anti-frothing agents.

### CLINICAL USE OF ANTI-FROTHING AGENTS

A limited number of natural cases of bloat have been treated by the injection of anti-frothing agents directly into the rumen via the flank with excellent results. In a few instances belching did not occur spontaneously after treatment. It would appear that a very high intra-ruminal pressure interferes mechanically with belching. In such cases the gas is easily removed by the passage of a stomach tube. In extreme emergency a trocar should be inserted and the remedy injected through it.

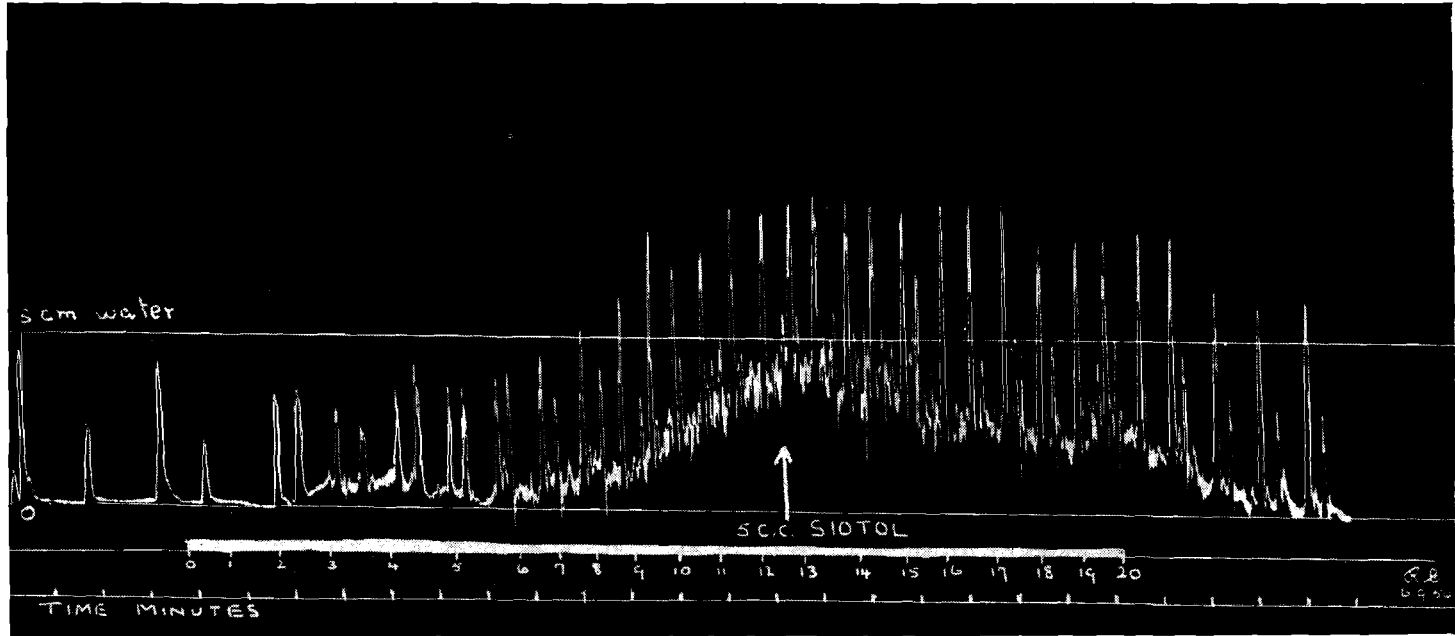
It is hoped that these preparations will shortly be available to veterinarians and that the findings in clinical practice will be communicated to this journal.

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FIGURE 1.

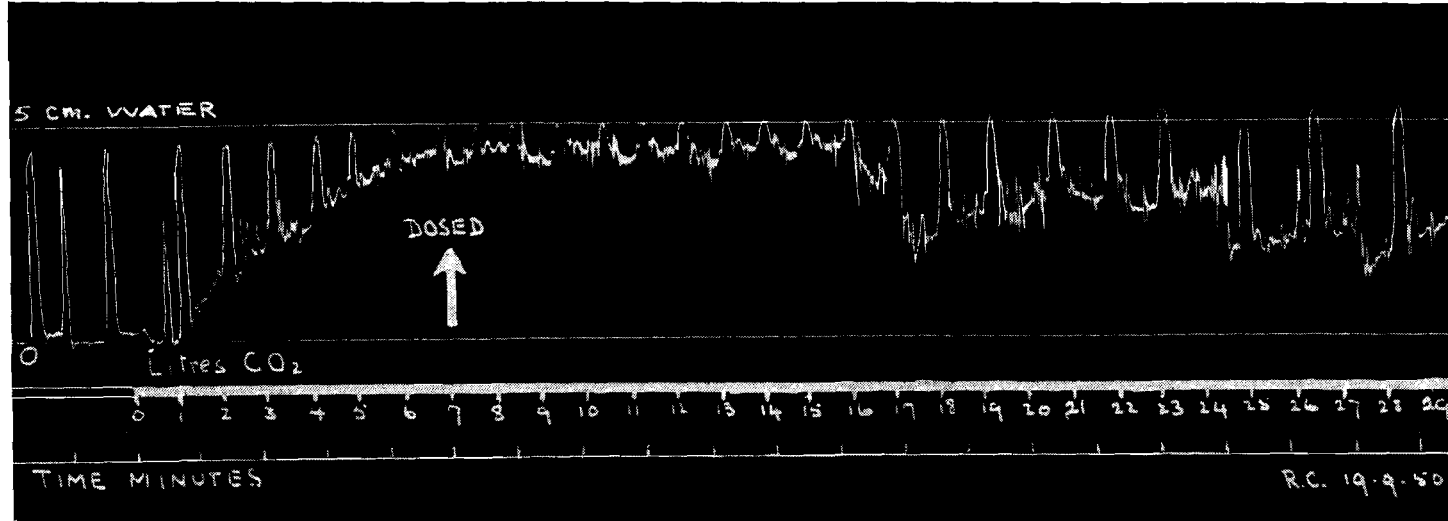
*The effect of an anti-frothing agent on ruminal pressure during the introduction of carbon dioxide.<sup>1)</sup>*



*Carbon dioxide was introduced under pressure during the period covered by the heavy white marker line, the figures representing litres of gas. Note the marked reduction in intra-ruminal pressure almost immediately after dosing.*

FIGURE 2.

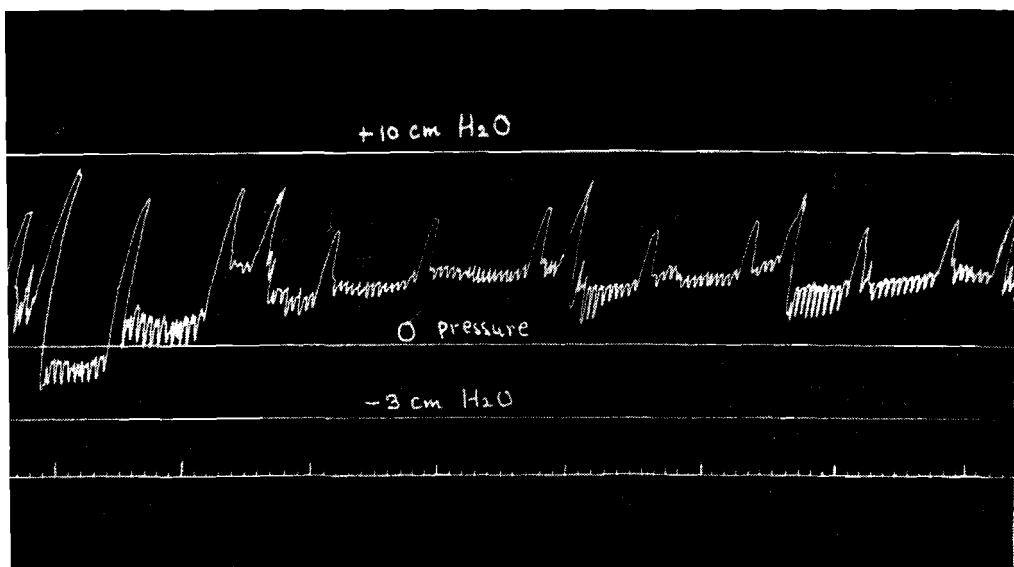
*The effect of an anti-frothing agent on ruminal pressure during the introduction of carbon dioxide.<sup>2)</sup>*



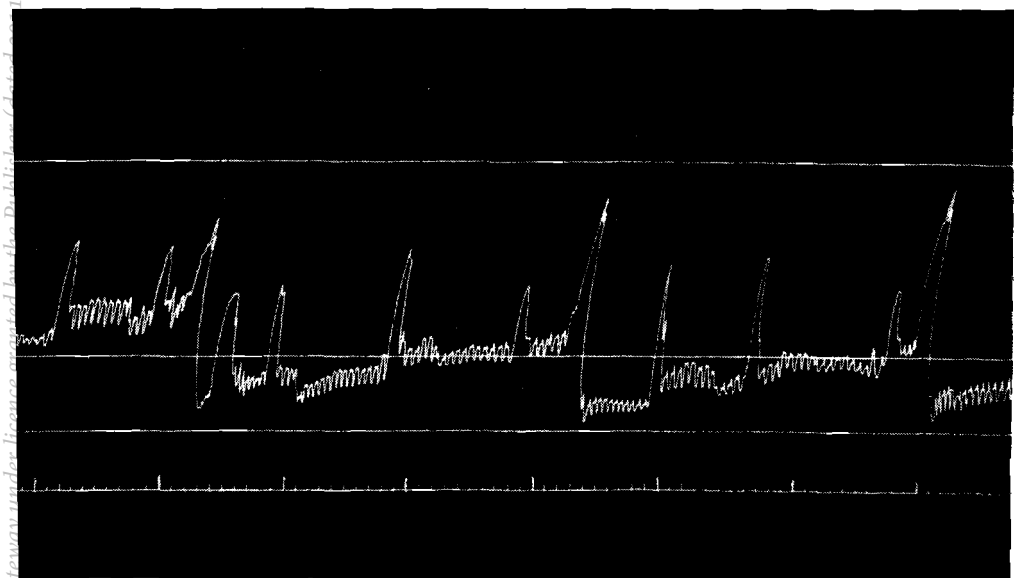
*Experiment identical to figure 1. Note sudden drop in pressure 10 minutes after dosing. The smallness of the contractions recorded at high pressure is probably not actual, but due to faulty recording. The maximum pressure that can be recorded is that at which all air in the balloon is extruded. Compare with figure 1 where ruminal tension increased the force of the contractions.*

FIGURE 3.

*The effect of turpentine on belching.*



A. — Moderate rise in ruminal pressure 10 minutes after dosing with 100 gms. sucrose and 2 gms. saponin.



B. — Immediately after introduction of 2 cc. turpentine into rumen. Note facilitation of eruction within two minutes. Note also that turpentine did not influence amount of gas formed as indicated by the steady rise in pressure between eruptions.

# ENTEROTOXAEMIA OF SHEEP.

K. C. A. SCHULZ and G. D. SUTTON,  
Onderstepoort.

*(Paper read at the 45th Annual General Meeting.)*

In his presidential address to the South African Biological Society in 1931, Robinson,<sup>1)</sup> in reviewing some of the anærobic infections of animals, pointed out the part played by the *Clostridium welchii* group in causing sheep diseases in various parts of the world and referred briefly to its economic importance. The better-known diseases set up by this group are Lamb Dysentery (Bloedpens) and Enterotoxæmia (Pulpy kidney) in South Africa, Lamb Dysentery, Enterotoxæmia and Struck in England, Enterotoxæmia in Australia, New Zealand and Tasmania and "Overeating" or "Diabetic Coma" (Enterotoxæmia) of feed lot lambs in the United States of America. These diseases are of considerable economic importance especially at present with soaring wool prices and the increase in value of sheep.

*History:* Enterotoxæmia is probably not new in this country even though it was only diagnosed for certain in 1948. In the past mortality in sheep from Enterotoxæmia was probably ascribed to Geilsiekte, Malignant Oedema, Plant poisoning or Poisoning from crops damaged by frost. During 1948, Schulz and McIntyre<sup>2)</sup> investigated outbreaks of a fatal disease affecting sheep, principally young lambs 6-9 months old, and made a tentative diagnosis of Enterotoxæmia. This was confirmed on a subsequent occasion when the causal organism (*Cl. ovitoxicum*) and its toxin were demonstrated by Scheuber<sup>3)</sup> in the intestinal contents of sheep affected with a similar disease. Since then the disease was been found in many parts of the Union and South West Africa.

*Incidence:* The disease has a wide incidence and has been encountered on sheep farms in different parts of the Union and South West Africa, but more particularly in the southern part of the Orange Free State (Smithfield district) and parts of the Eastern Province (Somerset East, Queenstown and Grahamstown districts). It is of interest that Lamb Dysentery (Bloedpens) is also enzootic in these areas.

*Cause:* The disease is caused by a bacterium *Clostridium welchii* type D (*Cl. ovitoxicum*). There are four known types of *Cl. welchii*. Type A causes gas gangrene in man and animals after wound infection. Type B (*Cl. agni*) causes Lamb Dysentery (Bloedpens). Type C (*Cl. paludis*) causes Struck and Type D Enterotoxæmia. There are five toxic factors associated with the *Cl. welchii* group, namely  $\alpha$ ,  $\beta$ ,  $\gamma$ ,  $\delta$  and  $\epsilon$  toxins. Type A produces  $\alpha$  toxin, type B all of them, type C all except the  $\epsilon$  toxin and type D the  $\alpha$  and  $\epsilon$  toxins.

*Predisposing Causes:* Before the causal organism can actually set up the disease some predisposing factors appear to be necessary. The action of these predisposing conditions is not known precisely and there may be many of them interacting with each other. Any condition which leads to atony of the intestines is a possible predisposing cause. The disease frequently occurs after sudden changes in the diet such as when sheep have been moved from poor to good pasture, after rains and the appearance of young green grass, overfeeding on concentrates such as maize, peas, barley and oats or when grazed on fodder crops such as green oats, wheat and lucerne, especially if these are damaged by frost. Another aspect to be considered is that substances normally present in the intestinal tract of sheep such as trypsin and pancreatin may in certain conditions augment or activate the potency of the toxins formed by *Cl. welchii*. Enterotoxæmia has an apparent seasonal occurrence. The incidence usually increases in the winter months. The mortality, however, may vary considerably from year to year. In some seasons a large proportion of the lamb crop may die, in others losses are negligible.

*Symptoms:* Peracute, acute and chronic cases of the disease may occur. The disease often has such a peracute course that even shortly before death no clinical symptoms are observed. The affected sheep are generally found dead in the morning, but sudden deaths may also occur during the day.

When clinical cases are seen they show either a syndrome characterised by coma and quiet death or one characterised by nervous symptoms and convulsions.

In the comatose form of the disease, the affected sheep lags behind the flock, tires easily, shows signs of exhaustion, has a staggering gait or knuckles over in the fore limbs, supporting itself on the knees and hind feet. As the disease progresses the sheep lies either in the sterno-abdominal position with its head resting on the flank, or is recumbent. It becomes comatose, and death supervenes without any signs of struggling. In addition the visible mucous membranes are infected and in some cases a sero-mucoid secretion is seen in the medial canthus of the eye and the nostrils. Laboured respirations and dilation of the pupils associated with impaired vision occur. Occasionally lachrymation, salivation, grinding of the teeth, hyperæsthesia, diarrhœa, bloating and a rise of temperature are noted.

In the nervous form of the disease a period of restlessness, characterised by wandering about aimlessly or barging into stationary objects is followed by dullness. The animal becomes depressed, holding its head just off the ground with its ears drooping. It may suddenly go into convulsions, when it lies flat on its side and exhibits more or less violent paddling or galloping movements, twitching of the muscles, rolling of the eyes, grinding of the teeth and frothing at the mouth. This continues almost up to the point of death. In the final stages it may be on its side with its head well back (opisthotonus) and the legs outstretched.



*Post-mortem Appearance:* The pathological features described are based on the examination of typical cases found dead, or cases *in extremis* either killed or allowed to die. Autopsy must be carried out immediately because putrefaction commences early and progresses rapidly even in cold weather. When putrefaction is advanced a blood-stained frothy fluid runs out of the nostrils, the abdomen is distended, the inguinal and axillary regions are swollen and dark purple in colour and the wool can be pulled out from any part of the body quite easily. The post-mortem appearance is not pathognomonic of the disease and depends largely on how long the animal has been dead. The most prominent and constant changes are seen in the kidneys especially two or three hours after death. They are extremely soft and pulpy, and subcapsular hæmorrhages may be present. The cortex is principally affected, its blood vessels being infected and its colour reddish brown. The pulpiness of the kidneys is presumably a post-mortem change, since, if the autopsy is made immediately after death, the kidney is still firm although the intermediary zone and medulla are markedly infected. Other features are congestion and cyanosis of the visible mucous membranes, marked congestion of the blood vessels, hæmorrhages, and reddish discoloured patches on the internal surface of the skin especially over the neck and forequarters. Oedema of the subcutaneous tissues is seen in these areas, the œdema fluid being reddish in colour. There is pronounced tympany of the intestinal tract, submucosal petechiæ and venous stasis of the mesenteric vessels. There is slight hydrothorax and marked hydropericard, the fluid being blood-stained and partially coagulated. Subepicardial and subendocardial hæmorrhages, degenerative changes in the myocard, marked hyperæmia and a variable degree of the œdema of the lungs are found. Pronounced congestion and catarrh of the nasal mucous membranes and enlargement, venous stasis and degenerative changes in the liver occur. All carcases examined were very fat.

*Diagnosis:* After taking the history, symptoms and post-mortem lesions into account and excluding other causes of death, the small intestine should be examined. Make smears from the contents of the jejunum or ileum. Stain these by Gram's method and examine microscopically for *Cl. welchii*. This is a short, thick, gram-positive rod. A few spores may be present. It is present in large numbers in positive cases. It is essential to examine smears from the intestines of sheep which have been sacrificed *in extremis* or not been dead long. Putrefactive organisms might be mistaken for *Cl. welchii*.

In the laboratory the disease can be diagnosed by the demonstration of the toxin in the intestinal contents. Unfortunately, a negative result does not necessarily exclude the disease. Attempts can be made to isolate the organism in cultures, but the organism may be present in small numbers in the intestines of normal sheep. Its mere presence cannot be regarded as diagnostic.

*Specimens required for laboratory diagnosis:* Smears from the intestinal contents and short lengths of the intestine tied off at both

ends to prevent escape of the contents should be sent. Use 50% glycerine to preserve the small intestine. Instead of intestine, the fresh intestinal contents preserved with  $\frac{1}{2}\%$  of chloroform (1 drop to 10 cc.) could be submitted for examination. Portions of mid brain, kidney and liver in 10% formalin are required for pathological examination.

*Differential diagnosis:* Conditions which might be confused with Enterotoxæmia are plant poisoning, Geilsiekte, Heartwater, Malignant Oedema, Anthrax, Pregnancy Disease of Sheep (Domsiekte), Listarelliosis, Tetanus, Braxy, Black Disease, Struck, Botulism and any condition which might set up a toxæmia.

*Control measures:* The most important of these is the use of the Enterotoxæmia vaccine. A dose of 5 cc. subcutaneously followed by a second dose of 5 cc. subcutaneously one month later is recommended. A third dose of 5 cc. subcutaneously a month later can be given on farms where the disease is very prevalent and severe. After that a single dose per year should be sufficient. Lambs under three weeks of age should not be inoculated. All other sheep, irrespective of breed or age, should be given the full dose of 5 cc. at the intervals recommended. Pregnant ewes can be inoculated at the time of mating and a second time about 14 days before lambing. A dose of 5 cc. should be given each time. The young lambs will obtain an immunity from the colostrum. The immunity lasts about one year and sheep should be inoculated annually.

The Lamb Dysentery vaccine does not protect against Enterotoxæmia even though there is some toxin in it. It should not be used instead of Enterotoxæmia vaccine even as an emergency measure. Other vaccines will not protect against the disease either.

When the disease actually breaks out on a farm the factors predisposing to the disease should be eliminated if possible. The sheep should be given sufficient bulky, fibrous material in their food, and exercise to promote healthy bowel movement. Overfeeding on easily digested concentrates should be avoided. Move the sheep to another part of the farm and try to keep them off short grazing particularly in camps where the causal organism is prevalent. Heavy concentrations of sheep on small areas such as lands and overstocking should be avoided or rectified.

As far as is known there is no substance which can be dosed orally which will stop the mortality, but it is a line of investigation which needs further study.

## DISCUSSION

Dr. J. Watt related his experiences of the disease in South West Africa and indicated that Sulphaguanidine appeared to control outbreaks but was only a practical measure with valuable sheep. He found the vaccine very effective.

Dr. N. T. van der Linde described the symptoms seen in sheep in the Orange Free State. They were very similar to those given in the paper. He confirmed that vaccines such as the Lamb Dysentery

and Blackwater vaccines did not control outbreaks of Enterotoxæmia, but that he had had good results with the Enterotoxæmia vaccine.

Dr. D. J. Louw inquired if cattle were susceptible to the disease as he had encountered cases in cattle similar to Enterotoxæmia. At the time the authors were not certain on this point. Subsequently, a search of the literature has revealed references to other animals being susceptible.

Taylor and Gordon<sup>4)</sup> isolated Cl. welchii type D from the intestinal contents of an apparently normal bovine killed for human consumption. Thus Enterotoxæmia might well be the cause of death in certain unexplained fatal illnesses of short duration in cattle.

Lesbouygries and Berthelon<sup>5)</sup> state that Enterotoxæmia may occur in calves 2-4 weeks old and goat kids, the disease ending fatally in 6-24 hours.

Gordon<sup>6)</sup> reports isolating the toxin of Cl. welchii type D and this organism from the intestinal contents of acute cases of grass-sickness in horses.

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# ARTIFICIAL INSEMINATION AND THE BREEDING OF STUD CATTLE.

(Summary of a talk given by Professor F. N. Bonsma.)

The first recorded use of artificial insemination was carried out on a mare in Arabia in 1322, but the first scientific research on the subject was done by the Italian physiologist Spallanzani in 1780. The possibilities of the application of the procedure on a large scale were first realised in Russia where by 1914 between 300 and 400 persons had been trained in the technique. By 1938, 120,000 mares, 1,200,000 cows and 15 million ewes had been artificially inseminated. The first centre in Denmark was established in 1936 and by 1947 there were 100 such establishments. The practice extended to Great Britain during the last war and later to America.

The survival period of stored semen and long distances are the greatest limiting factors to the use of artificial insemination, especially in the Union. Semen can now be stored four to five days, but the viability decreases rapidly with storage. This limited period during which semen can be stored affects the stud breeder more than it does the commercial herd owner, as the latter can usually obtain semen from a convenient nearby centre. According to experience in England there must be over 5,000 cows within a radius of 15 to 20 miles from a centre in order to make it an economic proposition.

It must be emphasised that the application of artificial insemination to commercial and stud herds presents entirely different problems.

## THE GENERAL ADVANTAGES OF ARTIFICIAL INSEMINATION

1. *The number of calves obtained from each bull.* Some 40 - 50 cows can be inseminated from one ejaculation. Overseas, up to 1,000 calves have been obtained from one bull in a year. By means of artificial insemination the maximum use can therefore be made of an outstanding sire. By natural methods a bull rarely leaves more than 500 to 600 progeny even though he be heavily worked and survives to a reasonable age.

2. *The possibility of proving a bull at an early age.* In co-operation with an artificial insemination centre a young bull can be bred to some 200 cows before he is 18 months old. Provided full production records of the dams and offspring are kept, his breeding potentialities can be accurately assessed very rapidly.

3. *The possibility of seasonal breeding.* In nature, nearly all species have an optimum breeding season. Although milk cows will breed all the year round the highest fecundity has been found to be seasonal. From an economic point of view it is also advantageous to have the cows calve in the early summer when food is plentiful. Only artificial insemination makes it possible for the majority of cows to be

inseminated within a short period. This seasonal breeding of cattle is the general practice in England, New Zealand and Holland. The percentages of the annual insemination done in England during the four seasons illustrate this point, viz.:—

Spring (April to June) .....	20%
Summer (July to September) .....	13%
Autumn (October to December) .....	30%
Winter (January to March) .....	37%

4. Artificial insemination is naturally of great value to the owner of only a few cows, as he obviously cannot economically keep a good bull. This aspect is becoming of increasing importance round our growing cities.

5. The control of venereal diseases.

6. Insemination is made possible where natural mating cannot be carried out due to injury or discrepancy in size.

7. The commercial breeder is made aware of the value of stud bulls.

8. An indirect yet important advantage of artificial insemination is that the stock owner is brought into closer contact with veterinarians and animal husbandry experts.

9. The use of scrub bulls is reduced.

#### THE EFFECTS OF ARTIFICIAL INSEMINATION ON STUD-BREEDING UNDER SOUTH AFRICAN CONDITIONS

1. The greater use of a smaller number of bulls will lead to increased in-breeding, thus limiting the choice of matings, a difficulty that has already arisen in Denmark. In-breeding leads to:—

(c) Production of families of uniform type.

(b) Homozygosity and prepotency are increased.

(c) Recessive undesirable characteristics appear.

The last-named effect of in-breeding is one of the greatest dangers attached to artificial insemination. A few of these undesirable characteristics already encountered are: Smooth tongue, misformed hooves and legs, udder abnormalities, wry-tail, bulldog calves (lethal) and smooth skin. As these factors are mostly recessive they usually cannot be discovered until the second generation, by which time enormous damage may have been done if unrestricted use of certain popular bulls has been allowed. The State protects the stock-owner against many infectious diseases; what is our duty with regard to these hereditary faults?

2. Stud-breeding is both a science and an art. It is based on the scientific principles of genetics and an intimate knowledge of the breed and lines within the breed. It is an art in that every breeder worthy of the name must work towards his own ideal with an intuition born of long association with his animals and not to be gained from a printed book.

Artificial insemination will tend to standardise breeding and suppress the personal element.

3. For successful breeding the animal must be genetically in harmony with its environment. Even in Holland, which is no bigger than the Heidelberg district of the Transvaal, a difference is recognised between Fries cattle from the "Zwaar Kleibodem" and those from the lighter soil. By largely eliminating distance, artificial insemination may lead to a standard type regardless of local conditions.

4. On the other hand, artificial insemination may frequently make it possible for a breeder to mate a particular bull to certain of his females.

#### *The Influence of Artificial Insemination on the Demand for Stud Bulls.*

There are some million milk cows in the Union, while approximately 1,250 dairy bulls are registered annually. It would therefore appear that there is a shortage of good bulls. The experience overseas has been that artificial insemination increases the demand for good bulls with high production potentialities, while the poorer class of so-called stud bull is decreased in value.

#### *The Effects of Feeding and Management.*

Apart from technical difficulties, artificial insemination can only be a success in commercial cattle breeding in South Africa provided it is realised that an increase in genetic potentialities must be accompanied by better feeding and management. The fact that the average annual milk production per cow is only 242 gallons is not primarily due to breeding, but rather to poor feeding, management and the prevalence of disease, such as mastitis and sterility.

#### *The Essentiality of Milk Recording.*

As has been explained the great advantage of artificial insemination is the widespread use of bulls whose progeny will be better producers than their dams, while the danger is the wholesale distribution of undesirable factors. In other words the success of artificial insemination depends on our exact knowledge of the breeding potentialities of the bulls used. This cannot be obtained without accurate and widespread recording of the milk and butter-fat yields of both dams and progeny. The hopelessly inadequate facilities at present available for such recording among commercial herds is one of the biggest stumbling blocks in the use of artificial insemination on a large scale.

#### *Actions taken by the State to Protect Stud Breeders.*

(a) Regulations approved by the Minister empower the Breed Societies to lay down very high standards for bulls used for artificial insemination. The State can prohibit the use of undesirable bulls and further no bull may be used for the insemination of more than 25 cows outside the owner's own herd until he has been proved.

(b) The legal protection of farmers against the commercialisation of artificial insemination shall be under a board on which stud-breeders will be represented.

# PITFALLS OF ARTIFICIAL INSEMINATION AND SEMEN EXAMINATION.

S. W. J. VAN RENSBURG,  
Onderstepoort.

*(Paper read at the 45th Annual General Meeting.)*

According to available literature, artificial insemination has been practised in South Africa since 1932 when it was introduced by Quinlan and co-workers of Onderstepoort. Since then and up to a year or two ago it was used on a small scale and mainly at research and experimental stations. It is only during the past 18 months that this method of breeding has become a subject of major importance among farmers and breeders. The chief incentive for this has no doubt been the recent unpleasant revelation that, apart from the many other forms of infertility, we have at least three types of infectious infertility in cattle, namely contagious epididymitis and vaginitis, trichomoniasis and vibrio-fœtus abortion, whose presence here was not suspected previously. Associated with this is the realisation that artificial insemination, apart from its many other advantages, affords the best and safest method of combating and preventing these diseases.

Rapid development of artificial insemination in South Africa during the past year has taken place along three main lines, namely:—

- (a) In large dairy herds where the owners inseminate their own cows with semen obtained from their own bulls.
- (b) The insemination of beef cows and heifers on large ranching herds.
- (c) For commercial dairy herds on a co-operative basis by approved centres.

The institution of artificial insemination even under the most favourable conditions is always complicated by a great variety of pitfalls and difficulties which are often very discouraging to the promoters of the system. Unless remedied or prevented these may shake the confidence of breeders and may wreck the scheme in its infancy. Difficulties were accordingly anticipated here, but owing to a large number of adverse conditions peculiar to South Africa, these growing pains are proving extraordinarily severe.

Besides the usual difficulties which beset such a new undertaking our breeders have to contend with two additional handicaps which add greatly to, and intensify, the other obstacles which are normally to be expected.

In the first place there is the very high incidence of infertility of all types in both bulls and cows in this country. Notwithstanding all previous warnings that artificial insemination must be regarded as a

preventive rather than as a cure for sterility, and that a cow that will not conceive to natural service by a fertile bull cannot reasonably be expected to settle to insemination, we still find many farmers applying this as a last resort for cows which are hopelessly sterile.

Secondly, South Africa suffers in comparison with the rest of the world in that the introduction of artificial insemination is not financed by a State department or any other organisation with the necessary funds at its disposal to meet the initial heavy capital expenditure, without which no artificial insemination centre can be started on a sound basis. Before a centre can start functioning and earning revenue, ground must be purchased, premises erected, equipment obtained, a number of bulls acquired, personnel trained and transport provided for inseminators. Under present conditions a few enthusiasts in areas that are eminently suitable for insemination on a co-operative basis have to take upon themselves the difficult task of finding the necessary funds to cover the initial expenditure. To do this they are obliged to resort to measures which ordinarily we would deprecate as being too hazardous. Thus, for instance, they have to enrol as many members and cows as possible right from the start. That means that a centre, instead of beginning on a small scale and expanding gradually, has to function at full speed immediately it starts operating. The danger of this in a country where there are few trained and experienced personnel is obvious.

It would be far safer for centres to start on a small scale and develop in the course of time. For instance Reading, the oldest and one of the most successful centres in Britain, inseminated only 346 cows during the first year of its existence (1943). The number increased rapidly as good results were obtained and farmers gained confidence, and in 1948 17,982 cows were inseminated.

For a new centre it is more necessary than ever to guard against accepting herds in which infertility is rife. Yet financial stringency sometimes leaves the organisers no option in the selection of herds. The results of this are poor conception rates and loss of confidence.

The many other difficulties and snags may be discussed under the following heads: (1) Personnel, (2) Bulls, (3) Cows, (4) Technique, (5) Semen Examination.

### (1) THE PERSONNEL

Too many people still regard artificial insemination as a very simple operation, like a hypodermic injection, which requires no special training, skill or experience. This is especially the case in private herds, in which the owners consider that once any one of the farm hands has mastered the technique of passing a pipette through the cervix, he is an accomplished inseminator. They are either oblivious or sceptical of the many snags involved, which in such cases may only be revealed to them after months of failure and often at heavy cost. Thus in one case investigated by us it was found that the farm foreman had been merrily inseminating the cows with dead sperm for many weeks, and it was only after some months that the return to service of 100 per cent of inseminated cows convinced the owner that all was not well.



The first point on which a breeder who proposes to apply this method of breeding must be disillusioned is that the collection and handling of semen and the insemination of cows are merely little part-time jobs which can be undertaken by one of his labourers at odd spare moments at any time during the day. It cannot be too strongly stressed that the various duties involved in insemination must have prior claim above all other farm activities on that individual's time. Quite recently a progressive farmer who last year decided to engage a full-time veterinarian to inseminate and to control infertility in his large ranching herd called on us to express his great satisfaction with the results they were getting. This example is quoted to show that the many difficulties can be overcome if tackled in the right manner. What a pleasant contrast this offers to the other case quoted above.

In a paper presented at the International Veterinary Conference in London last year A. I. Holt of Darlington stated that somewhere in the regions of 800-1,000 inseminations must be performed by an inseminator before he can be regarded as experienced and efficient, and his conception rate will rise by about 10% during this process.

A knowledge of the technique of insemination is by no means all that is required of the technician. He should in addition be well versed in all aspects of collection and handling of semen. He should further have a thorough knowledge of animal management, and should specially study the characteristics and idiosyncrasies of the individual bulls. A good knowledge of bull psychology is indispensable if one is to get the best out of every animal. Here one must emphasise, too, the desirability of having as far as possible the same people always handling the bulls. No two collectors employ exactly the same technique, and to have a different lot of people collecting from the same bulls at various times will sooner or later put them off service. In addition the collector should always wear the same type and colour overall or coat when taking semen, so that the bull on seeing him approaching with the necessary equipment and in proper dress will immediately know what is required of him.

The actual handling, grooming, feeding, etc., is usually left to Natives or Coloureds. There is no objection to this as long as the boys have a thorough knowledge of this work and can be trusted not to ill-treat the animals. Any pain or injury inflicted on a bull, particularly when collecting semen, will soon make him a reluctant worker. Recently we found a bull refusing flatly to serve, because the boy had pulled too hard at his nose when leading him out. Having once been roughly handled in this manner the bull associates the act of service with pain and may thus be ruined for good. The Coloured and Native mind cannot always appreciate these finer points in bull management, which may make all the difference between success and failure. It is therefore necessary that even the most reliable boys should be under very close supervision.

Each bull is an individual entity and no hard and fast rules can be prescribed for their management. It is only a very close study of every animal that will indicate what the best method of handling each will be. It has thus been found that many mature Afrikaner

bulls are most obstinate and unwilling to serve when led with a bull stick, but when freed from all control they will readily mount the teaser cow and serve into the artificial vagina.

## (2) THE BULLS

One of the first mistakes which most new centres and breeders make who start insemination is to work out their bull requirements by mathematical calculation. They reckon that because the semen of one bull can theoretically be used for several thousand cows, two bulls will be ample for their immediate requirements.

In practice this just does not work out that way. Due allowance must be made for all the unforeseen contingencies such as a bull not taking readily to service in an artificial vagina, sickness, injuries, a drop in the quality of the semen, and many other conditions which may make the temporary withdrawal of a bull from service imperative. Once the artificial insemination service has started it must be maintained with regularity and efficiency. It is fatal for the organisation if it has to advise clients that cows cannot be served on any one day because something has gone wrong with one of the bulls.

One misfortune leads to another. When an inadequate number of bulls is kept, the withdrawal of one invariably involves too heavy a strain on the other. We have in the past six months encountered three different cases in which bulls which had been giving high quality sperm have been overworked to such an extent that the quality of the sperm had dropped so low as to cast serious doubts on its fertility. In two of these cases it will take the bulls months to recover full fertility.

Another common method of overtaxing bulls is by imposing excessive demands on the willing server. This is seen particularly where the collector is a part-time worker. He has other more pressing duties awaiting his attention, and simply has not got the patience or the time which is sometimes required to get a lethargic bull to serve. He therefore calls upon the willing worker to an excessive degree.

Young bulls are usually bought when starting new centres and sight is lost of the fact that even though a young bull may be apparently mature he has not got the same capacity for service as older ones. Special care should be taken not to make excessive demands on them. Observations made over five years at Reading have shown that even mature bulls do not respond well to being worked three times per week, although some do exceedingly well when worked twice per week. The optimum appears to be once every 5-7 days for mature animals and once every 7-10 days for young bulls.

Another factor that must be reckoned with in purchasing animals is that a very large percentage of apparently normal bulls are not suitable for artificial insemination work. The fact that a bull has served once into an artificial vagina and that he has given semen of good quality in one or two tests is no guarantee that he will continue giving good service or that the standard of his semen will be maintained. A bull yielding semen of moderate quality may still achieve 100% success when serving naturally, because in this case the full ejaculate is deposited directly into the vagina without delay or interference of

any kind. Under artificial insemination conditions, however, there is quite a different story. The semen is collected, diluted, divided into small doses, stored and transported for some distance and then injected artificially. None but the highest quality semen will stand all this unnatural interference and the rough handling which it sometimes involves.

Bulls with moderate quality semen are not consistent in the quality of semen they produce, and a striking feature of such semen is that although the conception rate obtained with it on the first day may be about the same as that of good semen, it deteriorates rapidly on storage and shows a marked drop in fertility on subsequent days, apparently on account of its poor viability.

It is obvious therefore that for artificial insemination purposes bulls must not merely be fertile but they must consistently yield semen of the highest quality. It follows, too, that it is not advisable to buy bulls outright and unconditionally for this service. When it can be arranged it is preferable to hire the bulls with the option of buying if they prove satisfactory after a period of some months. An alternative is to buy on condition that a bull which proves to be unfit can be returned to the seller within a specified period. Few sellers will agree to these conditions except at an increased price, but to the artificial insemination centre the bull that has shown himself to be a good worker and a consistent producer of high quality semen is worth double his usual price.

### (3) THE COWS

Three conditions which are necessary for the successful insemination of any cow are:—

- (a) The cow must be free from all disease or abnormality of the genital tract which is likely to interfere with conception;
- (b) oestrus must have started not more than 20 - 24 hours prior to insemination;
- (c) viable fertile sperm must be deposited into the cervix or uterus.

As already indicated the widespread prevalence of infertility and the financial handicap which beset the inauguration of artificial insemination in South Africa have the inevitable result that many cows are presented for insemination which are hopelessly sterile. In many of these cows there is no veterinary history or other information available which can give any indication as to the cow's fertility. We did, however, come across one case in which the owner complained about the low conception rate (20%) in one of his two herds and in which the past history of the animals was known. Investigation showed that of 16 cows inseminated in this herd 13 had returned to service, equivalent to a conception rate of only 18.7%. Their veterinary history, however, revealed that no less than 10 of the 13 failures either had some or other morbid condition of the uterus or were showing evidence of abnormal vaginal discharge at the time of insemination. A completely different picture was shown by the clean herd of the same owner. Here 10 out of 18 cows conceived, giving a conception rate of 55.5%, which is very satisfactory for the first insemination.

Once insemination is well on the way and proper records are kept in respect of all the cows, these will prove to be of invaluable assistance to veterinarians engaged on sterility work. This is one of the great advantages of artificial insemination which is all too frequently overlooked. In South Africa, more than in any other country, it is very desirable that sterility work and insemination should be very closely allied. It is naturally not possible to carry out a veterinary examination of every cow before she is inseminated, but the need for a thorough veterinary examination of herds before they are accepted for membership of artificial insemination centres cannot be too strongly stressed. Inseminators, too, should be able to recognise the common symptoms of infection of the genital tract and refuse to inseminate cows showing these symptoms.

In areas where contagious epididymitis and vaginitis is prevalent it may, however, be very difficult to determine when to inseminate cows again after they had been affected. In many cases it would be imposing too great a hardship on the owner to refuse service until all trace of the disease has disappeared. It is therefore suggested that such cows be inseminated once they are over the acute stage of the disease, but in all such cases the owner should be warned not to expect a high conception rate, and it is further advisable to make separate entries of infected herds in the records.

It has been found that on many farms observations for oestrus are made only once daily, usually in the early morning. Cows which start oestrus later in the day are therefore missed or may only be detected the following morning, and may therefore not be inseminated before 24 hours or more after the commencement of oestrus. In such cases the conception rate will be very low. It is therefore advisable that observations be made at least twice daily.

The onus for detecting oestrus and holding the cows available for the inseminator rests on the owner. Not infrequently he or his servants select the wrong cow. The inseminator should therefore have a thorough knowledge of the signs of oestrus, and satisfy himself that he is not inseminating a cow that is not on heat, or, worse still, one which may be pregnant. Failure to detect pregnancy recently led to a three months pregnant cow being inseminated, with the inevitable result that abortion followed. Such a mistake naturally leads to a great loss of prestige for the inseminator in that area.

The inseminator should be sufficiently skilled and experienced to detect early pregnancy, and should be able to do this without undue manipulation of the genital organs. Excessive handling of these parts during oestrus may cause a mature graafian follicle to rupture, and in such cases of manual rupture of the follicle the ovum is usually lost by dropping into the abdominal cavity.

Another point against which inseminators should be on their guard is depositing the semen too far into the uterus. The disadvantage of this may be twofold. All the sperm may be liberated in the horn on the side of the non-ovulating ovary. Secondly, there appears to be no difference in the results obtained between intra-cervical and intra-uterine insemination. Many believe that the cervical mucosa has a

definite filtering and purifying action on the sperm, and therefore consider it advisable to liberate at least some if not all the semen in the cervix.

#### (4) TECHNIQUE

The emphasis laid on correct technique is frequently regarded by the untrained and inexperienced as being exaggerated, and they consider that many of the details can be disregarded with impunity. Sight is lost of the fact that sperm are highly sensitive body cells which, although they can under favourable conditions survive for many weeks, are nevertheless very easily killed or rendered inactive by faulty handling.

A very common calamity which befalls semen is temperature shock. Exposure to this destroys a very large percentage of sperm however good its quality may be, and it may be sustained in many different ways.

Many collectors do not take the necessary precautions to keep the collecting tube at body temperature, with the result that on ejaculation warm semen is immediately brought into direct contact with the cold glass surface. After that it may be exposed to very rapid cooling, especially on cold winter mornings. We recently encountered a case in which the temperature of the semen had dropped from 37°C to 18°C during the few minutes while it was taken from the collecting pen to the laboratory. During winter it is particularly necessary to immerse the tube containing the freshly collected semen in a water bath at the same temperature and to let it cool gradually. On the other hand semen should be protected against sunlight, which also acts deleteriously on sperm.

Prior to dilution one must ensure that the semen and the diluent are at approximately the same temperature. The addition of cold diluent to warm semen will also cause shock. It must always be borne in mind that the purpose of dilution is not merely to increase the volume for insemination purposes, but chiefly to provide suitable media to enable sperm to survive in vitro.

In diluting, due consideration is often not given to the quality and density of the semen and the dilution is carried out to the desired extent irrespective of whether it is good or poor. We have thus seen semen of very poor density and with a motility rating of 1 being diluted 15 times. It is extremely doubtful whether such semen, even if used undiluted or by natural service, would fertilise a cow, and it is quite certain that none of the cows inseminated with this batch conceived.

Tests should be conducted from time to time to ensure that the diluent is up to standard and is not harmful to sperm. We have seen two instances in which the diluent, instead of preserving semen actually killed it within a few hours, and in the one case already quoted an inexperienced technician was using the dead sperm for weeks, quite oblivious of the fact that it was useless.

Although sperm is very resistant after proper dilution, a variation in temperature on storage will nevertheless decrease its fertilising capacity, and maintaining a constant temperature in the refrigerator is an important point which is often overlooked. Too low a temperature

such as a drop to below zero will also cause irreparable damage to the sperm.

Frequently sufficient attention is not paid to the selection of suitable premises in which the semen work is to be carried out. While it is not essential that a large elaborately equipped laboratory should be available, it is yet highly desirable that a separate room should be used for this purpose and for nothing else. Strong chemicals and pungent odours of any type should not be tolerated in the semen laboratory. We have seen the following types of semen "laboratories" actually in use in this country, namely, the implement shed on the farm, a corner in the cow stable, a portion of the farm dairy, and the kitchen in the farmhouse! One can hardly expect the most robust sperm to survive in this kind of environment.

Lack of attention to the artificial vagina may not only be harmful to sperm, but may also put bulls off service. There is a tendency to increase the temperature of the vagina unduly, especially for lethargic animals, and we have noted cases in which it was over  $50^{\circ}\text{C}$ . This may work on one or two occasions, but satisfactory service by the bull will not be maintained for long by taking the temperature of the artificial vagina over  $50^{\circ}\text{C}$ .

The cleansing and sterilisation of instruments seldom receive the expert attention and supervision which they ought to have. Too much reliance is still placed, especially by farmers and laymen, on the use of strong antiseptics or disinfectants. It is no exaggeration to state that more sperm are probably killed than saved by some of the methods of "sterilisation" at present employed. Frequently, too, equipment that has been properly sterilised is exposed to contamination again immediately afterwards. Great care is particularly necessary with the insemination pipettes. There is only one efficient method of sterilising them, and keeping them aseptic, dry and free from substances injurious to sperm. That is by putting them, after cleansing, into the round metal container provided for that purpose, with the pointed ends going in first, and then to place container with pipettes into a dry oven at  $120^{\circ}$  -  $150^{\circ}\text{C}$ . The container is left closed after sterilisation until it is necessary to take out a pipette immediately prior to inseminating. Even then the pointed end of the pipette, which has to be dipped into the tube of semen and thereafter to be placed into the cervix of the cow, is never touched by hand.

The chief mistake that has been observed in the despatch of semen is to send the diluted semen in only one bottle which may only be half full. The disadvantage of this is that semen has to be taken from the bottle at its destination for use on the first day. The bottle is then closed again and stored for use on the second or third days. Once a tube or bottle of semen has been opened and some of it used, the remainder should either be used on the same day or discarded, but not stored for further use. It would therefore be preferable to ship semen in a number of smaller tubes which are completely filled, one to be used on the first day and the balance to be stored in the refrigerator without having been opened, for subsequent use.

The objection to transportation in incompletely filled tubes is that

the air left in the tubes and the undue agitation of the semen during the journey are both detrimental to sperm.

### (5) EXAMINATION OF SEMEN

The examination of semen for fertility not only forms an integral part of the artificial insemination work, but also constitutes an essential routine for every veterinarian engaged in sterility work. The sudden realisation by farmers and breeders that there is a very high incidence of lowered fertility and complete sterility in bulls in this country is rapidly increasing the demand for this service.

Notwithstanding, however, the many advances made in recent years in the methods and technique of semen examination, it must be pointed out that this on its own does not constitute an infallible or entirely reliable criterion of a bull's fertility. There is as yet no satisfactory method of assessing the significance of many of the sperm abnormalities which may be found on morphological examination. Besides, semen may show marked variations from time to time, and a sperm picture which may mislead the unwary examiner into condemning a bull on one examination, may under favourable conditions improve so rapidly as to be quite normal again within a relatively short period. It is therefore always advisable to postpone judgment and to re-examine a month or two later.

One cannot emphasise too strongly the great danger of either condemning a bull as sterile or passing him as fully fertile merely on an examination of his semen. It is but only in a very small percentage of cases that the information obtained from such an examination is definite and conclusive. Wherever possible it should be supported by a thorough clinical examination of the whole of the genital tract.

The first essential in a seminal examination is to obtain a good sample. As a rule this can only be obtained by collecting in an artificial vagina. Massage of the ampullæ is most unsatisfactory, and the so-called ejaculate frequently consists of nothing more than the secretion of the accessory glands. Yet we know of cases where bulls have been certified as sterile merely because this ejaculate showed aspermia.

When examining for motility it must be remembered that sperm only shows optimum motility at body temperature. One incident was reported to us of semen being returned to the main centre because it was stated to be dead on arrival at the sub-centre. When the same sample was examined at the proper temperature many hours later it was found to be very much alive. Therefore the best method of examining for motility is on a thermostatically controlled warm stage, and such a stage should form an essential part of the equipment of every artificial insemination centre.

While motility may furnish a good indication of the degree of fertility in the daily routine examination at artificial insemination centres of the semen of bulls which have passed the other recognised tests and clinical examinations, one must be mindful of its limitations, especially in dealing with unknown animals. It is quite possible for completely infertile semen to show the highest degree of motility. This is usually seen in sperm which are defective on account of abnormalities

of the head. Propulsion of sperm is brought about by the tail, the head playing no part in movement. Gross abnormalities of the head may therefore not in any way affect motility. Yet aberrant sperm heads are of far greater significance in the etiology of sterility than the different types of abnormalities found in the tails.

We were recently consulted about a bull which was sold some time ago on a certificate that examination of his semen showed it to be of high fertility. This opinion was apparently based on an examination for motility only. After several months' use the buyer discovered that not one of his cows served by this bull was pregnant, whereupon his veterinarian forwarded a sample of semen. Morphological examination of this showed an abnormality affecting 100% of the sperm heads, and it was clear that the bull was quite sterile. The complications which arise when cases like this one result in litigation are obvious.

A mistake frequently made in the examination of sperm, especially when it has been diluted, is to regard any evidence of life or movement in the sperm as proof of good fertility. It does not follow that a live sperm is necessarily fertile. Those, for instance, that lie wriggling or oscillating in the same spot are but merely giving the last kick before passing out, and none of them will ever fertilise an ovum. Good, fertile sperm must show directional motility, that is, they must move forward as if with a definite purpose and aiming at a goal which they are determined to reach.

It is not claimed that the above completes the list of all pitfalls to which artificial insemination and sterility workers are subject. This paper is based merely on some of the mistakes committed and snags experienced by such workers and which have come to our notice. Further, we have dealt here mainly with the veterinary aspect. Other problems such as the danger of in-breeding, the risk of transmitting latent defects, the possibility of causing deterioration instead of improvement in the progeny, the disasters which may result from not keeping proper records, and many other points have not been touched upon, but these furnish sufficient material for a separate paper.



## NEW APPARATUS AND DRUGS MEDICAL DISTRIBUTORS

Medical Distributors of 236, Jeppe Street, Johannesburg, are the suppliers of two pieces of electro-medical apparatus now finding a useful field of work in veterinary surgery. These are both manufactured by the Birtcher Corporation of America, well known in this country for their medical and surgical short-wave diathermy units. They are the "Hyfrecator" Diathermy Cautery Unit and the Spot Quartz Ultra-Violet Lamp with Wood's Filter.

The "Hyfrecator" has been improved recently to give more power and finer control. Desiccation (light drying-out of tissue), fulguration (shallow destruction of tissue) and coagulation (complete destruction of tissue) can all be carried out with speed and efficiency. Most authorities agree that the advantages of electro-desiccation are:—

- (1) Rapid and effective destruction of abnormal growths without loss of blood.
- (2) A considerable area of tissue may be destroyed without infringement on normal tissue.
- (3) Resulting wound is always sterile.
- (4) Blood and lymph channels are sealed.
- (5) Little after-pain.

The "Hyfrecator" is normally supplied for operation on 220-250 volts A.C., but models are also available for 110 A.C., 110 D.C. and 220-250 D.C.

The Birtcher "Spot Quartz" answers the need for a quick means of diagnosing ringworm in domestic animals. Areas of infection caused by either *Microsporum andouini* or *Microsporum lanosum* will display a brilliant green fluorescence when viewed under filtered ultra-violet light in a darkened room. Of far greater importance is the fact that they not only are the larger patches of *Tinea capitis* thus visualised, but each individual isolated hair or follicle which is harbouring the organism will also fluoresce green, thus it is possible to prove visually whether an infection has been completely eradicated or not. The following causative organisms of *Tinea capitis* also fluoresce under the lamp: *T. violaceum* (dull blue-white), *A. shoenleini* (green-grey), *T. crateriforme* (dull blue-white), *M. fulvum* (light green) and *T. sulfureum* (pale grey), but by far the greatest offenders are *M. lanosum* and *M. andouini*.

The "Spot Quartz", used without the Wood's Filter, is also ideal for localised application of ultra-violet to small areas in the treatment of superficial infections, infected wounds (sluggish ulcers), dermatoses, etc.

The "Spot Quartz" is available for operation on 220-250 volts A.C. only. (See advertisement on page x.)

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## NEW 'CETAVLON' PREPARATIONS

'CETAVLON' CONCENTRATE 20%

'Cetavlon' Concentrate contains 20% of 'Cetavlon' in a dilute alcohol/acetone solution, distinctively coloured with a non-staining yellow dye.

This preparation affords an easy and rapid means of preparing both aqueous and alcoholic solutions of 'Cetavlon' of any desired strength for immediate use. It is specially useful on farms where 'Cetavlon' is being used as part of the prophylactic measures against *Str. agalactiae* infection. A 0.1% solution of 'Cetavlon' is prepared by mixing 1½ fl. oz. (35 cc.) of the Concentrate with about 1½ gallons of water (7 litres). This quantity will be found sufficient to wash the udders and teats of about 50 cows. It is not necessary to change the solution at all during the washing, as 'Cetavlon' remains effective against *Str. agalactiae* organisms even in the presence of quite large proportions of milk. When 1% solutions are required, 'Cetavlon' Concentrate should be diluted in the proportion of 1 part to 19 parts of water.

#### 'CETAVLON' UDDER CREAM

'Cetavlon' Udder Cream contains 0.5% 'Cetavlon' in an emollient cream base. It is an invaluable preparation for eliminating *Str. agalactiae* infection in chaps, fissures and cuts commonly found on the cow's teats, and facilitates healing.

*Method of Application:* The cream is rubbed well into the teats after each milking until the lesions have completely healed and disappeared.

#### 'CETAVLON' JELLY

'Cetavlon' Jelly contains 1% of 'Cetavlon' in a colourless, pleasantly perfumed base, specially prepared for application to the skin.

*Indications:* As a lubricant and protective application for the skin of the operator prior to making rectal and uterine examinations. 'Cetavlon' Jelly may also be employed in the extemporaneous sterilisation of the hands and in the treatment of wounds and burns. It will be found especially useful for obstetric work on small animals.

*As a Protective Application:* The hands and arms should be smeared with sufficient of the Jelly to cover the whole surface and allowed to dry. The film remaining exerts antiseptic and deodorant properties and is non-irritant to the user and to the mucous membranes of the animal. After a rectal or intra-uterine examination any secretions and adherent matter may be easily removed by washing with water, leaving the hands clean and free from objectionable odour.

*For Sterilising the Hands:* Prior to surgery the Jelly should be rubbed well into the skin, the hands moistened with water, and rubbed together; they should then be rinsed with water and dried on a sterile towel. This procedure effectively removes all particles of dirt and leaves the hands sterile.

*Treatment of Wounds and Burns:* The Jelly is applied to abrasions, wounds and burns and this is followed by washing the affected area. A further application of the Jelly is then made so as to form a protective covering.

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## SOUTH AFRICAN BUREAU OF STANDARDS POULTRY FEEDS

Hens, ducks and particularly chickens and poults need very carefully balanced diets, if they are to do well.

Some time ago the South African Bureau of Standards, in consultation with government officials and representatives of the balanced feed mixers

and the South African Poultry Association, drew up comprehensive specifications for nine different mashes, including chicken meal, laying meal and maize-free laying meal.

The underlying idea is that every mash should meet the minimum requirements of the type of bird for which it is intended. The specifications deal mainly with the amounts of protein, fibre, calcium, phosphorus, manganese, riboflavin, pantothenic acid and vitamin A in the mashes, and the Bureau not only makes chemical analyses to ascertain that these substances are present in the right proportions, but actually feeds some of the mashes to chickens hatched and specially reared for the purpose.

The bags in which the mashes are sold have to be free of germs and parasites injurious to poultry.

In future the purchase of poultry mashes will be very simple. All the farmer will need to do will be to buy the cheapest mashes sold in bags carrying the mark of the Bureau—an ellipse containing the letters S.A.B.S.

No firm actually has the right yet to use the mark, because it takes some little time to arrange everything after an application has been received, but it is worth recording, from the historical point of view, that the first firm to apply is the Verecniging Consolidated Mills, the sellers of "Epol" products.

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## BOOK REVIEWS

*PRACTICAL VETERINARY PHARMACOLOGY, MATERIA MEDICA AND THERAPEUTICS.* H. J. Milks. 6th Edition (1949), pp. xiv + 720. Baillière, Tindall & Cox, London. Price 76s. 6d.

In its sixth edition this well-known book has been enlarged by some 30 pages and the addenda to the 5th edition dealing with antibiotics and sulphonamides, have been incorporated in the text.

This progressive increase in size makes the reviewer wonder whether such a comprehensive book on Pharmacology, Materia Medica and Therapeutics is really required by the average student or practitioner. Much of the material incorporated would be more appropriately sought for in works on medicine and diagnostics. For instance, the last chapter headed Biologic Diagnosis, Prophylaxis and Therapy contains descriptions of the mechanisms of defence of the body, hypersensitivity, tuberculin test and the preparation of various vaccines. This chapter adds 37 pages to the book yet is perforce so superficial in character as to be practically valueless.

What is urgently required is a short, concise book dealing mainly with Pharmacology as defined by Milks as being the study of the actions of drugs on living organisms. The essential difference between quackery and the ethical use of drugs lies in a precise knowledge of their action. In the reviewer's opinion pharmacology should be taught as an extension of physiology and the drugs dealt with according to their actions on organ systems. For the student stress should only be laid on substances of present-day therapeutic importance.

With the modern trend towards the use of factory-made preparations

the study of *Materia Medica* is fast losing its importance. A detailed study of the "physical and chemical properties of drugs, their sources and history" is not required.

To return to the book, the general arrangement is unchanged in that the drugs are dealt with partly according to their main pharmacological action and partly according to their chemical composition. This fact frequently interferes with speedy reference, but the reviewer is well aware of the difficulties entailed in both these types of classification.

It is surprising to find the autonomic nervous system dealt with under the chapter headed "Drugs acting upon the Circulatory System" and not under the following chapter which deals with drugs acting on the nervous system. Furthermore the whole autonomic system is discussed in two pages despite the enormous importance of the subject in modern physiology, pharmacology and therapeutics.

It is extremely disappointing to find, in a modern book on *veterinary* therapeutics, no special chapter devoted to the ruminant alimentary tract which presents such peculiar and specific problems. The only reference to the rumen in the index is that of "ruminatoria" under which are listed arecolin, barium chloride, eserine, lentin and tartar emetic. In 1942 Dougherty reported on a series of critical experiments on drugs affecting the motility of the bovine rumen. It was found that barium chloride had a marked depressing action on rumen motility when administered intravenously and no effect when introduced into the rumen itself. Tartar emetic was also found to be an unsatisfactory stimulant of the rumen.

Despite the above mentioned shortcomings the work is a valuable reference volume, but the reviewer feels that a more concise book, better adapted to modern veterinary practice, is required by overburdened student and busy practitioner alike.

R.C.

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*THE THEORY OF INBREEDING.* By R. A. Fisher, Edinburgh and London. Oliver & Boyd, 1949. viii + 120 pp. 10s. 6d.

Animal scientists will agree with the author's statement that the possibilities which the process of inbreeding opens out for the practical improvement of domestic plants and animals should not be neglected, and that both the theory and practice of this subject should form an essential part of future programmes either of genetic or agricultural research.

In this specialized monograph R. A. Fisher, the celebrated statistician and geneticist, gives the theoretical background of his recent researches in this field. These theoretical arguments make difficult reading and probably will be of most interest to mathematical geneticists. The statistical formulations are of the high standard always associated with the author, but presuppose a good knowledge of various mathematical techniques.

The introductory chapter and a final appendix on the function of inbreeding in animal and plant improvement make for easier reading, however, and should be of interest to veterinarians concerned with this field. It is shown that inbred stocks, while in themselves unprofitable, are invaluable as a tool for precisely controlled progeny tests, while in agricultural development their value lies rather as the parent stocks of desirable crossbred combinations.

D.v.D.R.

## OBITUARIES

### FREDERICK CHARLES GAVIN

We regret to announce the death of Major Frederick Charles Gavin, D.S.O., M.R.C.V.S. (London), at Kloof on the 17th October, 1950.

Major Gavin was a foundation member of our Association. He qualified in 1889 and came to South Africa with the Royal Army Veterinary Corps during the Boer War. He was later appointed Head of the Hygiene Department and Veterinary Surgeon to the Johannesburg Municipality.

He again saw service with the Royal Army Veterinary Corps during World War I, when he was awarded the D.S.O. for his services in France and Belgium.

After his retirement from the Johannesburg Municipal services, Major Gavin settled in Natal.

We extend our greatest sympathy to his widow.

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### ALEXANDER SCRYMGEOUR McCHLERY

During September, 1950, the veterinary profession of Southern Rhodesia lost one of its most popular members with the death of Alexander Scrymgeour McChlery. Alec was born in Rhodesia and was educated at Prince Edward School, Salisbury. After leaving school he studied at the Royal (Dick) Veterinary College, from where he qualified in 1929. He joined the Southern Rhodesian Veterinary Service during that year and remained in the service until his death. During 1949 Alec spent some eight months on study leave at Onderstepoort and at the Colleges in the United Kingdom, where he made many good friends. A few months previous to his death he was appointed Assistant Director of Veterinary Services, being in charge of the field side.

Alec McChlery's cheerful, courteous manner and his integrity will be remembered by all with whom he came in contact. He was a true and trusted friend of all members of this veterinary service.

To his widow, Mrs. J. McChlery, and his brother, R. McChlery, this Association extends its deepest sympathies.

H.M.H.

# SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION

*Minutes of Council Meeting held at Prudential House  
on August 24, 1950, at 2.15 p.m.*

*Present:* J. H. Mason (President), A. M. Diesel (Vice-President), G. D. Sutton (Hon. Treas.), J. G. Boswell, H. P. Steyn, P. S. Snyman, S. W. J. van Rensburg, R. Clark (Editor), W. D. Malherbe and M. de Lange (Hon. Sec.).

*Apologies:* P. J. du Toit, R. A. Alexander, G. Pfaff, C. J. van Heerden.

1. *Minutes of meeting held on August 10, 1950.* Adopted.

2. *Arising from these minutes:—*

- (a) *Dog Licences in the Province of Transvaal:* Secretary reported that from 1951 provincial licence fees for bitches would be increased to £1-0-0, irrespective of whether they were spayed or not, licence fees for dogs to remain at 10s. per annum. Decided, Hon. Secretary to write to Provincial Secretary to recommend reduction in fee for spayed bitches.
- (b) *Appointment of Lecturer by Pretoria University:* Secretary reported that no acknowledgment of receipt of the President's letter had yet been received from the University Council, in spite of reminders by letter and by phone. Secretary again telephoned the Secretary of the University Council. Decided that Dr. van Rensburg contact the Chairman or Vice-Chairman of the University Council and to report to the next meeting of Council.
- (c) *Delay in filling posts of D.V.S. and Chairman of the Veterinary Board:* Dr. Diesel reported that it was becoming increasingly embarrassing that the Veterinary Board was not functioning. There were, however, indications that the position would be corrected in the near future. Decided to leave the matter for the next Council meeting.
- (d) *Presentation to Mr. Cloete, former Registrar of Veterinarians:* Secretary reported that Mr. Cloete would be unable to attend the Conference dinner and dance. Decided that Secretary arrange a suitable occasion to make the presentation to Mr. Cloete.
- (e) *Resignation of Mr. S. T. Jackson:* Noted. Decided: since no reply had been received from Mr. Jackson, Council would recommend acceptance of resignation to the General Meeting.

3. *Notification of Election of Council for 1950/51.*

President: S. W. J. van Rensburg.

Vice-President: G. Pfaff.

Honorary Secretary: M. de Lange.

Honorary Treasurer: G. D. Sutton.

Members 1950/52: J. G. Boswell, A. M. Diesel, P. S. Snyman, A. D. Thomas.

President congratulated the above members on their election.

As a result of the election of Drs. van Rensburg and Pfaff to higher office, two vacancies had arisen in Council for the period 1950/51. Decided to request Drs. M. C. Robinson and H. P. Steyn to accept office as Council members for this period.

4. *Standing Committees 1950/51:* The following were elected:—

*Finance Committee:* A. D. Thomas (Chairman/Convenor), R. A. Alexander, W. D. Malherbe, B. S. Parkin, G. D. Sutton (Hon. Treas.).

*Editorial Committee:* R. Clark (Chairman/Convenor), G. P. Bishop, H. P. A. de Boom, E. M. Robinson, P. S. Snyman.

*Library Committee:* J. D. W. A. Coles (Chairman/Convenor), E. M. Robinson, G. D. Sutton.

*General Purposes:* A. M. Diesel (Chairman/Convenor), R. A. Alexander, P. J. J. Fourie, B. S. Parkin, P. S. Snyman.

*Book Fund:* G. D. Sutton (Chairman/Convenor), A. D. Thomas, M. de Lange.

5. *Financial Report, 1949/50:* The examiners' report was discussed in detail and accepted.

Hon. Treas. recommended that all financial work in connection with the Journal be taken over by Mrs. Coles and that she be paid £2-10-0 per month for this, in addition to her monthly salary of £7-10-0. Agreed.

The President paid tribute to Dr. van Drimmelen for his work as business manager of the Journal and would write a letter of appreciation.

Dr. van Rensburg proposed a vote of thanks to the Treasurer, Editor and the two lady assistants for their services to the Association. Unanimously accepted.

6. *Amendments to Veterinary Act:* Minutes of special meeting of Council held on June 26, 1950, adopted.

7. *General:*

(a) *Resolutions Committee:* Drs. Parkin and Malherbe elected.

(b) *Conference Agenda:* Dr. Diesel inquired whether more time could not be allocated for the discussion on Infectious Sterility. It was explained that provision had been made for this.

(c) Suggested that Deans of Faculties of Medicine and Agriculture be invited, with their staff, to attend any of the meetings of Conference. Agreed.

(d) Suggestion that in future it should again be considered to have Conference opened by some Public Official. Agreed.

8. *Correspondence:*

(a) *Letter General Secretary N.V.M.A.,* re proposed Veterinary Codex read. Dr. Parkin elected as corresponding member of sub-committee dealing with action and uses of Drugs, and Dr. Sterne for Biological products.

(b) *Letter Col. J. W. Rainey,* withdrawing resignation, read.

Before closing the meeting the President mentioned that, this being his last meeting, he wishes to thank all members of Council for their support and co-operation.

Dr. van Rensburg on behalf of Council thanked Dr. Mason for the very able way in which he conducted the affairs of the Association during his period of office as President.

The meeting closed at 5.15 p.m.

S. W. J. VAN RENSBURG,  
*President.*

M. DE LANGE,  
*Hon. Secretary.*

## SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION

*Minutes of the 45th General Meeting, held at Onderstepoort  
from Sept. 5-7, 1950.*

*Present:* J. H. Mason (retiring President), A. M. Diesel (retiring Vice-President), S. W. J. van Rensburg (incoming President), G. Pfaff (incoming Vice-President), L. Abrams, T. F. Adelaar, A. A. L. Albertyn, R. A. Alexander, W. H. Andrews, J. A. Badenhorst, W. G. Barnard, N. Barrie, J. G. Bekker, M. Bergh, J. H. R. Bisschop, C. W. A. Belonje, J. G. Boswell, H. N. Botha, J. W. A. Brookes, M. H. V. Brown, P. H. Brown, B. J. Brummer, R. Buchalter, P. H. Bunton, A. S. Canham, F. E. Cavanagh, D. Coles, V. Cooper, L. L. Daly, J. A. de Kock, H. P. A. de Boom, O. T. de Villiers, S. W. de Villiers, G. J. de Wet, C. Dickson, J. L. Dickson, A. R. Doré, J. L. Doré, F. W. B. du Casse, A. J. du Plessis, J. F. du Plooy, P. J. du Toit, R. du Toit, J. Fick, C. Flight, P. J. J. Fourie, F. W. Goodall, M. M. Greathead, H. Graf, J. J. Hamman, L. L. Hansmeyer, R. E. Hartig, F. J. D. Hempstead, M. W. Henning, C. F. B. Hofmeyr, H. E. Holtz, P. P. Hugo, C. E. Isaacs, B. C. Jansen, P. G. Joubert, V. R. Kaschula, A. C. Kirkpatrick, E. B. Kluge, M. J. S. Kropiwnicki, A. B. la Grange, M. C. Lambrechts, F. W. Langbridge, D. A. Lawrence, J. M. W. le Roux, D. J. Louw, R. K. Loveday, C. Maree, I. S. McFarlane, D. L. McWhirter, W. du T. Malan, W. D. Malherbe, M. J. N. Meeser, C. M. T. Meldal-Johnsen, B. Moring, I. Mowat, W. O. Neitz, J. J. Oosthuizen, V. E. Osborn, (Mrs.) S. M. Osborn, R. B. Osrin, B. T. Paine, R. A. Painter, J. W. Pols, B. S. Parkin, E. M. Robinson, J. E. Robinson, M. C. Robinson, K. A. Ross, W. J. Ryksen, G. Schmid, J. R. Scheuber, K. Schulz, J. D. Smit, P. S. Snyman, N. C. Starke, M. Sterne, J. L. Stewart, D. G. Steyn (Prof.), D. G. Steyn, H. P. Steyn, A. D. Thomas, J. A. Thorburn, J. G. Townsend, D. E. Truter, P. L. Uys, W. P. van Aardt, W. G. van Aswegen, N. T. van der Linde, G. C. van Drimmelen, J. S. van Heerden, K. M. van Heerden, G. F. J. van Rensburg, F. J. Veldman, J. H. B. Viljoen, J. S. Watt, C. C. Wessels, W. J. Wheeler, J. G. Williams, K. E. Weiss, M. Zschokke, J. Zwarenstein, R. Clark (Editor), G. D. Sutton (Hon. Treas.), M. de Lange (Hon. Secretary).

*Guests:* Prof. F. N. Bonsma, Drs. R. Bigalke, G. Theiler, R. J. Ortlepp.

*Apologies:* W. L. S. Mackintosh, J. J. G. Keppel, D. M. Walters, H. Nelson, N. J. G. da Camara.

*Tuesday, September 5, 1950.*

8.45 a.m. The President opened the Conference with a welcome to the members and guests present. He thanked the Acting Director of Veterinary Services for once again placing the facilities of Onderstepoort at the disposal of the Association for the Conference.

A motion of condolence with the relatives of the following members who had died during the year was passed, those present standing as a token of respect: Drs. Quin, Simpson, Wadley and van der Westhuizen. Dr. van der Westhuizen had passed away suddenly only the day before the conference, and a telegram of condolence was sent to his relatives on behalf of the Conference in session.

*Notification of Election of Council for 1950/51:* President: S. W. J. van Rensburg; Vice-President: G. Pfaff; Hon. Secretary: M. de Lange; Hon. Treasurer: G. D. Sutton; Members: J. G. Boswell, A. M. Diesel, M. C. Robinson, P. S. Snyman, H. P. Steyn, A. D. Thomas. (Sitting members: R. A. Alexander, W. D. Malherbe).



*Presidential Address:* The retiring President gave his address and in conclusion thanked the office-bearers for their work in the past year.

Assumption of the chair by incoming President Dr. Mason paid tribute to Dr. van Rensburg who had for many years served the Association as Hon. Secretary and thereafter as member of Council, and congratulated him on his election as President.

In his reply, Dr. van Rensburg on behalf of the Association thanked Dr. Mason for the very able way in which he had conducted the affairs of the Association during his four-year term of office as President, and appealed to members for their continued support in furthering the interests of the Association and the profession.

9.30 a.m. Dr. V. Kaschula gave a paper on "The Epizootology of Newcastle Disease and its control by Vaccination." In the discussion which followed Dr. Zwarenstein briefly outlined the vaccination campaign in the Durban area.

10.15 a.m. Major L. L. Daly gave his paper on "Some Veterinary Problems in Natal." In the discussion which followed after the tea interval, Dr. Diesel paid tribute to Drs. A. S. Canham, E. B. Kluge, W. O. Neitz and R. du Toit for their services in connection with the research and eradication of East Coast Fever and Nagana.

11.45 a.m. Dr. G. Pfaff on the "Doping of Race Horses."

After lunch Dr. Alexander, Acting Director of Veterinary Services, welcomed the Trade Exhibitors to Congress. Mr. Taylor, Chairman of the Medical Exhibitors' Association, replied on their behalf.

1.40 p.m. Dr. P. J. G. Louw on the analytical samples of urine and saliva of race horses in connection with doping. This was followed by a discussion on both Or. Pfaff and Dr. Louw's papers, in which Prof. D. G. Steyn made an appeal for extending the term "doping" to embrace substances other than those usually associated with this malpractice.

2.15 p.m. Dr. W. D. Malherbe on "The Management of Drug-resistant Biliary Fever in Dogs." This was followed by a lengthy discussion in which particularly the private practitioners participated.

3.15 p.m. Dr. M. Sterne's paper on "A New Method of producing Clostridial Toxins and Toxoids." Dr. Sterns was warmly congratulated on this notable advance which was done with the able collaboration of Mr. Wentzel.

#### *Wednesday, September 6, 1950.*

8.45 a.m. Prof. P. J. J. Fourie's paper on "Tuberculosis in Man, an Animal (Bovine) Health Problem". A lengthy discussion followed.

10.10 a.m. Dr. S. W. de Villiers' paper on the "Treatment of Gid in Sheep."

10.25 a.m. Dr. A. M. Diesel on "Official Approval for the Testing of Stock and the Certification of Freedom from Notifiable Diseases."

11.30 a.m. Dr. R. Bigalke on "Science and the Conservation of Wild Life in South Africa." The discussion which followed this interesting paper was opened by Dr. M. J. N. Meeser who outlined the aspects of veterinary control in relation to wild life conservation. The President, in conclusion, thanked Dr. Bigalke for his contribution.

1.30 p.m. The afternoon session was devoted to a discussion on the "Diagnosis and Control of Infectious Sterility in Bovines," which was

opened by Dr. P. S. Snymman, who spoke mainly on the epizootology, regional distribution, etc.

Dr. J. G. Bekker dealt with various aspects including the diagnosis, control, ætiology, immunity, pathogenesis, etc.

This was followed by a lengthy discussion, to which the following contributed: Dr. H. P. Steyn on transmission of non-specific infections of the genitalia. Dr. D. A. Lawrence on the appearance of the disease in Southern Rhodesia, pathogenesis, infectivity, etc. Dr. C. C. Wessels on the gross pathological changes. Dr. R. A. Alexander on the specificity of the disease, Dr. W. J. Ryksen on the ætiology, Prof. van Rensburg on the clinical diagnosis, etc. Dr. M. de Lange on the histo-pathological changes and differential diagnosis.

7.30 p.m. A dinner and dance was held at the Union Hotel at which Professor and Mrs. F. N. Bonsma were the guests of the Association.

#### *Thursday, September 7, 1950.*

8.45 a.m. Prof. R. Clark gave a demonstration on Ruminal Bloat, its ætiology and treatment.

9.30 a.m. Prof. K. C. A. Schulz and Dr. G. D. Sutton on Enterotoxæmia, the discussion on which was postponed till the end of the morning session.

10.05 a.m. Prof. F. N. Bonsma gave a paper on "Artificial Insemination from the Breeder's Point of View."

In the discussion Dr. J. G. Boswell explained the organisation of an artificial insemination centre in Johannesburg. Dr. D. A. Lawrence informed the meeting of the formation of an emergency artificial insemination service in Salisbury primarily to combat infectious sterility. Prof. Coles supported Prof. Bonsma in his views on the dangers of transmission of hereditary defects, etc. Dr. F. W. B. du Casse emphasised that bulls should not be used until five years old to enable progeny tests to be carried out.

The President thanked Prof. Bonsma for his illuminating contribution.

12 noon. Prof. van Rensburg gave his paper on "Pitfalls of Artificial Insemination and Sperm Examination."

In the discussion which followed after lunch, Dr. M. Zschokke related his experiences in connection with artificial insemination of Karakul sheep in South West Africa.

2 p.m. President paid tribute to Mr. N. J. Cloete, the former Registrar of Veterinarians and Secretary to the S.A. Veterinary Board, for his valued services to the profession and made Mr. Cloete a presentation as a token of appreciation of the Association.

President then thanked the trade exhibitors for the trouble they had taken to put up attractive exhibits and expressed the opinion that this had become an important feature of the Annual Congress.

2.15 p.m. *Business Meeting.*

1. Minutes of 44th General Meeting: Adopted.

2. Arising from these minutes:—

(a) *Membership Australian V.M.A.:* Secretary reported that, due to certain formalities, Dr. Malherbe had not yet been elected to membership of this body. Decided to leave the matter to the judgment of the Secretary and Dr. Malherbe.

- (b) *Resolution No. 1 of 44th General Meeting*: Secretary read the reply received from the Secretary for Agriculture in which it was stated that an officer of the Department had been to the U.S.A. to study poultry diseases and had been charged with the opening of a new regional laboratory in Cape Town, dealing almost exclusively with poultry diseases and the B.W.D. blood test. In addition a further officer, who qualified at the end of 1949, had been appointed to assist in the Newcastle Disease campaign in Johannesburg, and would eventually be transferred to Onderstepoort to assist in the Poultry Section.

It was pointed out to the meeting that the latter officer had since resigned from the service.

- (c) *Resolution No. 2 of 44th General Meeting*: Reply received from the Secretary for Agriculture was read, in which it was stated that research was being carried out on the ætiology, diagnosis and transmission of Infectious Sterility and that a survey was being carried out to ascertain the distribution of the condition in the Union. Furthermore, that regulations had been drafted concerning the control of movement of animals from infected areas.

3. *Election of New Members*: The following were declared elected: L. Abrams, P. H. Bunton, D. J. de Waal, N. P. Dison, R. Every, H. Halenke, J. M. Huyser, G. E. Lay, A. Littlejohn, W. L. S. Mackintosh, D. L. McWhirter, A. C. Maree, C. J. Muller, J. J. Oberholster, J. Robinson, J. H. D. Snyman, R. A. Solomon, K. M. van Heerden, H. C. Watson.

4. *Resignation*: That of Mr. S. T. Jackson was accepted with regret.

5. *Reports of Standing Committee*:

*Finance*: President requested the Hon. Treasurer to read his report. Dr. Sutton drew the attention of members to the balance sheet which had been circularised to all members and thereafter presented the Auditor's Report for 1949/50. He dealt in detail with the Book Fund, Journal and Subscriptions and concluded that the finances of the Association were in a healthy state.

He also reported that the income and expenditure balanced each other for the past year, but increased costs had to be met in connection with the Journal, clerical assistance to the Secretary and Treasurer, etc. Hence the following motion of alterations to the Constitution, which had been duly circularised to all members, was proposed by Dr. Sutton, on behalf of Council:—

1. Rule 3(b) to read "Any ordinary member may at any time become a life-member by one payment of thirty-five guineas, in lieu of future annual subscriptions."
2. Rule 7(a) to read: "An ordinary member shall pay, on election, the sum of three guineas, which shall be the first annual subscription. After the first year or portion thereof an annual subscription of three guineas shall fall due on the first day of April in each year. No member whose fees are in arrear for more than 12 months shall be entitled to the privileges of the Association."

The motion was seconded by Dr. Boswell, and after some discussion was carried.

6. *General:*

- (a) *Papers for Annual General Meeting:* Dr. Clark suggested that papers to be presented at the Annual General Meeting be published in the Journal prior to the meeting, to enable members to study the subject. Thus the time allowed for each paper on the Agenda could be used for discussion. Dr. Clark pointed out that papers would have to be presented for publication four months ahead of the Conference. After discussion decided that Council examine the proposal and, if feasible, to consider its adoption for future meetings.
- (b) *Refresher Courses:* Dr. S. W. de Villiers requested that although refresher courses had, on account of lack of support, been abandoned for this conference, Council should again consider the matter with a view to arranging for such courses in future.
- (c) *Weed-end closing of Garages:* Dr. Hofmeyr proposed that the Motor Traders' Association be requested to arrange for facilities to be made available to effect repairs to veterinarians' cars, at reasonable cost, when breakdowns occur during week-ends. Agreed.
- (d) *Classified List of Veterinarians in Telephone Directories:* This matter was again brought up. It was pointed out that the Postmaster-General was not prepared to accede to Council's proposals some years back and that any further representations at this stage would probably fail, hence it was agreed to let the matter drop.
- (e) *Entertainment during Conference:* Dr. D. J. Louw expressed his appreciation and thanks to Dr. Parkin for organising a day's golf for members during the Conference, and proposed that Council should consider the extension of the sporting and social side of the Annual Meeting. Agreed.
- (f) *Dr. Maud Bales Scholarship:* President announced that Messrs. I. R. Banks and C. H. van Niekerk, final year B.V. Sc. students, had been awarded the above scholarship for the current year.

7. *Resolutions:* The following were proposed and, after discussion, passed:—

*Resolution No. 1:* Proposed by Dr. Diesel, seconded by Dr. Williams:

"This 45th Congress of the S.A.V.M.A. wishes to express its appreciation to the Honourable the Minister of Lands for approving the appointment of a biologist on the staff of the National Parks Board of Trustees, and it hopes that, when the time is opportune, a post for a veterinarian on the establishment staff of the Board will also be approved."

*Resolution No. 2:* Proposed by Dr. Hofmeyr, seconded by Dr. Pfaff:—

"This 45th Congress of the S.A.V.M.A. wishes to express its appreciation to the S.A. Agricultural Union and its Provincial Unions for the efforts they have made and are making to improve the conditions of service of veterinarians in the Division of Veterinary Services and consequently the recruitment of officers to the Division. This Congress realized that the failure to make good the wastage in the service over the last ten years does, and will in the future, detrimentally affect the provision of veterinary services to the rural community."

*Resolution No. 3:* Proposed by Dr. Hofmeyr, seconded by Dr. Pfaff:—

"The 45th Congress of the S.A.V.M.A. wishes to bring to the attention

of the Honourable the Minister of Agriculture its concern at the state of affairs in the Division of Veterinary Services with the effect on the Veterinary Profession and the livestock industry as a whole—which has resulted from the delay in filling the posts in the Division which have become vacant, including particularly that of Director of Veterinary Services and the twenty senior posts on the establishment which were approved more than a year ago.

“This Congress also wishes to draw the attention of the Honourable the Minister to the fact that the Veterinary Board has been unable to function for a considerable period due to the delay of the Minister of Agriculture in appointing a chairman to the Veterinary Board.”

*Resolution No. 4:* Proposed by Dr. Jansen, seconded by Dr. Thomas:—

“As the present ruling of the S.A. Kennel Union is that only dogs cropped outside the Union of South Africa are permitted on shows under its control; and as the operation of cropping when performed by a veterinarian under anæsthesia in properly selected age groups is no more severe than other operations permitted by the S.A.K.U., it is the feeling of this meeting of the S.A.V.M.A. that the Council of the S.A.V.M.A. be asked to open negotiations with the S.A.K.U. also to permit dogs to be exhibited at the shows under its control when cropped by veterinarians in South Africa.”

The meeting closed with a unanimous vote of thanks to the chair, to which the President replied and in turn thanked the other office-bearers, the Honorary Secretary, Honorary Treasurer and Editor of the Journal for organising the meetings and for their services during the past year. He also expressed his appreciation to the Acting D.V.S., Dr. Alexander, for his extension of the hospitality of Onderstepoort for the venue of the three-day Congress.

Dr. Alexander in replying remarked on the high standard of the papers presented.

The meeting adjourned at 3.45 p.m.

S. W. J. VAN RENSBURG,  
*President.*

M. DE LANGE,  
*Hon. Secretary.*

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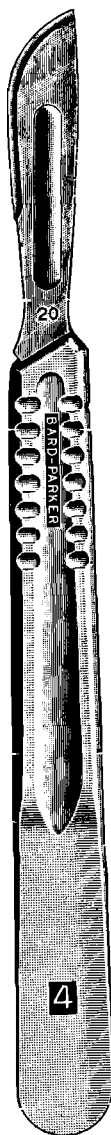
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