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PRETORIA

A MEMORIAL TO SIR ARNOLD THEILER.

Sir Arnold Theiler was a rare person. It would be a simple though lengthy matter to catalogue his achievements and to enumerate the rewards and honours showered on him. That, however, would make of his memory something remote and featureless: a collection of medals, a medley of titles and degrees. These things do not recall to us the dynamic and forceful man that set to his colleagues and his students an example of strenuous and disinterested work. He was a member of most famous scientific societies; his own and other governments recognized and rewarded him, and he received — a magnificent tribute to his greatness — the support and devotion of his staff.

To his memory an honour has been done unique in the history of South Africa. So outstanding has been his service to this country of his adoption that the Government has built a monument at the laboratory he founded.

On Wednesday, the 15th November, 1939, before a large and distinguished gathering, General the Rt. Hon. J. C. Smuts, Premier of the Union of South Africa, unveiled the statue of Sir Arnold Theiler. "Today," said General Smuts, "is not a day of mourning, it is one of rejoicing. We have come to honour the memory of one of the foremost men in the history of Science in South Africa. We are filled with a spirit of rejoicing and appreciation. South Africans have been prone to honour men who held high public posts in their country — statesmen, soldiers and outstanding public men. Sir Arnold Theiler, however, was not in the public eye, he did not occupy a high position. He was a scientist pure and simple and belonged to a branch of science which had no high honour attached to it — veterinary science. But," continued General Smuts, "this veterinary doctor made himself, this institute, this country, famous all over the world. More than any other man he has put South Africa on the science map of the world."

General Smuts touched on the history of Theiler. He mentioned how General Botha had encouraged the young Swiss veterinarian and how, realizing the vast importance of scientific work, he had sponsored the grant for the erection of the institute at Onderstepoort.

"There are many young men today who must have a vivid recollection of Sir Arnold Theiler's great personality. He was sincere, straight, powerful, enthusiastic, devoted to his work. Many books have been written on his scientific discoveries. There is not an animal disease of any importance which he did not tackle. Every time he found out the cause, the source and nearly always the cure. He worked on such scourges as East Coast

fever, redwater fever, horse sickness and blue tongue. And when we realize how seriously these diseases have retarded our progress in the past, we understand what a benefactor he was.

" It was not hard work alone which took Sir Arnold Theiler to the heights; he possessed that added something possessed by so few, ' the genius of insight '. And yet, in spite of his greatness, he was a simple soul; he lived and worked for his profession; money and fame meant nothing to him.

" We should be proud of this institute of Onderstepoort which he built up," said the Prime Minister. " The men here merely do their jobs. They are not politicians and I hope they never will be. They are here to carry on the scientific tradition of devotion to duty which he began."

The statue, made in granite quarried near Pretoria, was sculptured by Mr. Coert Steynberg. It stands on a grass plot in front of the main building — the first permanent laboratory building erected at Onderstepoort.

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Tuberculosis in the Cape Kudu.

By J. A. THORBURN, Grahamstown, and A. D. THOMAS, Onderstepoort.

The area where this investigation took place is the home of the Cape kudu. It is well defined and consists of the bush-covered, broken slopes of the ranges of hills that flank the Great Fish River and its tributaries. It is also known as the "Grahamstown Bush Veld" and includes the northern half of Albany District and portions of the Peddie, Victoria East, Fort Beaufort, Bedford and Somerset districts, and merges in the west with the Addo Bush. The area is fairly warm and dry, the rainfall averages 12 – 15 inches per annum, and extremes of cold are exceptional. The higher grass lands of Grahamstown to the south and the coastal belt beyond have a much heavier rainfall, 25 inches and more per annum, so that the humidity in the Fish River valley is probably fairly high in spite of the low precipitation.

The vegetation is quite characteristic and is sharply demarcated from the higher grass lands at a well-defined altitude on the northern aspect of the Grahamstown Range. The bush consists of shrubs and dwarf trees such as "Spekboom," "Bitter blaar," "Four Corner," "Haakdoorn," "Witgat," "Stinkbos," "Wilde pruim," Sneeze Wood, Wild Olive, "Noors," euphorbia, aloes, innumerable succulent plants, "vygies," and "karroo," with the first-named predominating. Grass is present only on or near the higher hill tops. Nearly all these bushes are edible and form the main source of pasture. The bush, of course, forms excellent cover and is eminently suited to the kudu, which is essentially a bush and tree feeder. Except in the deeper kloofs, most of this bush is only about 5 to 12 feet in height and not very dense, being criss-crossed by innumerable narrow paths, which render almost every bush or tree accessible for grazing. The vegetation is almost evergreen and, in its variety, represents a vast reservoir of food and succulence in times of drought. The condition of stock generally shows that the food value of the grazing is high. Aphosphorosis is evidently unknown.

It is not surprising, therefore, to find that cattle do very well in this region, and that, besides kudu, there is also a large variety of other wild animals such as bushbuck, duiker, steenbuck, rhebuck, oribi, bush pig, baboon, and the lesser carnivores.

The farms are still large, 1,500 – 2,000 morgen, and often two or more are held by the same owner. The soil is extremely shallow and rocky and, except in the irrigated valleys, is totally unsuited for agriculture. Ranching is therefore the only suitable type of farming. The water supply is some-

what inadequate and in parts even precarious. Numerous boreholes have been sunk and these supply more or less brackish water. There are a few springs, but most farmers have now built storm-water dams for their stock in the various camps. Characteristic of this region are the numerous pans situated on the hill tops. They have usually an impervious shale bed and are capable of holding rain water for months. Such dams and pans constitute the main and common source of drinking water for cattle, kudu, and other animals, when the river itself is too far away.

The Cape Kudu and its History.

Old inhabitants state that kudu were exceedingly rare in the whole Fish River valley towards the end of last century. At this time, two or three farmers in the vicinity of Fort Brown were already doing their best to protect the few survivors. In 1892 the Koonap Farmers' Association actually petitioned the Provincial Authorities and obtained complete protection for the kudu. Since then the kudu have increased in numbers and have spread over a wide area on both sides of the Fish River. They are found in greatest numbers on farms which are not overstocked, that is those on which good veld management is practised, and where they are not continually hunted. It is quite usual to find them in the farmer's best camps which are reserved for fattening slaughter stock. Kudu usually run in small herds of five to ten animals, but at times 40 to 60 may congregate. When undisturbed they will remain on the one farm and breed there. If, however, they are pursued by man or dog they immediately leave the neighbourhood, jumping fences 6 feet high with the greatest of ease, and travel many miles and may traverse several farms before they take cover again. Farmers complain that kudu, especially the young animals before they are able to clear the fences properly, do a lot of damage to costly jackal-proof fencing by throwing themselves against it. Lucerne farmers state that kudu pay unwelcome attention to their lands at night.

As regards disease, farmers in this area nearly all agree that kudu have been suffering from "boils" or abscesses of the throat glands for the past ten years. Some go so far as to state that they saw or heard of this condition 15 and even 20 years ago. Most farmers are well acquainted with the condition, but it is only in the last ten years or so that they have come to associate it with bovine tuberculosis.

Tuberculosis in the Kudu.

Payne and Martinaglia (1928) were the first to describe the disease in the kudu and to show that the bovine tubercle bacillus was responsible for the infection. However, the disease in the kudu presents many peculiarities not seen in the bovine.

Course of Disease.

In the majority of recorded cases as well as from farmers' observation it appears that the first evidence of the disease is a swelling of the parotid

lymphatic gland on one or both sides. This swelling becomes progressively larger, softer, and ultimately fluctuates and discharges a thick creamy pus, like an abscess. Instead of healing, however, pus drips from such abscesses for long periods, if one can judge by the indurated state of some of the glands and their persistent fistulous openings. (cf. actinomycosis, see later). From the parotid lymphatic gland infection evidently spreads to the mandibular, retropharyngeal, and the chain of cervical lymph glands along the neck. Sometimes the prescapular and ultimately the bronchial, mediastinal, mesenteric, periportal and other lymphatic glands may be involved. By this time the disease assumes the generalized form of miliary and tubercular pneumonia and pleuritis with "grapes," tubercular hepatitis, and splenitis, etc. There is always the same tendency to form a soft creamy pus rather than the usual caseous, granulomatous conglomerates of bovine tuberculosis. The swollen glands may be as big as an orange or even larger. Farmers assert that they may become twice that size, when they are noticeable even at a distance. Farmers also describe what are known as "roarers." These are usually old animals which can be heard emitting roaring or snoring sounds during respiration owing probably to pressure on the larynx by the swollen retropharyngeal lymphatic glands. It is also curious how little the condition of infected kudu is affected. Even in cases of advanced generalised tuberculosis one would scarcely call the animals emaciated, and certainly those seen did not show any difficulty in keeping up with the herd.

The decomposing carcases of several kudu were seen in the veld on several occasions. It is probable that most of them died after being shot, that is wounded and lost. We were informed, however, that carcases with obvious lesions had actually been found in the veld and that some showed marked emaciation. It would appear that older animals are more commonly affected than young ones and bulls more often than cows. The youngest kudu said to have been found with the disease was a calf under a year old.

Post-mortem Lesions.

The three cases obtained during the present investigation, a full-grown bull and two young cows, revealed the following features which, according to local farmers, are typical of the disease in that area. These kudus came out of the bush with the others and were shot at random, as they came, and in no way did they show signs of illness or of lagging behind the herd. The condition of the bull and one cow was fair, but the younger of the two cows was visibly emaciated. This state was apparently due more to an old

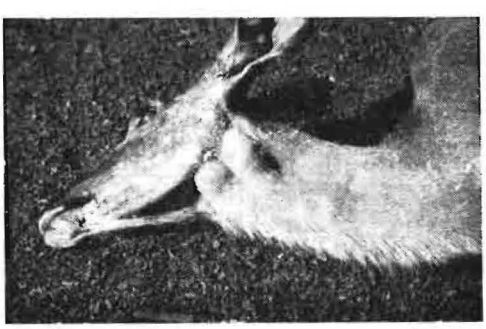

Fig. 1. — *Kudu cow — typical swollen parotid and mandibular lymphatic glands.*

healed fracture of one hind leg than to tuberculosis.

Externally all three animals showed obvious multiple, rounded, soft swellings in the region of the throat and neck. All showed slight caking of the hair with dried pus below the ear on one side of the head — the bull had a fairly big area of skin in this region smudged with sticky pus and dirt. The crusts covered the external opening of a small fistula leading into the pus-filled cavity of the parotid lymphatic gland from which pus could be squeezed out. The other glands of the throat and neck on one or both sides (mandibular, retropharyngeal, cervical and even prescapular) were all enlarged in varying degrees, were soft to the touch, and contained a similar creamy pus. All three animals had large tubercular abscesses in the bronchial, mediastinal, and mesenteric glands. The periportals of two were affected, and the liver of one of these only. Two of the kudu had extensive miliary lesions in the lungs and the other showed localised consolidation and abscesses. One cow in addition had a very extensive tubercular pleuritis with typical "grapes." See Fig. 1 to 3.

Fig. 2. — *Kudu bull — black speck below ear is a discharging fistula.*

Fig. 3. — *Advanced tubercular pleuritis with grapes in a kudu.*

Microscopic Changes.

Microscopic examination of affected tissues revealed the following changes. Fibrous tissue is not as predominant as in bovine lesions; there is a tendency to encapsulation rather than to induration. Langhanse giant cells and epithelioids are exceedingly numerous in certain organs while in others they are very rare. Necrosis or caseation is very diffuse and shows a preponderance of neutrophiles which are often grouped together to form nests or pus centres. In the latter, very small eosinophile actinophytes similar to the ray fungus or club colony of actinomycosis are sometimes seen. Calcification is never extensive nor very advanced. Acid-fast bacilli can

be demonstrated in affected tissue with relative ease. Thus the histological picture, except for the differences noted, resembles that of bovine tuberculosis fairly closely.

Points of Difference between Tuberculosis in the Kudu and in Bovines.

Nevertheless there are certain features which even at this early stage call for comment.

In the first place the regularity and frequency with which primary lesions are set up in the parotid lymph gland just below the ear is entirely different from the usual primary infection of bovines, namely the retropharyngeal and bronchial glands. From the assumed direction of lymphatic drainage it seems impossible to reconcile the parotid lesion with oral infection. The only path which would seem to fit the case is infection through the skin of the ear. It is necessary then to consider in what way penetration of the ear skin could be brought about. The ear of the kudu is very large and its inner skin is relatively smooth and hairless. It could be scratched by the very sharp hooked thorns of the " haakdoorn " abounding in this region. Then again ticks, and the irritating horn fly (*Lyperosia*) are known to attack the kudu — the latter may even cause raw patches on the inner surface of the ear. As a result of such irritation the animal may scratch its ear with its hind foot or claw and in so doing carry infected soil to the broken skin of the ear.

Although unsupported by any direct evidence, this hypothesis seemed sufficiently attractive to warrant an experiment. Two young Afrikander bovines were infected through the skin of one ear with pus from a kudu. In the one the material was injected subcutaneously; in the other the inner skin of the ear was scarified and pus mixed with mud rubbed on. Just before the injection the two animals were subjected to a tuberculin test and gave a clearly negative reaction.

Three months later both animals gave a positive reaction to the tuberculin test. In addition, the scarified one showed a large soft swelling of the parotid lymphatic gland just below the treated ear, and the site of scarification showed an oval indurated swelling about 3 cm. long. (Fig. 4.) In the other animal no lesions could be detected in the ear or in the regional lymphatic glands. These two tollies are being kept under observation for some time still in order to follow

Fig. 4. — *Bovine — swollen parotid glands after artificial infection of scarified ear with pus from a kudu.*

the course of the disease.

Two points emerge from this preliminary experiment. —

A. — The tubercule bacilli from the kudu (already shown to be of bovine type) are now also experimentally demonstrated to be highly infectious for bovines.

B. — That the parotid gland lesions in the kudu indicate that infection takes place probably through the skin of the ear.

Then there is a peculiar tendency in the kudu to soft abscess formation, instead of the hard, fibrous, cheesy and calcified tubercular lesions seen in bovines. Histologically this soft purulent nature of the caseous mass is explained by the greater proportion of neutrophiles present, which appear to congregate around the actinophytic colonies much as they do in actinomycosis. The significance of these small ray fungus colonies can not at this stage be explained.

Tuberculosis in Other Game.

In their publication, Payne and Martinaglia describe one case of generalised tuberculosis in a duiker which they were fortunate enough to capture alive. Several farmers said that duikers in this area show unthriftiness and are liable to die off in appreciable numbers. One man in the Fort Beaufort district has a small private park of some 20 acres in which he kept about 60 springbuck, some duikers, three bushbuck, and hares. During the last two years he states he has lost all but nine springbuck; the duiker and bushbuck have all perished, while the hares are greatly reduced. On being questioned as to the cause of death he stated that they died in ones and twos at odd times and that in those he opened he found abscesses in the lungs. On a farm reputed to be very badly infected with tuberculosis the owner said that on three occasions his herd boys had reported finding dead bush pigs. He was unable to give the cause of death, but thought that the bush pigs, being scavengers, had possibly devoured the infected carcases of kudu in the bush.

Tuberculosis in Cattle.

It has been mentioned already that the whole of this area is essentially a cattle-raising one, and that many of the farmers are stud breeders or keep greatly improved stock. Where the veld is suitable, black-head Persian sheep, and goats do very well. One disease farmers complain about is heartwater, and this they seem now able to control fairly well by regular dipping. Apart from this the so-called "Bushveld disease" has been causing more and more losses during the past five years or more. This disease is no other than tuberculosis. There is no question about its prevalence here, but no reliable or direct information could be obtained about its incidence.

An analysis of the local abattoir (Grahamstown) condemnation for

tuberculosis during the past two years does not suggest a particularly heavy incidence; but does indicate that the disease is fairly widespread.

It must be stressed, however, that the abattoir figures are a poor criterion since the farmers on whose farms this disease is prevalent do not send cattle to the local abattoir but sell them to cattle dealers from other centres. In any case, they are familiar enough with the advanced form of the disease to kill infected animals themselves. One farmer admitted having recently had 16 slaughter cattle condemned out of 36. So far, no tuberculin testing has been carried out in the Fish River area; so that the incidence of tuberculosis in ranching herds is a matter of conjecture. If admissions made by some farmers are taken into account, the percentage of infection on certain farms must be very high.

The association by farmers of tuberculosis in their cattle with the disease in kudu is quite recent. It has given rise to a conflicting state of mind: while some farmers wish to have the kudu destroyed, others wish to hush up the whole matter for fear of earning the reputation of having a tuberculosis-infected herd. This feeling is so strong that many farmers will not hear of tuberculin testing their cattle and would only consent to an investigation of the position in their kudu with the greatest reluctance.

Discussion.

From all accounts tuberculosis both in kudu and in cattle has been in existance in this area for at least 20 years. Some farmers believe that the disease was introduced with cattle from the Cape soon after the ban on moving such stock was withdrawn. It will probably never be known how and when the first kudu became infected, but it seems reasonable to suppose that this happened when the kudu increased on farms on which tuberculosis in cattle was rife.

Martinaglia (1928) showed that the disease in kudu was caused by the bovine tubercle bacillus and this was confirmed in the present investigation.

Because the water supply in this area is limited and used by both the cattle and kudu, one is naturally tempted to incriminate the drinking places as the sources of infection. In order to make an attempt at settling this point samples of water were collected from four dams in the worst camps. Inoculation of guinea-pigs failed to give positive results.

In spite of the wide open spaces, the sunlight, fresh air and total absence of stabled conditions, obtaining for both cattle, kudu and other game, there are probably a number of factors favouring the dissemination of tuberculosis. Amongst these must be mentioned —

(a) the bushy nature of the vegetation affords shade and protection to bacterial life and at the same time forces both cattle and kudu to walk along common paths during grazing and to watering places;

(b) the humidity of the atmosphere (see profuse lichen growth on trees and bushes) notwithstanding the low actual rainfall;

(c) the common drinking places, often no more than mud holes, into which surface rain water from neighbouring pastures drains;

(d) the intermittent discharge over long periods of infective material from fistulous glands on to vegetation, along paths, and into soil and drinking water.

It will be appreciated that the kudu not only constitutes an additional susceptible animal, but it also acts as an active disseminator of virulent matter both by reason of its habits and of the peculiar way in which it reacts to the disease. In other words, the kudu becomes an open case of tuberculosis (discharging glands) long before the disease is generalised, and when it does become generalised, the lung, intestine and other excretory organs merely become additional routes for the dissemination of bacilli. One infected kudu therefore is a far greater danger than an infected bovine, because:

(a) it discharges infective material over long periods, and

(b) it knows no farm boundaries and may carry infection to one or more clean farms perhaps miles away from the original source. This aspect is bound to be accentuated, for a time at any rate, when farmers start (as they have done already) to shoot and scatter these animals far and wide over the bush country.

Last but not least the presence of tuberculosis in this area, as anywhere else, whether in cattle or in kudu, is a menace to the health of the public (use of kudu meat for *biltong*). Tuberculosis has hitherto been associated with the urban type of dairy farming and those of the public who are aware of this can boil their milk. Cream and butter, however, are consumed raw and the most important sources of these commodities are the vast ranching areas of the Union which have so far been confidently regarded as free of tuberculosis. What is going to happen when it becomes known that cream, with tuberculosis bacilli nicely concentrated by the separator, finds its way day after day to our central creameries from infected herds such as these?

Summary.

(1) The so-called Bushveld disease of kudu and cattle is described and shown to be caused by the bovine tubercle bacillus.

(2) The peculiar distribution of the lesions in the kudu is shown to be due, in all probability, to infection *via* the skin of the ear.

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The Influence of Arsenical Compounds on the Development of *Rickettsia ruminantium.*

By W. O. NEITZ, Onderstepoort.

Introduction.

The organic arsenic compounds and the combined arsenic-antimony preparations have specific actions on various groups of micro-organisms: spirochaetæ, spirilla, bacteria, protozoa. Very interesting observations, which are mentioned in the appended table, were those made on the *Bartonella* and the *Eperythrozoa.*

The first record of the action of the organic arsenic compounds on *Bartonella muris* is that of Mayer, Borchardt and Kikuth (1927). They state that this therapy must be looked upon as a *"therapia sterilisans magna"* in the sense of Paul Ehrlich. Yoshiwara (1931), who tested the effect of stibosan on *B. muris*, showed that this compound had a very low chemotherapeutic index. Hans Schmidt* prepared several arsenic-antimony compounds, which on testing in *B. muris* infections of rats [Uhlenhuth and Seiffert (1931) and Kikuth (1932)] showed remarkably high chemotherapeutic indices of 1 : 400 – 1 : 5000. The specific action of several of these compounds on other species of *Bartonella* and three species of *Eperythrozoa* are given in the table.

In view of certain analogies that exist between the *Bartonella* and the *Rickettsiae,* Ronse (1935) tested the effect of Std. 386B (an antimony-arsenic compound) on *Rickettsia prowazeki.* He stated that a beneficial influence on the course of this disease in guinea-pigs was observed. This observation prompted the author to test the effect of the drug Std. 386B and neosalvarsan in heartwater (*Rickettsia ruminantium*) of sheep.

Observations.

For these tests highly susceptible merino sheep artificially infected with heartwater were used. Five sheep were injected intravenously with neosalvarsan (0·45 gm.) on the first or second day of the reaction, and eight sheep were inoculated intravenously with Std. 386B (1·0 gm.) on the first or second day of the reaction. No beneficial effect was observed. On the contrary, the nervous and other clinical symptoms were more pronounced and the duration of the disease, which invariably terminated in death, was considerably shorter than was usually observed in untreated

* Quoted by Kikuth (1937) in reference.

sheep. The examination of intima smears prepared from the jugular veins, according to the technique described by Jackson (1931), showed that rickettsia colonies consisting of very small or large granules were extremely numerous. In well-prepared smears 1 to 5 colonies per field were found. The size of the colonies varied from approximately 5 to 15 μ in diameter. In the smears prepared from the endothelial cells of the jugular veins of untreated sheep the rickettsia colonies were seldom as numerous.

Discussion.

The provocation of parasites by chemotherapeutic agents has been observed in several diseases. A latent infection of malaria in human beings can be activated by neosalvarsan. Monkeys (*Macacus* and *cercopithecus* sp.) suffering from *Piroplasma pitheci* infection can be cured with trypan blue. In these animals, however, a latent infection of *Plasmodium knowlesi* can be activated by trypan blue. In *Eperythrozoon ovis* infection of sheep, Neitz (1937) showed that these parasites disappeared very rapidly after the administration of neosalvarsan and Std. 386B, whereas the above experiments show that these drugs stimulate the development of *Rickettsia ruminantium* in sheep suffering from heartwater. How this is brought about is not clear. In the case of a latent infection of *Pl. knowlesi* trypan blue is supposed to block and throw out of action the reticulo-endothelial system, resulting in the exacerbation of the multiplication of the malarial parasites. However, the drugs under discussion do not block the R.E.S., and their action on *R. ruminantium* is probably either a direct stimulating action on the development of the parasites or on the cytoplasm of the endothelial cells of the blood vessels, which is rendered more suitable for the multiplication of the parasites.

Conclusion.

1. The specific action of the organic arsenic compounds and the arsenic-antimony preparations on the *Bartonella* and *Eperythrozoa* infections is briefly described.
2. Neosalvarsan and Std. 386B stimulate the development of *Rickettsia ruminantium*.
3. *R. ruminantium* cannot always be demonstrated in the endothelial scrapings of the jugular veins of sheep that have died from heartwater. The injection of these drugs, however, facilitates smear diagnosis in sheep used for the biological test.
4. These observations indicate that the use of these drugs and possibly other arsenical compounds are contra-indicated in the treatment of heartwater.
5. Experiments should be carried out to ascertain whether the arsenical drugs have a stimulating effect on other species of *Rickettsia*.

The Specific Action of Various Drugs on Bartonella *and* Eperythrozoon.

Parasite.	Host.	Drug.	Chemo-therapeutic Index.	Result.	Author.	Year.
B. muris.	Rat	Arsalyt	1 : 48	Therapia sterilisans magna.	Mayer, Borchardt and Kikuth.	1927
		Atoxyl	1 : 40	,, ,, ,,	,, ,, ,, ,,	,,
		Neosalvarsan	1 : 72	,, ,, ,,	,, ,, ,, ,,	,,
		Spirocid	1 : 80	,, ,, ,,	,, ,, ,, ,,	,,
		Tryparsemide	1 : 4·8	,, ,, ,,	,, ,, ,, ,,	,,
		Stibosan	1 : 8	Specific action.	Yoshiwara.	1931
		Std. 283	1 : 500.	Therapia sterilisans magna.	Uhlenhuth and Seiffert.	,,
		Std. 246	1 : 500	,, ,, ,,	,, ,, ,,	,,
		Std. 386B	1 : 2500 – 1 : 5000	,, ,, ,,	Kikuth.	1932
					Uhlenhuth and Seiffert.	1933
	Mouse	Neosalvarsan	1 : 5	Specific action.	Kikuth.	1932
B. canis	Dog	Neosalvarsan	?	Therapia sterilisans magna.	Kikuth.	1932
					Regendanz and Reichenow.	1932
B. bacilliformis	Man	Neosalvarsan	0	No specific action.	Arce.*	1910
		Std. 386B	?	Specific action.	Manrique.*	1937
Ep. coccoides	Mouse	Sulfarsenol.	?	Specific action.	Bruynoghe and Vassiliades.	1929
		Neosalvarsan	?	,, ,,	,, ,, ,,	,,
		Tryparsemide	?	,, ,,	,, ,, ,,	,,
Ep. ovis	Sheep	Neosalvarsan	?	Therapia sterilisans magna.	Neitz.	1937
		Std. 386B	?	,, ,, ,,	,,	,,
Ep. wenyoni	Cattle	Neosalvarsan	?	Specific action.	Neitz.	1940

* Quoted by Kikuth, 1937.

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Jl. S.A.V.M.A.,
XI(1) : 15,
1940.

Uleron in the Treatment of Heartwater.

By W. O. NEITZ, Onderstepoort.

A short account of the value of Uleron in the treatment of heartwater was published by Neitz (1939). It was pointed out that the mortality could be reduced considerably by the use of the drug. Of the 41 sheep that reacted to an artificial infection with heartwater 39 recovered after treatment, whereas 26 out of 37 untreated sheep died. A series of tests carried out subsequently has shown that the best results are obtained when the treatment is applied during the early part of the reaction, that is on the first or second day of fever.

At the time when the first observations were published it was not known whether or no Uleron produced a *"sterilisatio magna."* Immunity tests have shown that with very few exceptions, a solid immunity may be expected in the recovered treated sheep.

The results under laboratory conditions have been promising, because the treatment could be carried out early during the course of the disease. Extensive tests under field conditions have not been carried out yet, but it is anticipated that the results will not be as favourable, because by the time an animal is visibly ill the disease is as a rule far advanced. Nevertheless attempts should be made to treat such animals in the hope of saving a few.

The drug is sold in the form of tablets of 0·5 gm. each, and must be administered intravenously in the following way: The drug will only dissolve satisfactorily in a 1% solution of sodium hydroxide, a supply of which should be kept handy in sterile bottles. To every 10 cc. of this 1% solution of sodium hydroxide add 1 gm., that is 2 tablets of Uleron. The dose recommended is 1 gm. for every 50 Kg. body weight. Thus for an average-sized young animal weighing 300 Kg., six gm., that is 12 tablets of Uleron dissolved in 60 cc. of the sodium hydroxide solution should be given intravenously.

Uleron may cause an irritation of the intestine and even methæmoglobinuria. The latter symptom must not be confused with piroplasmosis. In addition, symptomatic treatment should be undertaken wherever possible: for example, astringents for diarrhœa. Drugs containing sulphates must be avoided.

Since few data are available on the use and results of Uleron treatment under field conditions, the writer will greatly appreciate any information on the value of this drug as a cure for heartwater.

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Jl. S.A.V.M.A.,
XI(1) : 16 — 19,
1940.

A Case of Congenital Porphyrinuria ("Pink Tooth") in a Grade Friesland Cow.

By C. H. FLIGHT, Cape Town.

Introduction.

Fourie (1936) described the occurrence of congenital porphyrinuria or "pink tooth" in South Africa, and dealt with a number of cases which occurred in Swaziland. The same writer (1938) described a further case of this condition in a living grade Friesland cow (Cedara case). Flight (1939) described two cases of the same condition encountered in young Friesland heifers in the Cape Province (Ladismith cases).

History.

On 16.11.38, while collecting ticks for the tick survey on Mr. J. Olivier's farm "Welgeluk," Oudtshoorn district, I was requested to examine a cow that had been suffering from an affection of the skin for a number of years. The application of numerous remedial agents had had no beneficial effect.

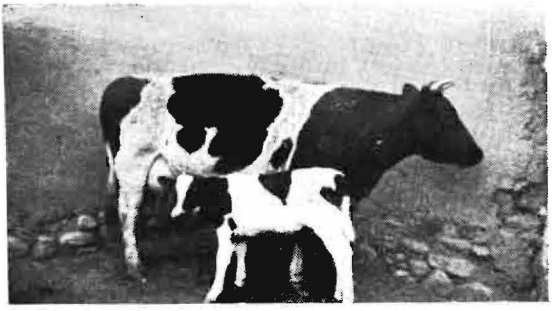
Symptoms.

The cow was a beautiful high-grade Friesland, called Vlermuis, approximately eight years old, in excellent condition, and very heavy in calf. She showed extensive skin lesions on the unpigmented parts of the body that were exposed to the sun, and discoloration of the teeth and urine. A diagnosis of congenital porphyrinuria was made, and was subsequently confirmed by the spectroscopical examination of the urine at Onderstepoort, when porphyrin bands were identified. Towards the end of November she gave birth to a calf, which was examined by me on 20.12.38, when it was a few weeks old. It was found to be a normal high-grade heifer calf, showing no clinical symptoms of congenital porphyrinuria.

On 1.11.39, almost a year after the first examination of Vlermuis, she was again examined by me. She had since calved again, towards the end of September, 1939, producing a high-grade heifer calf, which also showed no clinical symptoms of congenital porphyrinuria, when examined five weeks after birth. The previous calf born during 1938, and now about eleven months old, was again examined. It still showed no clinical symptoms of congenital porphyrinuria, was well grown out, and in excellent condition.

A clinical description of the cow Vlermuis when seen on 1.11.39, on which date the photographs were taken, may be of interest. She was in good condition, with glossy coat, of docile temperament, and in full milk. On superficial examination, except for the unsightly skin lesions, she had the appearance of a normal bovine. The conjunctival, nasal, and buccal mucous membranes were very slightly pale, and there was a very slight mucous discharge from the eyes and nose. The incisors and molars showed a pale pinkish discoloration. The temperature at 11 a.m. was 102·5° F. The pulse and respiration were slightly accelerated, probably due to the heat. Her appetite was good, and the fæces were very soft, due to the succulent food, which consisted almost entirely of green lucerne. The urine had a faint reddish-brown colour.

What struck the eye was the very extensive affection of the skin, confined, however, to the unpigmented parts exposed to the direct rays of the sun. Those parts of the body covered by black hair were entirely unaffected. The lesions were clearly defined, and in no instance did they extend from the white to the adjoining black parts of the skin. In the



Vlermuis with 5-weeks-old heifer calf.

frontal region was a small circular raised area approximately 3 cm. in diameter, consisting of a hard keratinized crust. Very extensive and irregular lesions were present extending on each side of the body over the withers and rump, from the top of the back to the ventral aspect of the abdomen, and continuous in the skin over the spinous processes. The lesions consisted, for the most part, of hard keratinized crusts, with horny, finger-like outgrowths in places. These were probably very old lesions; but, on the lateral aspect of all limbs, younger lesions were present in the unpigmented skin, either consisting of dry soft scabs and crusts, or having the form of raised reddish wheals. No lesions were present on the nose, muzzle, or on the middle of the back, where the hair parts, as was seen in some of Fourie's Swaziland cases.

The skin on the antero-lateral and posterior aspects of the udder (those parts directly exposed to the sun) showed reddening, with small areas

covered with soft scabs and crusts. The teats also showed reddening, and, according to the owner, this cow suffered from sore teats at each calving, and had to receive special treatment. No doubt this condition was due to photosensitization. She had never, however, suffered from mastitis. She was giving approximately 6 gallons of milk daily, was a prolific breeder, and had never been known to abort. In the opinion of the owner, she had again been successfully served since the birth of the last calf. According to the owner, who is a very progressive farmer, she has produced at least six normal calves, the last two of which were examined by me.

Discussion.

This is the second occasion on which a case of congenital porphyrinuria has been described in a cow which produced clinically normal calves, while she herself showed discoloured teeth, porphyrin in the urine, and extensive skin lesions of photosensitization. This case differs, however, from the Cedara case described by Fourie, where the animal in question was a shy breeder, aborted during her first pregnancy, and had to be repeatedly served before she eventually produced a normal calf, which was stated to be the first living calf known to have been delivered by a bovine affected with this disease.

The fact that this cow has probably been suffering from porphyrinuria since birth seems to have had but little adverse effect on her general condition, milking, and breeding qualities, and her economical value to the owner. This is remarkable when it is recollected that she has all her life been constantly exposed to the elements of nature, running both day and night on lucerne lands. Most of the Oudtshoorn milk cows are brought into sheds only for brief periods while milking operations are in progress, but this particular cow has always been milked either in an open kraal or in the lucerne lands.

In the Oudtshoorn District, where the rainfall is only about 10 inches per annum, sunless days are rare and the cattle are exposed to the sun both summer and winter; during the former season the heat is often intense. On the other hand, the sunny climate of the Oudtshoorn District, its fertile soil and rich lucerne lands, have proved ideal for normal cattle. For the last eight consecutive years Oudtshoorn Frieslands have headed the list for milk production in the Union; they are also renowned for their high fertility, and the birth of twins is a common occurrence.

The sire of the two heifer calves out of Vlermuis was the registered Friesland bull Bakenkraal Nico Dankert No. 13446, which animal incidentally died during January, 1939, as a result of extensive actinomycosis involving the head. The question of the hereditary nature of this disease is being investigated by Fourie, but a few remarks may be of interest.

The sire and dam of Vlermuis died some years ago, and as far as the owner could judge, were both normal. Her great-grand-sire was the

famous Preferent Bull "Arend" 6519, imported many years ago from Holland. The latter animal was also grand-sire to the cow "De Goede Laaste," which was the grand-dam of the two heifers encountered in the Ladismith District which were found to be suffering from this disease [Flight (1939)]. It is seen, therefore, that these three cases have a common ancestry. This, however, is not necessarily significant.

Summary.

(1) A clinical case of congenital porphyrinuria in a grade Friesland cow is described.

(2) This is the second case to be described of a cow showing clinical symptoms of porphyrinuria, but giving birth to clinically normal offspring.

(3) The fact that this cow has probably been suffering from this condition since birth, seems to have had but little, if any, adverse effect on her general condition, milking, and breeding qualities.

(4) The evidence here and in the Ladismith cases, suggests hereditary transmission of the condition through a pure-bred Friesland bull.

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1940.

The Slaughtering of Ostriches.

By C. H. FLIGHT, Cape Town.

Introduction.

Although the breeding of ostriches fell off considerably after the slump in 1914, many thousands of birds are still kept in the Oudtshoorn and other districts in the Union. In the Oudtshoorn District alone, where mixed farming is largely carried on, thousands of ostrich chicks are reared annually. Although fluctuations in the price of feathers occur from time to time, on the whole the keeping of ostriches is a profitable side-line, the cost of keeping birds on the rich lucerne lands of Oudtshoorn being negligible.

Not only is there generally a ready sale for the feathers, the eggs, and the dried meat or *biltong,* but during the last few years ostrich skins have been much sought after, for the manufacture of handbags, wallets, tobacco pouches, shoes, etc., and, prior to the outbreak of the present war, thousands of wet-salted ostrich skins were exported annually to Germany and, to a lesser extent, to America. During this period an adult ostrich was worth on a average from £2-10-0 to £3-0-0. The feathers of slaughtered birds would fetch approximately 18/-, the skin £1-5-0, and the *biltong* 12/-.

Slaughtering Centres.

Many thousands of ostriches are slaughtered in the Union every year, not only by individual farmers, for their own consumption; but also, in some districts where large numbers of birds are kept, by speculators who buy the ostriches from the farmers and send them in batches to various slaughtering centres. There are three or four centres in the Oudtshoorn District, the largest one being within a mile or two of the town, on the farm Onverwag, owner T. van Greunen, where over 3,000 birds are slaughtered every year.

This is done principally during the winter months. At this centre, during these months, slaughtering takes place late in the afternoon from Monday to Thursday of each week, Friday and Saturday being reserved for handling of the meat. The owner charges 2/- per head for slaughtering and making *biltong,* which, together with the feathers and skins, are fetched by the speculators and sold.

All the work is done by coloured labourers, mostly women and children, who receive as a rule 6d. for each bird handled. They are also allowed to take away those edible parts of the carcass that are not used for the

preparing of biltong, such as most of the viscera, the wings, tail, diaphragm, and odd bits of meat scraped off the skeleton. The head, portions of the neck and trunk, and the intestines, are disposed of by burial. The bones of the limbs, after removal of the meat, are buried separately, and are dug up after a few months, by which time the adherent flesh has rotted away leaving clean white bones, which are collected, bagged and sold.

Slaughtering.

The ostriches to be slaughtered are driven into a kraal or a fenced paddock. A coloured man or boy, by means of a stick about five to six feet long with a hook at one end, grips the bird at the back of its neck just below the head. The bird immediately rears, is caught at the back of the neck with the right hand and pulled to the slaughtering place, which is generally an adjoining cleared piece of ground.

Fig. 1.

The actual killing is carried out in a rather crude, but effective, manner. The neck just below the head is grasped with the left hand and the beak with the right hand. The head and anterior part of the neck are then rotated in a clockwise direction until the head is completely twisted round. Using the left hand as a lever, the operator jerks the beak downward with the right hand, and thus causes a fracture of the spinal column just behind the head. The neck of the ostrich is extremely sensitive, so much so that the bird can be easily killed by a blow on the neck with a sjambok or stick.

Fig 2.

It is remarkable how helpless an ostrich becomes, when hooked or held by the neck; were this not the case, it would be impossible for a boy of eleven or twelve years of age, unaided, to catch and kill 30 to 40 ostriches (the usual number dealt with during an afternoon) with little danger to himself.

Preparing the Carcass.

The feathers are plucked, and either tied into bundles or placed in bags; the carcass is then skinned, bushes being placed on either side to prevent spilling of blood on to the soil. Each leg is then severed from the trunk by cutting ventrally through the hip joint. The whole limb, together with the large muscles of attachment to the trunk, is hung up in a shed after the shank has been cut off. A few muscles on the postero-lateral aspect of the trunk are also cut off and used for *biltong*. The thigh (or in Afrikaans "*bout*") is extremely well developed in the ostrich, and the above-mentioned muscles are the only ones used for *biltong*.

The following morning the meat is cut into strips and placed in baths or drums containing a mixture to flavour the meat. For every 100 birds, 150lb. salt, 7lb. cloves and 1lb. spice are used. The meat is left in this mixture for approximately 24 hours, after which a thin wire is passed through the end of each strip of meat, and the strips are hung up on strands of wire in the open. After being thus dried for about eight days, the *biltong* is ready for consumption. About 15 to 16lb. are obtained from a bird.

The slaughter centres in the Oudtshoorn District are all situated on farms outside the municipal boundary, and although they are by no means models of hygiene, they are inspected at regular intervals by Divisional Council inspectors, and a certain amount of cleanliness is thus ensured.

No proper ante- or post-mortem inspection is carried out, but as a general rule the meat of birds injured en route to the centres is not used for preparing *biltong*.

SUMMARY.

The slaughtering of ostriches, and the manufacture of *biltong* as carried out in the Oudtshoorn District, Cape Province, are described.

ABSTRACTS.

The Date of the First Use of Guinea-pigs and Mice in Biological Research.

As the guinea-pig and the mouse are indispensable in conducting biological research, the following notes on the original home of the guinea-pig and the first use of both animals in scientific experiments will be of some interest to all of us.

The information under A, B, and C was very kindly collected by Messrs. W. A. Pool and J. Tweedale Edwards, of the Imperial Bureau of Animal Health.

A. — " Magendie reports numerous experiments mostly confined to rabbits and puppies in his book *Elementary Compendium of Physiology*, 1823. In the English translation by Milligan, on page 33, there is a description of research in the vitreous humour in which he used white mice.

In Houlton's " Formulary, etc.," 1828, translated from the 6th edition of Magendie, on page 88 there is mentioned that he has used both a linnet and a guinea-pig for his experiment on the action of cyanuret of potassium and hydrocyanate of potass on animals.

In the Report of the Royal Commission on the Practice of Subjecting Live Animals to Experiments for Scientific Purposes, published in 1876, there is a very interesting historic section. On page 374 of this Report it says that in 1867 Dr. Burdon Sanderson inoculated two guinea-pigs with pyæmic liquids.

There are records of the use of mouse blood as a cure for warts by the colleagues of Hippocrates circa 300 B.C., and in the early 12th century mice were recorded by St. Hildegarde of Bingen as a cure for epilepsy. A manuscript of the middle of the 13th century prescribes a formula for a fumigant as containing fourteen bats and twenty-four mice. Perhaps this cannot be called serious biological experiment. It seems that the use of mice for breeding in laboratories dates from about 1850."

B. — (*From Prof. Chas. Singer, Professor of Biological History at London University.*)

" 1) *Mice.* These were very early in use. Thus John Mayow's *Tractatusquinque medico-physici* Oxford, 1674, contains two figures of mice being used for experiments on the exhaustion of the " nitro-aerial spirit," that is of what we now call oxygen. On March 5th, 1664, Robert Hooke performed experiments on the effect of fixed air on mice. See Birch *History of Royal Society* (1756) Vol. 1, page 428. In the famous second edition of Robert Boyle's *New Experiments .... touching the Spring of the Air* Oxford, 1662, which mentions Boyle's Law for the first time, on page 170 *et seq* are descriptions of experiments on a mouse and there are perhaps others elsewhere in the book. This is the earliest that I have traced, but it would be worth looking at the first edition of 1660 which I have not by me (and which is devoid of the statement of Boyle's Law).

2) *Guinea Pig.* The *terminus a quo* of the use of this beast is naturally that of its introduction into Europe. It is American and its name should be " Guiana pig." I have not traced its exact date of introduction, but it does not seem to have been known clearly until the very end of the 17th century. (The tracing of the species is more difficult than might be thought. Linnaeus, writing in 1758, knows it as a domesticated animal, while Ray in 1699 knew of it only as a curiosity). I think it was first generally in use as a pet about the middle of the 18th century. This was not a period when biological experiment was much to the fore, and I rather think that

the first to use it for experimental purposes was Magendie (1783 – 1855) the teacher of Claud Bernard, round about 1820. A definite answer would, however, demand some research."

C. — (*From the Librarian, Royal College of Physicians, London.*)

"Albrecht von Haller (1708 – 1777) was the pioneer in experiments on live animals, but the earliest use of the guinea-pig was Brown-Sèquard's work in the fifties of the last century, when he was engaged in interference with the nervous systems of these animals in an attempt to induce epilepsy.

So far as mice are concerned there has been less success in the enquiry, but August Weismann (1834 – 1914) is credited with attempts to breed a tailless species of rat in experiments with animals from which the tails had been previously cut."

The following is copied from an article by W. S. Dallas in Cassell's "Natural History," under Order Rodentia *Family XVI* Caviidæ *(Cavies).*

"The Restless Cavy *(Cavia aperea)* which is commonly regarded as the wild original of the so-called guinea-pig (*Cavia cobaya* of some authors), is abundant on the banks of the Rio de la Plata and extends northwards through Paraguay into Bolivia and Brazil.

The specific name of the Restless Cavy seems to be derived from its popular name in the country where it occurs. According to Mr. Darwin, it is very common about the banks of the La Plata, sometimes frequenting sandy hillocks, and the hedgerows formed of the agave and the prickly pear, but apparently preferring marshy places covered with aquatic plants. In dry places it makes a burrow; but when it frequents wet localities contents itself with the concealment afforded by the herbage. Reugger describes it also as generally haunting moist situations in Paraguay, and he adds that it keeps near the borders of forests, but is never found either in the forests or in the open fields. It lives in small societies of from six to fifteen individuals, in the impenetrable thickets of Bromelias, where its presence is revealed by the numerous beaten paths which it produces by going to and fro. In Bolivia, according to Mr. Bridges, it is peculiar to low lands, and there takes shelter among the loose stones of the walls enclosing the fields. It is active in search of food in the morning and in the evening, but will also come forth on gloomy days. Reugger and Azara both agree in the statement that the female produces only one or two young at a time; but the former says that this takes place only once a year, whilst the latter describes the animal as breeding all the year round, and indeed, in this way accounts for its abundance, notwithstanding its being preyed upon so extensively by rapacious birds and quadrupeds.

The question whether our common guinea-pig is really the domesticated descendant of the animal just described can hardly be regarded as finally settled, and, indeed, independently of colour, there are sufficient differences

between them to justify some doubt. The name guinea-pig may, as Mr. Waterhouse suggests, be a mistake for Guiana-pig, and the first specimens may very probably have come from that part of America. Its prevalent colours, as is well known, are combinations of white, black and yellow, and as these colours are shown in drawings of Aldrovandus, dating back to within fifty years of the discovery of South America, there seems every reason to believe that the animal must have been long domesticated in America prior to its introduction into Europe. On the other hand, Dr. Reugger says he saw fourteen apereas representing the fifth or sixth generation from a single couple domesticated about seven years before, and that these exhibited no difference of colouring from the wild animal. Several allied species inhabit the great plains of South America."

The following is abstracted from *"Die Naturgeschichte des Meerschweinchens"* by Alexander Sokolowsky in H. Raebiger's book *"Das Meerschweinchen, seine Zucht, Haltung und Krankheiten"* (M. and H. Schaper, Hannover, 1923).

According to A. Nehring, Brazil is usually given as the home of the guinea-pig and *Cavia aperea* Erxl. as its progenitor. However, from a study of mummified guinea-pigs from pre-Spanish Peru, Nehring is of the opinion that this country is the true home of the animal and that the cavia species, *Cavia cutleri King resp. Tschudi,* widely spread in that part (according to Waterhouse closely related to *Cavia aperea*) is probably the ancestor of the guinea-pig.

The Spaniards introduced the guinea-pig to Europe in the 16th century, where it became a pet. The first account of its presence in Central Europe was recorded in 1551 to 1554. It was mentioned by Gessner in Zürich in 1554.

J. H. M.

The Introduction of Agar-agar into Bacteriology.

Until Koch solidified nutrient media with gelatine, the isolation of bacteria in pure culture was a matter of chance or patience. In 1881, at the International Medical Congress in London, he amazed his audience with the demonstration; and his French colleague, Pasteur, was full of praise.

One disadvantage of gelatine-media is that they do not remain solid at body temperature. The replacement of gelatine by agar-agar overcame this difficulty and marked another and a very important advance in bacteriological technique. The principal algæ from which agar is derived live in the Eastern Asiatic seas, and are said to be mainly the species

Eucheuma (*E. spinosum*) and *Gelidium* (*G. corneum, G. amansii*)*.

According to Hitchens and Leikind**, Frau Fanny Eilshemius Hesse, the wife of Dr. Walther Hesse, a district physician of Schwarzenburg, Saxony, was responsible for the introduction of agar-agar into bacteriological work. She had used agar in solidifying jellies, and whilst assisting her husband in studies of the bacterial content of air, the idea of its use in gelling media had apparently come to her. The recipe had been given to her by her mother, who, in turn, had received it from a Dutch family; they had brought it from Batavia in the Dutch East Indies. Agar was first used in the early 1880's.

Dr. Hesse communicated his discovery to Koch by letter, and Koch, recognising its value, adopted the use of agar in his laboratory.

Frau Hesse, of German descent, was born in Jersey City, N.J., in 1850. She met her husband while she was travelling in Germany and spent the rest of her life in that country. She died in 1934, in Dresden.

J. H. M.

*Abstracted from "The History of Bacteriology" by William Bulloch, 1938. Oxford University Press.

**Slightly abridged from an article "Notes on the History of Bacteriology. The Introduction of Agar-agar into Bacteriology" by Arthur Parker Hitchens and Morries C. Leikind, *J. Bacteriology (1938) vol. 35, pp. 21-22.*

THE ASSOCIATION.

Extracts from Minutes.

Meeting of Council held at Polley's Hotel, Pretoria, 14 November, 1939.

Present: H. H. Curson (Chairman), P. J. du Toit, R. Alexander, J. Quin, H. O. Mönnig, A. C. Kirkpatrick, A. D. Thomas, A. S. Canham, M. Sterne, J. G. v. d. Wath, and S. W. J. van Rensburg (Hon. Secretary).

Apologies for absence received from Messrs. S. T. Amos, A. M. Diesel, and D. G. Steyn. The latter was represented by Dr. Quin.

The Chairman expressed regret at the absence of the President on account of illness. Decided that a letter be sent to him wishing him a speedy recovery.

(1) *Minutes of meeting held 23rd August, 1939.* — These were discussed and passed.

(2) *Arising from these minutes.* —

(a) *Arrear Subscriptions.* — The case of one member was considered. The Chairman offered to see him during the next two weeks. If no success attended this interview, the Secretary was instructed to refer the matter to the Association's legal advisors.

(b) *S.A. Permanent Force.* — Dr. du Toit reported that as a result of negotiations carried on with the Defence authorities, the granting of a professional allowance to the veterinary officer had now been sanctioned. The officer would be appointed with the rank of Captain, but on a special scale of £800-£900, all pensionable, and the allowance was the difference between this and the usual salary applicable to the rank of Captain. He stated that Mr. A. M. Howie had been appointed and would assume duty within a few days.

The Chairman thanked Dr. du Toit for all his labours and for getting a satisfactory settlement of a grievance which had occupied the attention of the Association for many years.

(c) *Hormone Treatment.* — The Secretary reported that the committee appointed at the last meeting had drawn up a report which was circularised among and approved of by members of Council, and had since been forwarded to the Jockey Club by whom it was to have been considered on November 22nd.

The Chairman moved a vote of thanks to the Committee (Drs. Alexander, Quin and Quinlan).

(d) *Blue Cross Veterinary Hospital, Johannesburg.* — The Secretary reported that the proprietor of this hospital, who is not registered, had recently issued a veterinary certificate and that a prosecution was being instituted.

(3) *Dr. Alexander proposed.* — "This Council of the S.A.V.M.A. views with misgiving the interference with professional veterinary education in this country by the unilingual policy of the Pretoria University, to which is attached the only veterinary faculty in South Africa. It is resolved that active steps be taken by this and the incoming Council to eliminate this interference by adequate representation to the Minister of Education, the Minister of Agriculture, their Secretaries, the Veterinary Board, and any other persons deemed necessary, on lines to be decided by the Council."

On the motion failing to find a seconder the matter lapsed.

(4) *Standing Committees.* — These were elected as follows:—

1. Editorial: P. J. du Toit, A. D. Thomas, C. Jackson, J. H. Mason, J. H. Cloete, and M. Sterne (Convener).

2. Finance: H. H. Curson, A. D. Thomas, B. S. Parkin, J. G. v. d. Wath (Convenor).

3. Library: P. J. du Toit, G. de Kock, D. Steyn, C. Jackson (Convenor).

4. General Purposes: H. H. Curson, H. O. Mönnig, P. J. J. Fourie, A. C. Kirkpatrick, A. S. Canham, and C. J. van Heerden.

Assistant Secretary: J. G. v. d. Wath.

(5) *General.* —

(a) *Finance.* — On the recommendation of the Finance Committee it was decided that the fixed deposit of £300 with the United Building Society, which matures on 30.11.39, be renewed for 12 months and that the deposit of £250 with the Standard Building Society due on 30.11.39 be raised to £300 and redeposited for 12 months.

(b) *Theiler Memorial.* — Mr. v. d. Wath proposed that this Council expresses its appreciation of the Government's action in erecting a statue to Sir Arnold Theiler and that the thanks of the Association be conveyed to the Government. Unanimously agreed.

Dr. Alexander suggested that Council consider the question of inviting members of the Association at the time of the unveiling of the memorial to order a plaque of Sir Arnold. Agreed that a Committee consisting of Drs. du Toit and Alexander consider this question.

(c) *Complaints.* — (i) Dr. Curson produced a letter from a member stating that he had heard a rumour that prospective students had been warned that students of one university would be given preference for the course in Veterinary Science.

Dr. du Toit stated that in view of the large number of applicants, students of *all* universities had been warned that only a limited number would be accepted for the course.

The Secretary said he had also investigated this complaint and found it to be based on a misapprehension.

(ii) A letter from a member complaining against the advertisement of certain kennels. This did not constitute a breach of the Act, but it was decided to refer it to the Veterinary Board for recording.

(iii) Complaint from another member against the letterhead of a member was referred to the Board with a request that the member's attention be drawn to it.

(d) *General Meeting.* — Dr. du Toit proposed that Dr. Curson take the chair at the General Meeting. Agreed.

The meeting concluded at 10.15 p.m.

S. W. J. van Rensburg,
HON. SEC.-TREAS., S.A.V.M.A.

34th General Meeting held at Onderstepoort on 15th November, 1939.

Present.—H. H. Curson (Chairman), P. J. du Toit, A. D. Thomas, J. Nicol, M. Sterne, W. D. Malherbe, J. G. v. d. Wath, H. O. Mönnig, J. I. Quin, J. H. L. Cloete, R. Clark, A. S. Canham, V. Cooper, R. du Toit, W. O. Neitz, F. B. Wright, J. R. Frean, J. R. Scheuber, J. W. A. Brooks, A. E. Lund, H. P. Steyn, A. M. Diesel, M. M. Neser, D. Coles, P. S. Snyman, E. M. Robinson, P. J. Fourie, J. J. G. Keppel, D. T. Mitchell, R. Alexander, and S. W. J. van Rensburg (Secretary).

The Secretary read a letter from the President regretting his inability to attend on account of ill-health. Unanimously agreed that the Secretary write to Mr. Amos regretting his illness and wishing him a speedy recovery.

Dr. du Toit expressed regret that he would not be able to be present throughout the meeting, and said he again wished to extend a hearty invitation to all members to attend the unveiling of the Theiler Memorial that afternoon.

S.A. Permanent Force.—At the request of the Chairman, Dr. du Toit briefly outlined the negotiations with the Defence Authorities, and stated that the latter had at last acceded to the Association's request for a professional allowance to be paid to the veterinary officer on the lines of that paid to medical officers. This allowance would be equal to the difference between the scale of £800 - £900 and the salary pertaining to the ordinary rank of Captain, and it would be pensionable. Mr. A. M. Howie had been recommended for the post and would assume duty within the next few days.

The Chairman thanked Dr. du Toit for all he had done in this matter, and also on behalf of Dr. Schulz who had obtained substantial damages for his injury, and for allowing the Association to meet at Onderstepoort to-day.

Dr. du Toit then left the meeting.

1. *Minutes of General Meeting* held on 25th - 27th October, 1938, having been published, were taken as read and confirmed.

Arising from these minutes the Chairman wanted to know what had been done with regard to the Constitution. The Secretary replied that the last General Meeting had approved the translation with the exception of the name. This was referred to a Committee for investigation and report to Council, the latter to have power to act. After receiving this report and giving it full consideration Council had decided on "Suid Afrikaanse Veterinêr Mediese Vereniging."

2. *Greetings.*—A letter of greetings from the N.V.M.A. Congress at Great Yarmouth was read. Decided that the Secretary should reply reciprocating the good wishes.

3. *New Members.*—The following were unanimously accepted: C. F. B. Hofmeyr, P. R. Mansvelt, A. J. McGilvray, D. T. Mitchell, G. L. Muller, N. C. F. Steenkamp.

4. *Elections of Council.*—The following four members were elected to Council in the present election: R. A. Alexander, J. L. Dickson, P. J. du Toit and A. C. Kirkpatrick.

Council for 1939-40 would thus be.—President: S. T. Amos; Vice-President: H. H. Curson; Secretary-Treasurer: S. W. J. van Rensburg; Members of Council: R. A. Alexander, A. S .Canham, J. L. Dickson, A. M. Diesel, P. J. du Toit, A. C. Kirkpatrick, D. G. Steyn, A. D. Thomas, and the Editor of the Journal.

5. *Honorary Life Vice-President.* — The Chairman moved that Mr. F. J. Carless be elected an Honorary Life Vice-President of the Association. In doing so he said that ever since Mr. Carless arrived in South Africa 44 years ago he had always had the interests of the profession at heart. He had been President several times and represented the Association on the Veterinary Board for several years. He had also taken a leading part in public matters especially in agriculture and agricultural education and had been a member of the Meat Control and Shipping Boards. By his tactful personality and sterling character he had set an example of which we could all be proud. Mr. F. B. Wright seconded, and the motion was passed unanimously.

6. *Presidential Address.* — In the unavoidable absence of the President no address was delivered.

7. *Arrear Subscriptions.* — The Secretary stated that only one member was over three years in arrear. Council was dealing with this case and it was suggested that the matter be left to Council. Agreed.

8. *Secretary's Report for 1938-9.* — This was accepted.

9. *Reports of Standing Committees.* — These were agreed to. During discussion on the report of the Finance Committee, Dr. Alexander proposed: "That in view of the present international position the Association donates £25 of its fund to Charity, half to go to the International Red Cross and half to the Mayor's Fund earmarked for the purchase of South African produce." This was seconded and agreed to by 20 votes to 5.

The Chairman wanted to know what the position was with regard to the Library. Drs. Cloete and Robinson replied that this was being kept up-to-date in the Students' Hostel.

The Chairman appealed to members to support the Editorial Committee and also to support those firms that advertised in the Journal.

Mr. Coles said that members were sometimes offended if articles were refused publication or were changed by the Editorial Committee, and he appealed to all members to abide by the decision of this Committee.

10. *Motion by Dr. Steyn.* — "That pure strychnine should not be sold to the public save under cover of a medical or veterinary prescription."

The Secretary read a letter dated 6.11.39 from Dr. Steyn, regretting his inability to be present and giving his views on the question. A letter dated 14th August, 1939, had also been received from the Maritzburg branch of the S.P.C.A. asking for the Association's support in an endeavour to obtain further legislative control over the sale of pure strychnine.

The Chairman considered that this matter should be more fully investigated.

After discussion Dr. Mönnig proposed that a Committee consisting of Drs. Steyn, Parkin, and Thomas be appointed to consider the whole question of the sale of dangerous drugs. Dr. Alexander suggested that Dr. Mönnig be included, and Mr. Clark proposed that a member of the Field staff be co-opted. Agreed.

11. *General.* — (a) Apologies for absence were received from Messrs. S. T. Amos, F. J. Carless, F. Verney, and J. L. Dickson.

(b) The Chairman referred to the loss sustained by the Association through the death of the following members since the last meeting: Major W. G. Barnes, Messrs. W. E. Footner, F. Hutchinson, J. A. Maybin, and W. A. Simson. The meeting rose and stood in silence as a mark of respect.

(c) Mr. Diesel reported on the action taken by the responsible committee with regard to the Footner memorial. The Chairman thanked the Committee.

(d) On the proposal of Mr. Nicol it was decided that invitations for Municipal Veterinarians to attend General Meetings be sent to the respective Town Clerks in future.

In conclusion the Chairman expressed his pleasure at seeing several members who had come a long distance to attend this meeting which was being held under unusually difficult circumstances. He also thanked the members of Council, the Secretary and Assistant Secretary for their services during the past year.

Dr. Alexander proposed a vote of thanks to Dr. Curson for taking the Chair and the meeting ended at 12.30 p.m.

S. W. J. van Rensburg,
HON. SEC.-TREAS., S.A.V.M.A.

Meeting of Council held at Onderstepoort, 14th December, 1939, at 9.30 p.m.

Present.— S. T. Amos (President), H. H. Curson, P. J. du Toit, A. C. Kirkpatrick, R. Alexander, A. D. Thomas, M. Sterne, M. M. Neser, B. van der Vyver, J. Quin, J. G. v. d. Wath, and S. W. J. van Rensburg (Secretary).

Apologies for absence received from Messrs. A. S. Canham, A. M. Diesel and Dr. D. G. Steyn. These members were represented by Messrs. Neser, v. d. Vyver and Quin, respectively.

The President stated that before proceeding with the business of the meeting he wished to explain that his absence from the last General Meeting was entirely due to medical orders. He said we are going through very trying times and appealed to all to stand 100 per cent. behind the Association and the profession by sinking all personal feelings and opinions which might cause dissension.

1. *Minutes of Council Meeting* held on 14.11.39 having been read, were confirmed.

2. *Arising from these minutes.*—

(a) *Arrear subscription.*—The case of one member was considered. After full discussion it was decided that in the event of non-payment of arrears before the next General Meeting he be dealt with under Rule 7 of the Constitution.

(b) *Blue Cross Veterinary Hospital.*—The Secretary reported that the proprietor of this hospital in Johannesburg had recently been prosecuted and found guilty under the Veterinary Act.

(c) *Resignation: Dr. Alexander.*—The Secretary read a letter dated 15.11.39 from Dr. Alexander resigning as member of Council. Dr. Alexander left the meeting. After full discussion Dr. Curson proposed that the President should ask Dr. Alexander to reconsider his decision. Mr. Kirkpatrick seconded and the motion was agreed to unanimously.

Dr. Alexander was recalled and informed of this decision. He stated that he had taken up that attitude because he felt he would be doing a disservice to the Association if he did not give members an opportunity of expressing an opinion as to whether the motion he introduced at the last Council meeting should be discussed. If he was re-elected, he would feel that he had a mandate from members to re-introduce the motion. He would withdraw his resignation now, but would hand it in before nominations are called for Council for 1940-41.

The Secretary pointed out that it was frequently advisable to make certain deletions from the minutes before these are published in the Journal. The President suggested that discretion as to what should be published be left to the Editor and Secretary. Agreed.

Dr. Alexander considered the discussion on his motion should be deleted. Agreed.

3. *General. — Use of Association's Funds for Charity.* — The President stated that on receipt of letters from certain members objecting to the proposal adopted at the General Meeting on 15.11.39 when it was decided to donate £25 to War charities, he obtained Counsel's opinion. This was submitted to the meeting and was to the effect that the Constitution did not make provision for the utilisation of the funds of the Association for purposes other than those specified under the objects of the Constitution, (Rule 2).

The proposal was therefore *ultra vires* and he (the President) acted within his rights in instructing the Secretary to withhold payment.

Dr. Alexander proposed that Council expresses its thanks to the President for his work in this connection, accepts Counsel's opinion as outlined by the President and also recommends its acceptance by the Special General Meeting. Mr. v. d. Wath seconded.

Dr. Curson proposed that Council proceed on the lines decided upon at the last General Meeting on 15.11.39. This was not seconded and Dr. Alexander's motion was carried.

The President pointed out —

(1) That according to information supplied by the Secretary the Constitution of the Association had apparently not been registered.

Dr. Curson, however, stated that it was registered under the Companies Act.

(2) That it was necessary to make certain amendments to the Constitution and these would have to be considered by Council in the near future.

The meeting adjourned at 11.20 a.m.

S. W. J. van Rensburg,
HON. SEC.-TREAS., S.A.V.M.A.

Special General Meeting held at Onderstepoort, 14th December, 1939.

Present. — S. T. Amos (President), J. L. Dickson, R. Alexander, J. G. Boswell, I. P. Marais, J. Quinlan, H. P. Steyn, P. J. Meara, K. Schulz, S. J. v. d. Walt, M. M. Neser, J. G. Williams, J. H. R. Bisschop, J. G. Bekker, B. v. d. Vyver, V. Cooper, A. C. Kirkpatrick, B. S. Parkin, Mrs. J. A. Robinson, R. Clark, A. E. Lund, W. A. Dykins, J. R. Scheuber, E. M. Robinson, M. Sterne, W. O. Neitz, H. Theiler, H. Graf. J. Quin, M. W. Henning, P. J. Fourie,

C. Jackson, A. D. Thomas, H. H. Curson, P. J. du Toit, J. G. v. d. Wath, and S. W. J. van Rensburg (Secretary).

The President, after reading the notice convening the meeting, outlined the steps taken by him after receiving letters from some members protesting against the motion adopted at the last General Meeting voting £25 to War charities. He had obtained Counsel's opinion and read this to the meeting. It was to the effect that "The Association's objects are clearly defined and do not include the right to grant donations other than for the purpose defined therein. The donations in question were therefore unconstitutional and *ultra vires* the objects, and any member of the Association would have the right to interdict the payment of such donations."

He informed the meeting that the Council meeting just concluded had passed a resolution accepting Counsel's opinion and also recommending its acceptance to the Special General Meeting.

The President stated that he had only one object in view, the furthering of everything that is good for the profession and the Association, and appealed to members to be tolerant, to respect the other man's view, to cultivate unity of thought, goodwill, mutual understanding, loyalty to the profession and to continue our good work.

Dr. Quin proposed that the meeting approved of the resolution passed by Council as stated by the President.

Dr. Curson said he could not agree with Counsel's opinion, but as a democrat and one who had the interests of the Association at heart he was prepared to accept the opinion of the majority.

On being put to the vote Dr. Quin's motion was carried, there being three dissentients.

General. — (1) Apologies for absence were received from Messrs. Canham, Diesel and Dr. Steyn.

(2) The Secretary read a letter from Mr. F. J. Carless, thanking the Association for honouring him by his election as Honorary Life Vice-President.

(3) Dr. du Toit proposed a hearty vote of thanks to the President.

The President said he wished all members a happy Christmas and a prosperous New Year in spite of the War.

The meeting concluded at 12.45 p.m.

S. W. J. van Rensburg,
HON. SEC.-TREAS., S.A.V.M.A.

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Some Economic Aspects of the Milk Problem.*

By J. H. L. CLOETE, Springs.

Introduction.

The problem of nutrition has been defined (Report, 1937a) as "the direction of consumption, both quantitatively and qualitatively, along the lines which the new science of nutrition has indicated as being conducive to health and efficiency." In this new science the rôle of milk is all-important. So far we, as veterinarians, have regarded as our special charge the qualitative aspect of milk production. If, however, we are really desirous of contributing towards the solution of the evil of malnutrition, then, far from resting content in our present efforts at the production of wholesome milk, we should endeavour to assist, as far as is in our power, in ensuring for each person a sufficiently large daily ration of this wholesome food.

The factors which govern the consumption-level of any commodity are well stressed by Anning (1939) — "make the foods available, sell them at a price at which people can afford to buy them, and teach the people how to use them." The third, or educational factor falls more within the province of our medical colleagues, and indications are not lacking that they are fully aware of its importance. However, this propaganda can act only within a certain definite range, the limits of which are set by the first two factors. It is these, the economic aspects, which are to be considered in this paper. It is hardly necessary to mention that a detailed economic study is far beyond my abilities; moreover such an article would not interest you. My sole aim is to induce you to broaden your outlook on the milk problem by allowing your thoughts to dwell, be it ever so lightly, on such matters as quantity and cost.

Briefly, I wish to consider, in the light of nutritional requirements, our present levels of milk consumption; if these are found to be deficient an attempt will be made at identifying the main causes, and thereafter certain remedial measures will be suggested. My remarks will be confined to the larger urban areas, because for them alone relevant statistics are available.

Milk Consumption.

Here we must calculate, according to accepted standards, the daily requirements of each of the centres included in this study. In its report (1936a) the League of Nations Committee places the minimum daily

* This paper was prepared as part of the symposium on milk which, but for the outbreak of the war, would have been presented at the 34th General Meeting of the South African Veterinary Medical Association.

allowance for children under two years of age at 1·3 pints, while for children between the ages of two and fourteen, as well as for expectant and nursing mothers, this figure is raised to 1·75 pints. The requirements of other classes are lower, yet even here the allowance does not drop much below one pint per day. If these standards are considered in conjunction with the age-distribution of our population, conveniently shown by Haylett (1939), one concludes that, for our European section at least, a fair average *per caput* requirement would be approximately 1·2 pints per day. The non-European element presents a much more difficult problem. Nutritionally their requirements must be similar to those of Europeans, and to see this standard reached must be our ideal; however, in view of their present economic state, the attainment of this ideal must remain a matter for the very distant future. In the calculation of present possible non-European consumption our *per caput* allowance must be modified fairly severely. After consulting people interested in such matters, I have decided to place this figure as low as 0·2 pints* per day, i.e. at one-sixth of the European estimate.

On the basis of these two standards the daily requirements of eleven urban centres have been calculated. When these are presented together with figures indicating the actual daily consumption in these areas (table 1) it becomes evident that in all but one instance minimum *per caput* levels are not reached. By expressing present consumption in terms of these requirements a "satisfaction percentage" is obtained. Apart from Kimberley, the highest percentage is that of Maritzburg (80); the majority of the remainder are around the seventy mark, while Durban is lowest with only 52, and this despite the exclusion of roughly one-third of the population (see foot-note to table 1). In considering the extent of these shortfalls we must not lose sight of the ridiculously low level at which non-European requirements were assessed. There would be little cause for rejoicing even if the "satisfaction percentage" were close to the hundred-mark. In order to force home this point I have included in the second half of table 1 figures to show what these percentages would have been had the non-Europeans been included on the European basis. In this section (last column of the table) the index drops to approximately 40%, only Pretoria and Kimberley reaching the 50% level. The former centre figures high on the list mainly because of its relatively low native population, while Kimberley leads, in spite of a heavy disadvantage in this respect.

The foregoing very conservative estimates indicate fairly conclusively that grossly inadequate quantities of milk are being consumed in our larger urban areas. Even if we regard the second half of table 1 merely as an indication of future scope, we still find that in these eleven centres

* Note that this is not an estimate of present non-European consumption, but merely an allowance for what this section might consume if sufficient milk was available at a cost of say 2d. per pint. Domestic servants probably consume much more. In Benoni location (Anning, 1939) the "upper strata" were found to have 3d. per week per individual available for the purchase of dairy produce.

there is urgent need for the augmentation of daily supplies by approximately 48,000 gallons.

TABLE 1.

Milk Consumption in Urban centres — Data taken from Report (1939) *and Municipal Handbook* (1939).

Name of Centre.	ALLOWANCE. E=1.2pt. & Non-E.=0.2pt. daily.				ALLOWANCE. Both E & Non-E=1.2pt. daily		
	Gallons per day.			Satisf't'n	Gallons per day.		Satisf't'n
	Supplied.	Required	Shortage	Per cent.	Required	Shortage	Per cent.
Johannesburg	32,209	46,695	14,486	69	76,320	44,111	42
Cape Town	20,000	27,303	7,303	73	46,262	22,262	43
East Rand[1]	12,500	19,353	6,853	65	32,986	20,486	38
Pretoria	8,500	11,775	3,275	72	16,950	8,450	50
Durban[2]	8,000	15,309	7,309	52	24,625	16,625	32
Pt. Elizabeth	7,000	9,721	2,721	72	16,893	9,893	41
Kimberley	3,500	3,226	—	108	6,604	3,104	53
West Rand[1]	3,500	6,755	3,255	52	11,580	8,080	30
Maritzburg	3,000	3,758	758	80	6,424	3,424	47
Bloemfontein	3,000	4,300	1,300	70	7,650	4,650	39
East London ..	2,700	3,956	1,256	68	7,035	4,335	38
Total	103,909	152,151	48,408	68	253,329	149,420	41

E. = Europeans, Non-E. = Non-Europeans.

[1] Mine natives excluded — where correct figures for these were not available the non-mining native population was taken as being equal to the European figure for the particular centre.

[2] 83,523 Indians excluded from calculations.

South Africa is faced with a so-called surplus dairy product problem. The solution of this is sought in the compulsory export of certain quotas of cheese and butter. For the year 1937 these were fixed at 1,668,382 and 7,214,535 lb. respectively. However, as a result of our inability to produce economically enough to compete on the low-priced world market, subsidies have to be paid on all exports. During the year under consideration (1937) these subsidies were as follows (for 1st, 2nd and 3rd grade products): butter, 4·5d., 3·5d. and 2·5d. and cheese 4·0d., 3·5d. and 3d. per pound. If we work on the basis of milk having 3·5% butterfat and 8·5% solids-not-fat, and we allow for an overrun of only 17%, then we find that our daily requirement of 48,000 gallons would represent an annual output of 17,520,000 lb. of cheese or 7,174,440 lb. of butter. It becomes evident that the diversion of this amount of milk away from the factories would relieve completely the compulsory export burden under which the industry is labouring. Moreover, it must not be forgotten that the funds for subsidization accrue from a levy imposed upon all local sales of cheese, butter and, recently, liquid milk too.

The point to be decided is whether this diversion is practicable. It is well known that much of our cheese-milk and our cream is produced

in areas remote from the large urban concentrations. However, there is evidence that probably the major portion of it is actually produced within the possible drainage areas of these cities. Of the Union's 54 registered creameries, 44 are situated within such areas; for cheese factories the corresponding figures are 58 out of 116. Although these plants undoubtedly draw from far distant localities, it is felt that they will nevertheless tend to be located in close proximity to their chief sources of supply. Here there appears to be scope for rationalization of the industry in such manner that the fluid-milk and factory-milk zones become more clearly segregated. Factories should be located towards the periphery of each fluid-milk zone, thus enabling them to draw mainly from outside areas while still remaining readily accessible for any surplus from within. It is well to remember that for a large city the radius of its drainage area may be anything up to 200 miles. As consumption increases so it becomes more and more necessary to draw supplies from the remoter parts of this area. This is well illustrated by the following figures relating to Johannesburg. In ten years the daily consumption of milk increased by approximately 12,000 gallons. During the same period local production actually decreased by about 2,000 gallons. Thus not only the entire increase but also the equivalent of this decrease had to be drawn from distant sources.

Admittedly the situation of every farm within such a drainage area is not favourable to participation in the fluid-milk trade. However, since dairy farming is one of those intensive forms of agriculture which tend towards close grouping along a country's main lines of communication, it is felt that the number of farmers who would be excluded from exploiting this market would be quite small and that most of the milk produced in such areas could, with proper arrangement, be diverted from the factories to the urban liquid-milk trade.

That such diversion would be financially beneficial to the producers concerned is the only conclusion to be arrived at from a comparison of factory prices (i.e. 5d. per gallon for cheese-milk and the equivalent of about 4d. per gallon of milk separated for cream) with ruling wholesale prices (table 2). Transportation charges will be somewhat heavier, but in spite of this the fluid-trade remains the more lucrative by at least 2d. to 3d. per gallon. An additional 2d. per gallon on the 48,000 gallons required daily would increase producers' revenue by £146,000 per annum.

Thus we see that the factories are not competing with the liquid-trade. Producers are eager to get a footing on the latter market; many of them are willing to serve long and unprofitable apprenticeships in anticipation of the eventual acquisition of a fixed daily quota. We have the anomalous position of abundant supplies and yet inadequate consumption. The cause of this is easily ascertained — *the retail price of milk is too high.*

The quantity of milk sold is determined by the law of supply and demand. In the case of a wealthy family demand signifies the amount of

milk which the members of the family wish to consume; by effective propaganda this demand may be increased. However, in the vast majority of cases, demand means the amount of milk which can be purchased with the family milk allowance. Just how low this allowance may be is shown by Anning (1939). Amongst the poorer European families of Benoni (earning £150 per annum) the weekly expenditure on milk and milk products is 4·75d. per individual, while in the "upper strata" of location life (income up to £90 per annum), the corresponding figure is 3d. Careful consideration of the respective family budgets fails to disclose much scope for increase of these allowances. When incomes are low people "buy mainly the fuel foods — which are cheapest, bulk for price — to fill their bellies, to keep them warm and to give them energy for their daily work." Very little is left over for the "health-insurance foods" such as milk and fruit. If these are costly, the demand for them will be sub-minimal. In a statistical survey (Report 1937b) it was found that as the family income rises, so does the *per caput* consumption of milk. In the £150 income group consumption was only half that of the £250 group and one-third that of families with an annual income of £550. These South African findings are supported by similar results in other countries, notably the United States of America [Faith Williams, (1936)] and Sweden (Sociala Meddelanden, 1934 — cit. Report 1937a). As an indication of the comparative inelasticity of the allowance for milk in the family budget may be mentioned that the most significant feature of the deterioration of nutrition found in large families among the lower income groups is the decline in the consumption of milk [Burns (1933; Report 1936b)]. In Britain, Forrester (1927) found that the spring drop of 1d. per quart usually resulted in a 5 to 20% increase in sales of milk. This extra demand disappeared again as soon as the onset of winter conditions caused an increase in price.

The above will suffice to indicate that it is mainly the lack of communal buying power which is responsible for the striking discrepancy between nutritional requirement and commercial demand. Increased buying power, or in other words decreased cost of milk, especially if supplemented by effective propaganda, would do much towards raising our present low *per caput* consumption. For the most unfavourably situated classes, milk and its products are already being made more accessible through the process of State subsidization. However necessary they are, such schemes soon grow to the limits of their practicability, and at best only relatively small numbers can be assisted. There are certain sections of each community which will probably always require assistance, and for them such schemes will persist; but even then their efficacy would be greatly enhanced should they be secondary to a general reduction of the retail price of milk.

Although we may now be agreed on the desirability of such a decrease, we have as yet no indication that economically this step is possible. The consumer's payment goes to remunerate two distinct services — production and distribution. In general one would consider the former service to

have ceased when the milk leaves the producer's plant. However, as the cost of delivery to the wholesale depot is borne by the producer this bulk transportation will be included under *production*. By this division one is placed in the advantageous position of being able to regard the wholesale price as an index of the reward apportioned to the service of production, while the difference between the above and the retail price will indicate the payment made to distribution.

Production of Milk.

The League of Nations Committee (Report 1937a) states that "it may be taken as axiomatic that a comprehensive and well-considered nutrition policy should not be of a nature to cause losses to food producers, nor should its effects be to force the prices of food products below levels which cover the costs of the marginal producer whose output is required to meet current demand and give him a fair return on his labour and investment." We may add that it is equally axiomatic that wholesome milk cannot be produced at "cut-throat" prices. Unless we feel that the returns accruing to producers are unjustifiably high, we dare not sponsor any scheme entailing a diminution of their remuneration.

Data relating to South African costs of production are practically non-existent. Bonsma (1926) conducted an investigation covering four dairies in and around Pretoria. The highest figures were those of a plant situated within the municipal area. Here the average cost per gallon, over a period of twelve months, was just below 18d. However, as all was not well with the method of feeding employed, and as this type of dairy is gradually disappearing, we shall leave these figures out of consideration. On three farms just beyond the municipal borders the costs were 10·86d., 10·16d. and 14·60d. The high costs on the third farm were due mainly to initial inexperience of the farmer; by the end of the twelve months over which the study ran his costs had fallen into line with those of the other two, longer established, dairies. It must be mentioned that in these calculations the American system of cost accounting was followed; thus, interest on capital, cost of grazing, cost of feed produced on the farm, etc., are all included. In other words, everything the farmer receives above his estimated cost is true entrepreneurial profit. Then also his dairy revenue is augmented by the sale, to himself, of such commodities as stable manure used on his lands. On the above farms such sales amounted to 0·56d., 0.73d. and 1·00d. per gallon, thus leaving 10·3d., 9·43d., 13·60d. per gallon to be made up from sales of milk. We must not forget that a small profit on the dairy may be supplemented by a credit balance in the forage production account, due to "sales" of produce to the dairy enterprise. The first two farmers were handling approximately 3,000 gallons per month. From sales at 1/- per gallon they would have made "true" profits of £21 and £32 per month respectively. Actually they received 1/4 and 1/3 for their milk, and thus their profits were £71 and £68:10:0. These do not include

profits made on any other part of the farm. I think it will be agreed that at that time the wholesale price was excessive. It is interesting to note that at present the wholesale price in Pretoria is 1/1d., which still ensures for farmers a very good return.

As the site of production is moved farther and farther away from the larger centres, so will factors such as cheaper ground, more abundant grazing, cheaper labour, and the opportunities for more diversified farming tend to reduce cost of milk production. Chatterton (1939) estimates the cost of production (on the farm) in the Kimberley area to vary between 4d. and 6d. per gallon. This is a fairly remote region, but let us assume that the higher figure holds good for any locality just on the fringe of an urban drainage area, say 200 miles distant from the centre of consumption. To this cost will have to be added transportation charges consisting of firstly, delivery to the station and, secondly, railage. The former would be roughly similar to the transportation charges of the dairies considered above, i.e. between 0·5d. and 0·55d. per gallon. The railage for a 200 mile trip is 2·25d. per gallon, thus making the total cost of transportation something less than 3d. By adding this to our actual cost on the farm we arrive at the figure of 9d. per gallon.

It appears that the cost of the service of production, as we have defined it, varies between 9d. and 10d. per gallon. In the Kimberley area and in Natal the cost must be somewhat lower. Comparing these figures with ruling wholesale prices (see table 2, compiled from data supplied in Report, 1939) we see that the margin of profit is not great. Only on the Reef and in Pretoria does the price exceed 12d. per gallon. For the remainder the mode is in the region of 10d., while in Durban, Maritzburg and Kimberley prices are twopence to threepence lower. The high prices along the Reef are somewhat inexplicable. The condensery at Standerton obtains its full quota and budgets on a daily intake of 5000 in 1941, in spite of paying only 7.5d. per gallon. It must be remembered that the standard demanded is high and that the railage from Standerton to Johannesburg would amount to about 1·5d. In Kroonstad, producers are negotiating for the establishment of a dried milk factory — they are willing to guarantee the delivery of at least 1,200 gallons per day at 6d. per gallon. Again this milk could be delivered to Johannesburg at an additional cost of about 2·5d. Having regard to these figures and even allowing that these may refer to specially favourable areas, one cannot see why the wholesale price along the Reef should exceed 11d. per gallon. One cannot help feeling that the figures supplied to the National Marketing Council do not represent true average wholesale prices; they probably refer to periods of scarcity or to clients producing special high-grade milk.

Apart from this reduction (that is, if the wholesale prices on the Reef are really high) there appears to be little scope for decreasing the retail price of milk through reducing the producers' reward. However, if

our present artificial export trade is to be replaced by one that is sound and profitable, dairymen must learn to produce milk more economically.

For successful competition on the low-priced world market the first essential is low cost of production. The latter must be adapted to ruling prices. The ultimate solution of many of the problems to be overcome during the course of this adaptation lies in the hands of the veterinarian. Here is our opportunity of proving the economic worth of our services. Let us mention but a few of the lines along which we should work.

Most important of all is the problem of wastage of cows. Bonsma (1926) indicates that no other factor has so important a bearing on cost of production as a high average yield for the total herd (dry cows included). Large percentages of low-yielders, of cows failing to reach their maxima, or of cows running dry, immediately send costs per gallon soaring. Every dairyman does not possess record-breaking cows — moreover, this is not necessary. The main requirement is that for the type of cow (usually of medium production) kept, the maximum yield should be received.

It is well-known that a cow reaches her maximum output at her third or fourth lactation, maintains this for four or five years, and then begins to fall away. Her most profitable period is from about the fifth to the twelfth year. In any stable, the larger the percentage of cows between these ages, the higher is the average yield likely to be. Thus, the average age of our dairy population must be significantly correlated with cost of production. In England statistics show (Report 1937c) that this average age is only half of the expected figure! Moreover, while only 4% of cows are disposed on account of old age, disease is responsible for the relegation of 58%. What will similar investigations in South Africa disclose? What are the economic effects of the loss of teats and quarters due to tick-bites, barbed wire injuries, etc. Recently van Rensburg (1939) indicated the significance of mastitis. Add to this the economic effects of tuberculosis, contagious abortion, and our various parasitic diseases; the total damage must be astounding — and yet we find it necessary to justify our efforts at the eradication of disease amongst dairy animals on public health grounds alone!

A further factor in economic production of milk is maintenance of a fairly uniform supply — wide fluctuations result in considerable wastage. This is to be achieved mainly by regulation of calving dates — but how is this possible while infertility, abortion, and sterility are wide-spread? As a result of these conditions the farmer frequently finds himself saddled with large numbers of unremunerative dry cows at times when milk is scarce and when maintenance costs are rising (e.g. in the winter). These conditions too, in addition to calf diseases, have an adverse effect on the calf crop. This is of particular importance to the farmer who is fortunate enough to have sufficient cheap grazing to render profitable the rearing of heifers. To him the most advantageous method of acquiring a high-

producing herd is by grading. Naturally, high birth-rate and low mortality are significant factors in determining the degree of success he is likely to achieve.

However urgent the eradication of these conditions may be, we should be extremely guarded in the matter of formulating policy, otherwise the cure may well be worse than the disease. If our demands for quarantine services, for condemnation of animals and for special buildings and equipment are going to result in inefficient division of labour, in high herd charges, and in excessive capital expenditure, then we will be rendering the dairy industry a distinct disservice, for Bonsma (1926) has shown that these factors play no small part in determining the level at which milk can be produced without financial loss. Often our regulations suffer from rigidity, thereby inflicting much hardship upon producers, especially those of moderate means. In Canada, in the United States of America, and in Denmark it has been found that the eradication of a single disease is a matter not of years, but of decades; of what moment is it then whether one particular dairyman progresses towards the goal a little slower than his more fortunately placed neighbour. Our regulations should be sufficiently elastic to allow of this discrimination. It should be our aim, firstly to realize the extent of the disease, secondly to check its spread, thirdly to formulate an eradication scheme which is economically practicable, and, finally, to keep the campaign going at a pace which will not impose any severe strain upon the resources of the industry.

A matter meriting our attention is the feeding of animals. The extent of such feeding depends on many factors and, for the dairyman, the determination of the most advantageous level is not always an easy matter. While one fails to obtain his maximum possible yield, another raises his costs unduly by feeding expensive concentrates in amounts far in excess of requirements (See dairy P — Bonsma, 1926). At times this is carried so far as to prejudice the health of the animals. We cannot all aspire to become specialists in the complicated field of dietetics, but each of us should master its general practical principles, thus being able to furnish advice on the judicious use of such feeds as are most economically available.

We agreed to include railage under the heading of production. We have seen that the charge for a two-hundred mile journey is 2·25d. per gallon, or more than a farthing a pint. I do not think anyone can look upon this as cheap. It certainly appears to merit service infinitely superior to that now obtainable. Modern refrigerated trucks, cold storage at stations, facilities for rapid handling, and speedier train service are all highly desirable. Rather than consider reductions in railway charges, let us agitate for the provision of such services as will have a beneficial effect both on the quantity and the quality of milk coming to our larger cities. Let me quote but one example of what is done in other countries: daily 25,000

gallons, roughly one-third of Sydney's milk supply, is brought to that city by one special train which picks up milk in the country districts at a time convenient for the producers and then over the last eighty miles of the journey runs non-stop in order to deliver these supplies early enough for satisfactory distribution.

Distribution.

Without this highly important service production would be all but valueless. To look upon all concerned in this business as parasitic middlemen is ridiculous — they all play a most important and necessary part. However, because of the significance of the service and because of the importance of product handled it is absolutely essential that efficiency of the highest order, economically as well as technically, be attained and maintained.

Table 2 has been constructed to show what percentage of the retail price* of milk is apportioned to the service of distribution. In its Report (1939) the National Marketing Council draws attention to the part played by the proprietors of restaurants in Durban in raising the retail price of milk. This in conjunction with its low *per caput* consumption, must be responsible for the high cost of distribution (75% of the retail price) in this centre. In so far as the other centres are concerned, it is rather significant that, irrespective of actual price levels, the percentage cost of distribution is fairly uniform (between 50 and 55 per cent of the retail price). There is no Union-wide control of distributing interests and in each centre competition is sufficiently acute to rule out all thoughts of trading on a high margin of profit. In view of these facts the reasonable conclusion to be drawn from the observed uniformity is that the service in its present form has become stabilised and that the figures quoted represent actual costs plus only a reasonable return. The only comment we can make is that economically our distributing system must be very inefficient. In America distribution claims 44 cents of the consumer's dollar (Mortenson *et al,* 1938), and even this is considered unduly high.

Seeing that we cannot agree that present distribution charges are reasonable, and seeing that there are no high profits that can be curtailed, the only way to deal with the matter is to consider a change in the system of distribution.

The present system is costly because it is wasteful. There is overtrading and overlapping. There is endless duplication of rounds and equipment; the adverse effect of these is magnified by the reduction of turn-over. We have all seen ten or twelve boys delivering milk in the same street or even in one block of flats. We can all recall instances of three or four depots situated within a fifty-yard radius. Is it to be won-

* It will be argued that it is unfair to take the price per pint as the true retail price for all milk, because large customers such as restaurant proprietors enjoy special discounts. However, these are merely intermediaries, and by the time this milk reaches the consumer (in a cup of tea) its cost is probably well above the retail price per pint.

TABLE 2.

Wholesale and Retail Prices of Milk — Data from Report (1939).

Name of Centre.	Price per Gallon (Pence).		% of Retail Price Paid to Distribution.
	Wholesale	Retail.	
Johannesburg	13.0 — 14.0	28 — 32	53.6 — 56.2
Cape Town	10.5	20 — 28	47.5 — 62.5
East Rand	12.0 — 14.0	24 — 28	50.0
Pretoria	13.0	24 — 28	45.8 — 53.6
Durban	7.0 — 8.0	28 — 32	75.0
Port Elizabeth	9.5 — 11.5	24	52.1 — 60.4
Kimberley	6.0 — 9.0	12 — 16	43.7 — 50.0
West Rand	14.0	28	50.0
Maritzburg	7.0 — 8.0	No details	available.
Bloemfontein	9.0	20	55.0
East London	10.0	20 — 24	50.0 — 58.3

dered that the service is costly! In the face of such competition, firms vie with each other in the provision of facilities such as special deliveries and credit. Moreover, for fear of losing custom, they refrain from taking action for failure to return bottles or to pay accounts. All these items increase costs enormously, and all customers, innocent and guilty alike have simply to share the burden. Often money is spent on advertising when actually the only way of really stimulating demand is by reducing costs and thus selling milk at a low price.

The American investigation referred to above points the way to the solution of this problem. The superior efficiency of large over small distributing concerns is clearly shown. During the depression the former were still capable of showing a small profit at a time when the latter were already faced with ruin. Therefore, if efficiency is to be increased, it should be by way of centralization. To be really effective, centralization should be complete, for so long as small establishments exist these constitute the marginal concerns by whose standards the price-level is set while the larger firms merely reap increased rewards from their greater efficiency.

The problem now is — if the entire distributive trade in each centre is to be vested in one body, who is to be in control of this unified service? For various reasons neither consumers' nor producers' coöperatives will fill the rôle; besides, the history of the coöperative movement in South Africa is not at all inspiring. Thus the choice must lie between the large, all-embracing commercial concern and a form of public utility company probably under municipal auspices. Donnolly and Bekker (1935) have stated the case for municipalisation. It appears that all but one of its advantages could attach equally well to commercial centralization. The exception lies in the non-profit, social service basis of municipal trading. However, it is quite possible that by superior economy the commercial undertaking may be able to take a profit without trading on a higher margin than that of

the Council. The profit need represent no more than the merest fraction of the cost of the milk; in Johannesburg a profit of 0·5d. per gallon would produce £25,000 per annum.

Until recently it may have been argued that while municipal centralization through legislation was conceivable, commercial unification of all distributing interests bristled with so many difficulties as to be beyond the pale of practical politics. However, it must be remembered that with the advent of the Marketing Act new and revolutionary possibilities have been opened up. There appears to be no reason why under the aegis of the Dairy Control Board commercial centralization should not become a practical possibility. It is not contended that this Board should itself take over the business of distribution — far from it! However, it could work towards bringing all distributors in each area closer together and finally welding this association into one big distributing company the shares of which may be held by the milkmen themselves.

If all things are equal one would be inclined to favour distribution by the commercial combine, thus leaving the municipal council aloof to continue unhampered and disinterestedly in the exercise of its functions as the guardian of public health. The Dairy Control Board's powers are sufficient to prevent any distributing combine from misusing its monopolistic trading rights.

Let us indicate what can be effected through complete unification of distribution — Donnolly and Bekker mention that the Municipal Council of Wellington, New Zealand, runs its milk department, which includes complete pasteurization, on a margin of 5d. per gallon. With our cheaper labour we should be able to improve on this. Thus with a maximum wholesale price of 11d. per gallon the highest retail price of milk should be 2d. per pint, while in many centres the price would be nearer 1·5d.

Conclusion.

It has been shown that in almost all our urban centres a 25 to 30 per cent increase of present milk-consumption is highly desirable. It appears that only a decrease of the retail price would bring about this increase in demand. At present wholesale prices cannot be reduced to any appreciable extent; but, by centralization, costs of distribution may be lowered so effectively as to result in a retail price approximately 30 per cent below that ruling at the moment, thus enabling the community to buy its additional requirements without increasing its milk cheque. Meanwhile producers' revenue would be augmented by some £146,000 per annum.

Increased consumption would have the effect of further reducing the costs of distribution, firstly because the delivery of an additional pint to any particular address has an almost negligible bearing on the outlay for this service, and secondly because the higher level of consumption would

certainly tend to smooth out daily fluctuations,* thus going far towards the solution of the surplus milk problem, at present the bugbear of the milk trade. In this way a cycle would be created — reduced prices stimulating demand and increased consumption assisting in the further lowering of costs. Meanwhile the entire dairy industry would benefit from this increased absorption of milk into the more lucrative fluid-milk trade. However, even this wonderful process has its limitations. Costs cannot be reduced below a certain figure and thus sooner or later stabilisation will be reached. Meanwhile, the apparent boom will have stimulated production, and in the course of time the surplus dairy product problem will again loom large; that is, unless adequate precautions are taken. Foremost amongst these is the necessity for impressing upon producers that heavily subsidized export is to be terminated and that surplus products will have to be sold on the world market at their "face-value." This will have the effect of forcing producers, even during the course of this boom, seriously to consider reduction of production figures. Their success in this direction will depend greatly upon the measure of assistance we veterinarians can offer. With our present equipment not one of us would relish the idea of meeting such calls — the time for a change from an academic to an economic outlook on disease is long overdue.

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* It is because people make do with minimal quantities of milk that it becomes necessary to send out for an extra pint whenever there is an unusual demand, e.g. a visitor for tea. It is the sum total of these sporadic purchases that is responsible for wide daily fluctuations in demand.

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A Report on the Treatment of an Outbreak of Strangles with Sulphanilamide.

By H. P. STEYN, Onderstepoort.

During the past six or nine months strangles has occurred as an epizootic in racing and other stables, in various parts of the country. At Onderstepoort a large number of horses were affected during the latter part of last year, but the infection was mild and although deaths did occur in untreated animals fatal cases were exceptional. At that time several horses which were patients in the hospital for other reasons developed the disease, and were treated with prontosil and sulphanilamide. However the number of animals treated was not large enough to warrant any conclusions about the value of the treatment. The present report deals with a much more virulent outbreak of the disease which was started by the introduction of an infected two-year-old Thoroughbred. This outbreak is particularly interesting as the colt which reintroduced the infection was suffering from an atypical form of the disease, and strangles was not suspected until it had broken out in the stable.

Literature. — The use of sulphanilamide in the treatment of strangles has been reported on several occasions by overseas workers, but there has been no report of this nature in South Africa. Furthermore, I am not aware of any critical investigation of the value of sulphanilamide for the treatment of strangles. It is not claimed that this is a report of a critical investigation, but the results obtained were well controlled and they furnish strong evidence in support of the claims already made by clinicians in other parts of the world.

The Recommended Dosage of Sulphanilamide.

Before proceeding to a detailed report of the outbreak, and of the treatment of individual animals, I must indicate the principles employed in computing the dose of sulphanilamide for each horse and the dangers associated with the administration of the drug.

Stableforth (1938) indicated that it was to be expected that sulphanilamide would be of value in the treatment of infections in animals caused by Group C streptococci, and that one could therefore expect favourable results from the use of sulphanilamide for strangles, metritis of mares, certain types of acute bovine mastitis, and joint-ill. The results obtained from the use of sulphanilamide, however, primarily depend upon the type of infection present and the concentration of sulphanilamide obtained in the blood. A concentration of at least 5 to 10 mgm. per 100 cc. of blood is considered necessary before the full beneficial effects of the drug can

be expected [Boddie (1938), Stableforth (1939), Bazeley *et al* (1939)]. There is not much literature available on the correct dosage of sulphanilamide in animals for different diseases. Stableforth (1938) states that horses, cows and dogs tolerate well a dose of 1 gramme per 10 lb. of body weight, even when it is continued over prolonged periods. In 1939 he recommended that an initial large dose i.e. 1 gm. per 5 or 10 lb. of body weight should be given, and thereafter a dose of 1 gm. per 30 lb. of body weight should be given every 8 hours. This dosage will ensure a concentration of over 10 mgm. per cent in the blood. At present there appears to be no indication of the minimum concentration which is required in horses for the successful treatment of strangles. Hignett (1939) recommended a dose of 3 to 6 drachms per cwt. for the treatment of equine pneumonia. The dose could be given either as a single dose or repeated on the following two, three or four days. Bazeley *et al.* (1939) consider that a concentration of 4-5 mgm. per 100 cc. of blood should be effective in most cases, and they recommend the following dosage to obtain a concentration of 5 mgm. per cent:

Weight of horse.	
750 lb.	1st day 67 gm. 2nd day onwards 45 gm.
1000 lb.	1st day 90 gm. 2nd day onwards 60 gm.
1250 lb.	1st day 110 gm. 2nd day onwards 75 gm.
1500 lb.	1st day 135 gm. 2nd day onwards 90 gm.

These authors state that there is no advantage in giving the recommended dose in two portions at an interval of 8 hours. They found, however, that there was an accumulation of sulphanilamide in the blood i.e. that the concentration of sulphanilamide in the blood gradually attained a higher level.

The initial dose of sulphanilamide recommended by Bazeley and his associates does not differ greatly from the initial dose recommended by Stableforth, but their subsequent dosage differs considerably from that of Stableforth in that they recommend one dose per day which is nearly double the single dose of Stableforth. The latter however recommends repeating the dose at 8 hourly intervals. The advantage of Stableforth's method of dosage is that the possibility of toxic effects developing is minimized.

Gildow *et al.* (1939) recommend a dose of 5 to 10 gm. per 100 lb. body weight for 3 to 10 days, in the treatment of mastitis in cows.

The Method of Administration.

Sulphanilamide is most conveniently administered as an electuary, particularly in cases of strangles when the use of the stomach tube may be contra-indicated. The administration may however be undertaken by suspending the sulphanilamide in water and drenching it or using the stomach tube, or it may be given in powder form in capsules. Subcutaneous administration is actually less effective than dosing, as it has been proved that absorption takes place more slowly and that the desired concentration in the blood is attained much later. The intravenous administration of "Prontosil soluble" does ensure an immediate concentration in the blood, but it is followed by a much more rapid elimination and is therefore undesirable. [Stableforth (1939), Bazeley *et al.* (1939)].

The main object in the therapeutic application of the drugs of the sulphanilamide type is that a concentration of 5 to 10 mgm. per cent must be attained as rapidly as possible, and that concentration must be maintained until a day or two after recovery has occurred, or until treatment is stopped. It is considered necessary to continue treatment for a period after the infection is overcome as it is claimed that relapses occur if this precaution is not taken.

Toxic Symptoms following Sulphanilamide Administration.

The important warning that no sulphates or sulphur containing foods or preparations should be given during sulphanilamide treatment must be remembered. Failure to take this precaution may cause symptoms of intoxication which could be avoided. The doses of sulphanilamide recommended in the earlier part of this article appear to be entirely safe, but as there may be great individual variations in susceptibility to poisoning from sulphanilamide, animals undergoing treatment should be kept under careful observation. The first symptoms noted seem to be sleepiness or a somewhat comatose state which may be associated with some degree of inappetence. In more severe cases, according to Stableforth, marked incoordination of movement or paralysis, inappetence, and a varying degree of coma are seen. These symptoms appear within 2 or 3 hours after excessive doses and pass off in 6 to 8 hours. In one horse (see case 3) symptoms of what was considered to be mild intoxication were seen. There was drowsiness, the animal seemed to go to sleep on its feet and would fall to its knees, slight inappetence, and a slightly swaying gait when the animal was led out of the loose box. These symptoms were seen after recovery from strangles when the temperature was normal, and they passed off in two or three days without further treatment. Gildow *et al.* (1939) stated that in cases of toxaemia, sluggishness, anorexia, pyrexia, reduced secretion of milk, staring coat, hyperpnoea, and increased pulse rate were observed in cows. One of the affected cows developed enteritis and died. The

autopsy revealed cyanosis of the musculature. Apparently purging is a common symptom following overdosing in bovines.

History of Outbreak of Strangles at Onderstepoort.

All the horses involved, except one, were Thoroughbreds.

As previously stated, an atypical case of strangles was responsible for the infection which is reported upon here. This colt, case I, was castrated in Johannesburg about 3 weeks before his admission for treatment at Onderstepoort. He was admitted on the 5th March 1940, for the treatment of an extensive castration phlegmosis with multiple abscesses in the scrotal region and neighbourhood. At the time of admission there was no sign of nasal catarrh, swelling of the intermandibular lymphatic glands nor any healed out strangles abscesses. Although it was known that the colt had come from an infected stable, it was understood that there was no active infection present in the stable at the time. I learned subsequently, however, from the veterinary surgeon who castrated the animal that it showed a slight nasal catarrh at the time of operation. The malein test (intrapalpebral route) was negative, and a diagnosis of severe wound infection following incorrect treatment after castration was made. At the time of admission accommodation in the Hospital was limited and it was impossible to follow the routine semi-isolation which is usually adopted in similar cases. On the evening of the 11th March the first horse, case 2, which was standing in the loose box adjacent to case 1 showed a marked temperature elevation. On the following morning, 12th March, case 2 showed a further slight temperature rise, and the same afternoon strangles was diagnosed clinically because of a slight cough and salivation. The clinical diagnosis was later confirmed by Dr. Robinson, who kindly examined pus taken from abscesses in case I and a later case. He identified a *Str. equi* type of organism in both specimens.

Detailed Case Reports.

Case I. (See Fig. 1 for temperature chart).

The detailed treatment of all cases is summarized in Table I. It will be observed that the temperature of this animal was of an intermittent type and above normal almost throughout the duration of the recorded observations. After admission the patient showed slight improvement for a few days but on the 12th a setback was noticed and it was feared that death would be inevitable. Treatment with sulphanilamide was commenced on the 12th, and stopped on the 27th. The details of the treatment are given in Table I. This treatment was followed by a drop in temperature and rapid general improvement. A large number of abscesses reached the stage of fluctuation, and were opened at various times after the administration of sulphanilamide was commenced. At the time of writing, this animal is improving rapidly in health and is gaining weight, but has not yet entirely recovered from the original infection.

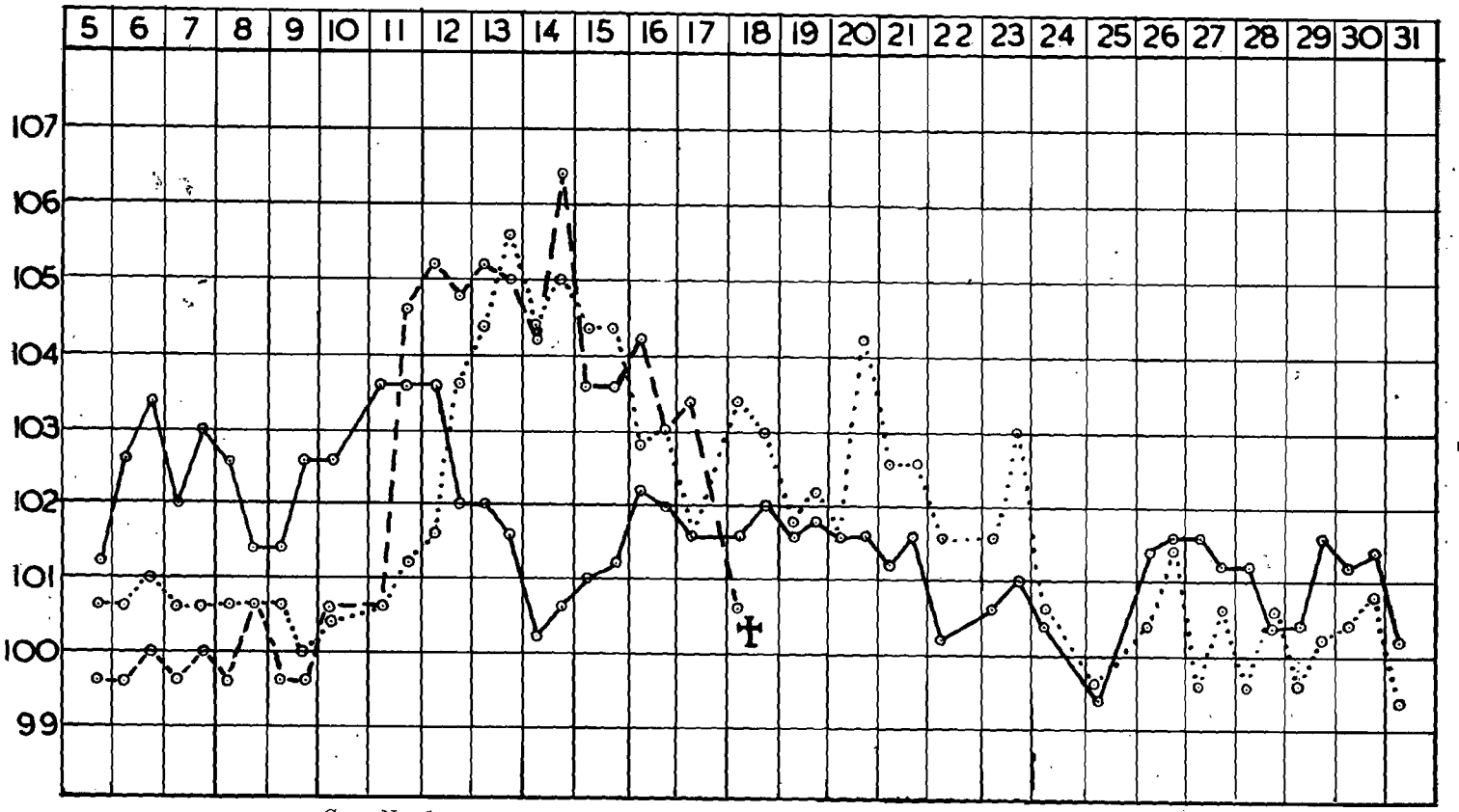

Fig. 1.

——— Case No. 1. — — — — — — Case No. 2. ..... Case No. 3.

Case No. 1. 5.3.40 Admitted for treatment. 12.3.40 Sulphanilamide treatment commenced.
Case No. 2. 12.3.40 Strangles diagnosed clinically and Prontosil rubrum administered.
13.3.40 Sulphanilamide treatment commenced.
14.3.40 Tracheotomy performed. 19.3.40 Died.
Case No. 3. 13.3.40 Sulphanilamide commenced. 16.3.40 Sulphanilamide stopped.
18.3.40 Sulphanilamide again given until evening of 19th. Thereafter dosage was repeated on the 21st, 22nd and 23rd.
23.3.40 Small intermandibular abscess opened.
Recovery uneventful.

Case 2. (See Fig. I. for temperature chart).

The disease took an exceptionally acute course in this case. The first temperature rise occurred on the 11th. Strangles was diagnosed clinically for the first time on the afternoon of the 12th, and treatment was immediately commenced by administering 60 cc. of a 2.5 per cent. solution of "Prontosil rubrum" intravenously. Fifty grammes of sulphanilamide were given in doses of 20, 10 and 20 gms. on the 13th. On the 14th the introduction of a tracheotomy tube became necessary. This was the last day on which sulphanilamide could be given because deglutition was becoming impossible. On the 15th 80 cc. Prontosil rubrum was given intravenously morning and evening. On the 16th the horse was so weak that the intravenous administration of Prontosil was considered dangerous. On the 27th a brownish and very offensive discharge mixed with blood and pus was flowing from the nose intermittently. There was practically no intermandibular swelling, and although the parotid and retro-pharyngeal region of the throat had filled out the swelling in this region was deep seated, and there was no sign of any externally accessible abscess. Intravenous and rectal infusions of saline and glucose were given at intervals from the 17th, but it had become obvious that the horse could not recover. Acute attacks of hyperpnoea, sweating and distress appeared on the 17th, and recurred at intervals thereafter until death. On the 18th pneumonia was definitely diagnosed, and it was obvious that this was of the gangrenous type. The horse died at about 5.30 a.m. on the 19th, an autopsy revealed extensive bilateral gangrenous pneumonia and pleuritis, and gangrene of the mucosa of the pharynx and larynx and adjoining parts of the mouth.

Case 3. (See Fig. I. for temperature chart).

This animal showed the first definite elevation of temperature on the 12th, and treatment with sulphanilamide was commenced on the morning of the 13th, together with all other cases which had shown a temperature rise. Treatment was therefore commenced in this and all subsequent cases immediately after a temperature elevation was noted, and before any clinical symptoms of strangles had made an appearance. The supply of sulphanilamide available was limited and the full dosage recommended by Stableforth could not be prescribed. Twenty grammes of sulphanilamide was given twice a day from the morning of the 13th to the morning of the 16th. On the morning of the 18th an initial dose of 30 gm. was given and thereafter three doses of 20 gm. each were given on the afternoon of the 18th, and morning and afternoon of the 19th, respectively. On the 21st 20 gm. were given morning and evening, and on the 22nd and 23rd a single dose of 25 gm. was given on each day.

On the 13th the animal was dull and respirations were accelerated. A cough and nasal discharge had developed by the 16th. On the 18th some difficulty in swallowing was suspected because the animal stood over its drinking trough mouthing the water for long periods without removing much water from the trough. There had been marked inappetence for a few days before this, but the animal's appetite had improved somewhat when the habit of mouthing its water was developed. On the 23rd a small abscess in the intermandibular region was opened and evacuated. On the 23rd and 24th the slight symptoms of intoxication previously described were noticed, but these disappeared entirely, and by the 28th the mare was looking very fit. A complete recovery had taken place in 16 days, after an attack of strangles which in the early stages had threatened to be severe.

Case 4. (See Fig. II. for temperature chart).

In this case a definite temperature elevation was noted on the morning of the 12th. On the 13th there was anorexia and general depression. Sulphanilamide was given in 20 gm. doses until the morning of the 16th. By then there was marked improvement, and a small abscess was developing in the intermandibular space. This was opened and evacuated on the 20th, and thereafter an uneventful recovery was made. On the 28th a colleague who saw the mare, remarked upon her excellent improvement in condition since he had last seen her a month before.

Case 5.

This was a Percheron stallion. His temperature chart and that of the following case, are not reproduced because they are similar to those of case 4 and 3 respectively. Case 5 showed a definite temperature elevation on the 12th, and treatment was commenced on the 13th; 25 gm. of sulphanilamide was given twice daily until the morning of the 16th. On the 18th a rise in temperature occurred, and 35 gm. of sulphanilamide was given in the afternoon. Two doses of 25 gm. each were given on the 19th and 21st and on the 22nd and 23rd single doses of 25 gm. each were given. Treatment was then discontinued and recovery was complete. This animal did not show any clinical signs of strangles and no abscesses developed. The diagnosis was made entirely on the history and the temperature reaction. The highest temperature recorded during the period of fever was 105.2°F.

Case 6.

A temperature reaction was noted on the 12th and the morning of the 13th, but there was a marked drop in temperature on the afternoon of the 13th and morning of the 14th. On the afternoon of the 14th the temperature was 104.2°F. and treatment was commenced. The details of treatment are given in Table I. This animal developed a slight cough but no other clinical signs of strangles. In this respect this case resembled Case 5, but its temperature reaction was more persistent and of a somewhat intermittent type, and somewhat similar to the temperature reaction of Case 3.

Case 7. (See Fig. II. for temperature chart).

This was an animal of no particular value and after 3 doses of 20 gm. of sulphanilamide on the 13th and 14th treatment was discontinued. The animal developed acute pharyngitis and respirations became laboured for two or three days from the 16th onward. At this time it was feared that tracheotomy might become necessary. The usual general treatment and nursing applied in similar cases relieved the pharyngitis somewhat. A nasal discharge and the typical cough set in at about the same time and these persisted until the 28th when it was decided to give a course of sulphanilamide treatment. This resulted in a rapid and complete clearing up of the condition in three days.

This case is particularly interesting as it acted as some sort of control to the observations made in the other cases. The real reason for stopping the treatment was to spare our available supply of sulphanilamide, but it would have been interesting to see the course of the disease if this animal had remained entirely untreated.

TABLE I.

Case No.	Age (Years)	Sex	Weight* (in lb.)	Dose of Sulphanilamide per body weight.		Total amount of Sulphanilamide given.	Remarks.
				Initial dose.	Subsequent dosage.		
1	2	Gelding	550	1 gm. per 55 lb.	1 gm. per 55 lb. twice daily.	300 gm. divided into 30 doses.	60 cc. of 2.5% solution of Prontosil rubrum given intravenously (i.v.) at commencement of treatment. Convalescent.
2	5	Stallion	±1,000	1 gm. per 20 lb. in 3 doses at 4 hourly intervals	1 gm. per 50 lb. twice daily.	90 gm. divided into 5 doses.	12.3.40 — 60 cc. of 2.5% P. rubrum solution i.v. 15.3.40 — 80 cc. of 2.5% P. rubrum i.v. morning and evening. 18.3.40 — 40 cc. of 5% P. rubrum subcutaneously. 19.3.40 — Died.
3	3	Mare	1,000	1 gm. per 50 lb.	1 gm. per 50 lb. twice daily.	180 gm. divided into 8 doses.	On three occasions larger doses than indicated were given, see case report. Recovered completely in 14 days.

* The weights recorded are within 25 lb of the actual weights and round figures were chosen for convenience. Case 2 was not weighed.

TABLE I. (Continued).

Case No.	Age (Years)	Sex	Weight* (in lb.)	Dose of Sulphanilamide per body weight.		Total amount of Sulphanilamide given.	Remarks.
				Initial dose.	Subsequent dosage.		
4	3	Mare	1,000	1 gm. per 50 lb.	1 gm. per 50 lb. twice daily.	140 gm. divided into 7 doses.	Recovered completely in 11 days.
5	3	Stallion (Percheron)	1,600	1 gm. per 64 lb.	1 gm. per 64 lb. twice daily.	385 gm. divided into 15 doses.	18.3.40 Larger does than usual given, viz. 35 gm. An uneventful recovery was made in 12 days.
6	5	Mare	1,000	1 gm. per $33^1/_3$ lb.	1 gm. per 50 lb. twice daily.	290 gm. divided into 13 doses.	There was some irregularity in the quantity of sulphanilamide given on certain days. The initial dose was repeated on the 19th, and on the 22nd and 23rd, 25 gm. were given in single doses.
7	5	Gelding	1,000	1 gm. per 50 lb.	1 gm. per 50 lb. twice daily.	180 gm. divided into 9 doses.	Recovery in 17 days was uneventful After having given this horse 3 doses of 20 gm. each treatment was stopped, and not recommenced until 14 days later. See Text. Recovery complete after 20 days.

* The weights recorded are within 25 lb. of the actual weights and round figures were chosen for convenience. Case 2 was not weighed.

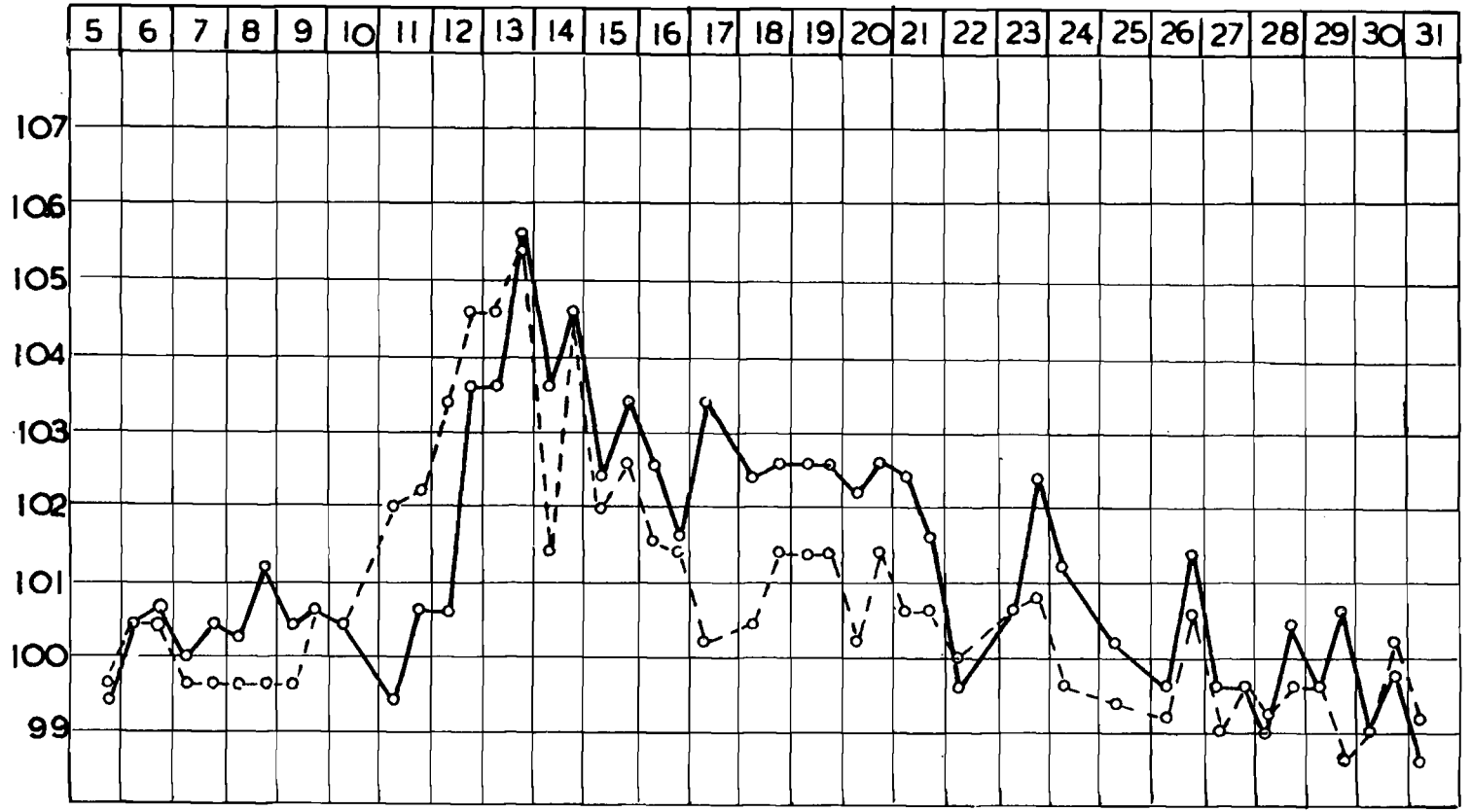

Fig. 2.

Case No. 4. 13.3.40 Sulphanilamide treatment commenced and stopped 16.3.40.
20.3.40 Intermandibular abscess opened. Recovery uneventful.

Case No. 7. 13.3.40 Sulphanilamide treatment commenced and stopped following day.
23.3.40 Nasal catarrh and cough still present, and treatment with sulphanilamide for 3 days resulted in complete recovery.

Discussion.

The results recorded do not justify drawing definite conclusions about the value of sulphanilamide in the treatment of strangles but there can be little doubt that the course of the disease was greatly modified in each case. It is particularly significant that case 7, which received a short course of treatment in the initial stages of infection developed more severe symptoms than any of the recovered animals and that the cough and nasal discharge persisted much longer than in the others. Similarly case 2 was the only animal in which treatment was delayed until a clinical diagnosis could be made and that animal died. The improvement in each case after the administration of sulphanilamide was commenced was even more marked than is indicated by the comparatively rapid drop of the temperature. The almost entire absence of abscessation is another notable feature and finally the very short period required for complete recovery must be attributed entirely to the treatment.

It is noteworthy that these apparently excellent results were obtained in spite of having employed very much smaller doses than those generally recommended, and despite having given rather short courses of treatment in the early stages of infection. This latter necessitated repeating the treatment at intervals when it became apparent that recovery had not taken place. It is possible, of course, that had the recommended dosage been employed early it might not have been necessary to continue for so long with the treatment, and consequently the recorded method of dosage might not have been economical. The possibility of finding a more successful and more economical method of dosage will be investigated if suitable opportunities occur in the future. Unfortunately the concentration of sulphanilamide obtained in the blood was not determined. The sulphanilamide which was used is para-amino benzene sulphonamide and cost approximately 16/4 per lb. The present price is unknown and is about double the pre-war price of the drug. At the price quoted the entire treatment of the seven cases amounted to approximately £2.17.0. This does not include the price of the "Prontosil" used. The retail price is considerably higher than that quoted, but even if it is double the price given the cost of treatment would not be excessive. Of course, the effect of the present war on the cost of sulphanilamide may make its use economically impossible.

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I wish to express my appreciation to Dr. E. M. Robinson for the assistance he so willingly rendered in doing the bacteriological examination of specimens for me, and to Mr. C. G. Walker and Miss G. E. Laurence for preparing the temperature charts at a moment's notice. My thanks are due to Mr. A. B. Teek, senior technical assistant, and the senior veterinary students, especially Mr. G. L. Faull, for their interest in and efficient care of all the animals.

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The Treatment of Colic in the Horse and of Hoven and Atony of the Fore-Stomachs in the Bovine with Special Reference to Liquid Paraffin and Lentin (Merck).

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The newly acquired knowledge concerning the use of liquid paraffin and Lentin in the treatment of impaction and tympany in the horse and hoven and paresis of the fore-stomachs in the bovine makes it advisable to record briefly the treatment which is at present recommended for these maladies. It is not the intention to give a full review of the literature on colic and on paresis of the fore-stomachs, but only to raise the most salient points in regard to the treatment of these conditions.

I. Colic in the Horse.

Pommer (1939) recommends that the injection of 20 to 28 cc. of *Colfin* in colic in horses should be combined with the oral administration of other drugs (liquid paraffin, oil of turpentine, etc.). *Colfin* contains extracts of *Flores chamomillae, Folia Sennae, Fructus Rhamni catharticae* and thymol, and small quantities of barium chloride. It has stimulant and sedative properties. Pommer warns against the use of drastic purgatives in cases of colic.

Weischer (1938) rightly states that more than ninety per cent of cases of colic are caused by wrong feeding. In a later publication he (Weischer, 1938a) draws attention to the danger associated with the feeding of horses on food which has been cut up finely by machines. Weischer (1938) gives a full review of his own experiences and that of his colleagues in Germany and in other countries in the treatment of colic in horses with liquid paraffin. The following are the most important points he makes:—

1. Liquid paraffin was proved to be the best of all colic remedies. Therefore the use of drastic purgatives, antispasmodics, sedatives, etc., can be dispensed with, as they are likely to do more harm than good.

2. Liquid paraffin is harmless even in large doses. Cheap brands of this mineral oil may be used, as even large quantities were found to exert no harmful effects upon horses. A small pony was given, over a period of 48 hours, 10 kg. (cir. 10 litres = circ. 2¼ gallons) of the cheapest liquid paraffin without suffering any harmful effect. Falck (Weischer, 1938) administered to approximately one hundred colic patients 5 litres of liquid paraffin daily and 800 to 1000 gm. of yeast and they all recovered. On the other hand vegetable oils (raw linseed oil, etc.) are saponified in the

intestine and cause gastro-intestinal irritation and catarrh lasting in some cases several days.

3. Liquid paraffin has no irritant action on the intestines, but softens the gastro-intestinal contents and lubricates the intestinal wall thus assisting the peristaltic movements in propelling the faeces. It also has an antizymotic action similar to that of oil of turpentine and is indicated in all cases of stomach and intestinal colic. Liquid paraffin furthermore prevents enteritis, which may follow an attack of colic. Each horse should be given 2 to 3 litres (3 to 5 pints) of liquid paraffin in one dose. This dose can be repeated once daily for a few days if no improvement sets in or if improvement does not take place rapidly. To the first dose of liquid paraffin 30-60 cc. (1 to 2 oz.) of genuine oil of turpentine (obtained from pine trees) should be added. This will enhance the antizymotic properties of the liquid paraffin.

If the colic is caused by constipation in the posterior portion of the large intestine, it is of great value to remove as much faeces as possible by hand and by enemas.

If the administration of liquid paraffin does not have the desired effects in cases of colic caused by constipation the treatment may be followed by a small dose of arecoline (0·01-0·03 gm.) injected subcutaneously. If necessary this dose may be repeated once or twice at half-hourly intervals.

In the instructions issued by Merck (Darmstadt) with their preparation *Lentin,* a subcutaneous injection of 2 cc. of *Lentin* is recommended in certain cases of colic in horses. Its use, however, could be combined with the oral administration of liquid paraffin and oil of turpentine. Aulfes (1937) injected *Lentin* in cases of constipation of the caecum and colon and found that it softens the mass satisfactorily but does not produce an increase in the peristalsis sufficient to bring about evacuation of the bowels. Hence he found it necessary to use arecoline or eserine in addition to *Lentin.* A contraindication to the use of *Lentin* is the presence of alveolar emphysema.

It is obvious that the treatment prescribed above is of particular value only in those cases of colic caused by abnormal fermentation in the gastro-intestinal tract or by constipation. In cases of severe colic 30 to 45 gm. (1 to 1½ oz.) of chloral hydrate (dissolved in approximately 300 cc. of water) should be added to the liquid paraffin. *N.B.: Tincture of opium is contraindicated in cases of colic caused by constipation.*

Mr. J. G. Boswell, B.V.Sc., of Johannesburg, informed the author that he was successfully treating cases of colic with a mixture of *Istin* (2 to 3 oz.) and chloral hydrate (1 oz.) in 600 cc. of water. Better results would be obtained if the *Istin* and chloral hydrate were administered in 2 to 3 litres of liquid paraffin.

II. Hoven and Paresis of the Fore-Stomachs.

(A) *Hoven.*

If hoven has not progressed to such a degree as to endanger life the following treatment may yield good results in many cases:—

(a) Driving the animal. Eructations frequently occur.

(b) Massaging ("kneading") the rumen, and pulling and moving the tongue by hand.

(c) The administration of antizymotics.

(i) Formalin in doses of 30 to 60 cc. (1 to 2 oz.) in 4 pints of water is recognised as a fairly successful method of treating hoven.

(ii) 120 cc. (4 oz.) of genuine oil of turpentine in a pint of raw linseed oil or, perferably, in a pint of liquid paraffin, is one of the most frequently used drugs in hoven.

(iii) Carbolic acid in doses of 2 to 4 gm. in a litre of liquid paraffin may also be used in cases of hoven, but oil of turpentine is preferable as it is safer.

(iv) Sodium bicarbonate is frequently used by stock-owners in cases of hoven. The results are often quite satisfactory as sodium bicarbonate renders the acid and fermenting ruminal contents alkaline, thus counteracting fermentation.

(d) The introduction of the stomach tube in order to allow the gas in the rumen to escape and also for the purpose of administering the formalin or oil of turpentine. It should be explained that in many cases of hoven very little or no gas will escape through the stomach tube as the gas is present in numerous small bubbles scattered throughout the contents. In these cases the administration of oil of turpentine or formalin will be beneficial.

If the animal already shows severe distress the trocar and canula should be used and the formalin or oil of turpentine could be administered through the canula into the rumen after the gas has escaped.

(B) *Atony of the fore-stomachs.*

The three chief causes of paresis and paralysis of the fore-stomachs and intestines of bovines in South Africa are (a) botulism (lamsiekte), (b) anaplasmosis, and (c) prolonged ingestion of dry and coarse feeds. Furthermore atony and hoven of the fore-stomachs are frequently seen in cases of poisoning (wilted and frosted green feeds, fungus-infected and decomposed feeds, poisonous plants, etc.) and overloading of the stomach with wheat, maize, etc. The fact that in the grass-veld areas many thousands of cattle and sheep have to exist on mature, dry, innutritious grass throughout autumn, winter, and early spring is undoubtedly the main cause of a disease known by farmers as "dry gall-sickness."

This disease appears to be more prevalent among trek-oxen than among animals which are not subjected to hard work. It is quite conceivable that exposure and fatigue are predisposing causes. The symptoms seen in cases of " dry gall-sickness " are a staring coat, progressive listlessness, anorexia, and progressive weakness, which is most pronounced in the hind-quarters and which results in a swaying gait and a difficulty in rising. In addition one observes sunken eyes, paresis and paralysis of the fore-stomachs, with severe impaction of the omasum in advanced cases, and constipation. There is usually an interval of a few days between the onset of symptoms and death. On post mortem there is nothing characteristic besides the pronounced impaction of the omasum. In many cases the ruminal contents and the contents of the large intestine are very dry and the contents of the latter are frequently covered with mucus. As a rule there is pronounced distension of the gall-bladder with normal, or viscid, flocculent, brownish bile. The above post-mortem appearances explain the name " dry gall-sickness " used by stock-owners.

Treatment of Paresis of the Fore-Stomachs and Impaction of the Omasum in Bovines.

In his studies on drugs stimulating the motility of the ruminant stomach Amadon (1930) found that:—

(a) One hundred and twenty grain (8·0 gm.) doses of tartar emetic in 2 pints (1200 cc.) of water induced a very pronounced increase in the strength of ruminal contractions within one-and-a-half-hours of administration. The stimulation of the ruminal wall lasted approximately two hours. Tartar emetic has a reflex action on the musculature of the rumen and reticulum through the abomasal mucosa.

(b) Barium chloride had no ruminotoric action.

(c) Arecoline hydrobromide administered subcutaneously in doses of 1/16 to 1/8 grain (0·004 to 0·008 mg.) is safe and has a powerful stimulant action upon the walls of the rumen and reticulum. N.B.: Doses of $\frac{1}{2}$ to 1 grain (0·033 to 0·065 mg.) are dangerous and not infrequently cause death through heart failure.

(d) Pilocarpine hydrochloride was less powerful than arecoline as a stimulant of the rumen but might be used in 1 grain doses.

(e) Eserine salicylate was the ideal ruminotoric in doses of $\frac{1}{4}$ to $\frac{3}{4}$ grain (0·016 to 0·048 mg.) administered subcutaneously. The quantities injected should not exceed $\frac{3}{4}$ grain.

(f) Lobeline sulphate was a weak stimulant of the ruminal wall.

Stammen (1934) found that there was a decrease in the ruminal contractions after injections of *Lentin* and he concluded that its use was not indicated in paresis of the rumen and atony of the fore-stomachs. He very rarely saw diarrhoea in bovines injected with *Lentin*. No abortion

occurred in 7-months pregnant cows injected with the recommended doses of this preparation. *Lentin* was contraindicated in diseases of the lungs. Benesch and Steinmetzer (1936) found that *Lentin* stimulated uterine contractions and that it had a very beneficial action in retained after-births in bovines. As *Lentin* has a stimulating action on the uterus it would be advisable, in spite of Stammen's findings, to exert care in the administration of this drug to pregnant animals.

Quin and van der Wath (1938) found that 1 mg. (1: 1000) of *Lentin* injected subcutaneously into sheep induced almost instantaneously a change in the tone of the ruminal wall and in the rhythm of the ruminal contractions. The tone rose above the normal level and the ruminal wall " may remain in a spastic state for a period of one hour or longer during which the normal movements are replaced by a series of rapidly recurring shallow, wave-like contractions resembling those noticeable during a state of incomplete tetanus." These cramp-like peristaltic contractions were present also in the large intestine. Frequent defaecation might occur within 45-60 minutes after injection. All these effects usually passed off within 2½ to 3 hours of injection.

In 1936 and 1937 the author experimented with *Lentin* (tablets and solution) upon full-grown cattle and sheep (results unpublished). Two bovines injected subcutaneously with 4·0 cc. of *Lentin* showed slight salivation and no diarrhoea; 8 cc. induced marked salivation but no diarrhoea, and 12 cc. caused pronounced salivation with a fairly watery diarrhoea which lasted about two days. Experiments were also conducted upon twenty-two sheep with *Lentin*. Sheep injected subcutaneously with 0·5 cc. of *Lentin* showed, soon after the injection, salivation which lasted 1½ to 2½ hours; there was no diarrhoea. With 1 cc. of *Lentin* salivation was marked, and in one of the three sheep injected the faeces were fairly soft. There were definite indications that *Lentin* induced slight stimulation of the ruminal wall with increase in the number of contractions. Sheep injected with 2 cc. of *Lentin* developed a fairly pronounced diarrhoea.

The following information concerning *Lentin* Merck is quoted from details supplied by Merck, Darmstadt. *Lentin* is a synthetic, uniform, and stable derivative of choline (carbaminoylcholine chloride). Through stimulation of the vagus nerve it causes increased glandular secretion and increased tone of smooth muscle, especially stimulating the peristalsis of the stomach, intestine, and uterus. Hence the action of *Lentin* resembles that of pilocarpine. The doses of *Lentin* recommended for bovines is 4·0 to 8·0 cc. subcutaneously. This dose may be repeated on the following day if necessary. The dose for sheep is 1 cc. of " Lentin for small animals."

The author feels that the administration of *Lentin* is contraindicated in cases where there is evidence of oedema and/or pronounced hyperaemia of the lungs, heart weakness and/or bronchitis.

Cases of paresis of the fore-stomachs and impaction of the omasum in bovines should be treated in the following order:—

Administer ½ to 1 gallon (2·2 to 4·5 litres) of physiological saline orally and massage (knead) the rumen, [Sigler (1939)].

(b) Administer 1 to 2 gallons of liquid paraffin to which 1 drachm (4 cc.) of croton oil or 1 pint (600 cc.) of castor oil has been added. This mixture should follow about half-an-hour after administration of the physiological saline solution. If improvement is unsatisfactory ¼ to ½ gallon of liquid paraffin to which 2-4 oz. (60-120 cc.) of genuine oil of turpentine has been added could be given daily for two days.

(c) In order to stimulate the tone and peristalsis of the fore-stomachs the administration of ruminotorics should be commenced as soon as possible. The following ruminotorics are recommended:—

(i) Eserine salicylate in ¼ to ¾ grain doses subcutaneously. This dose may be repeated three times at half-hourly intervals.

(ii) Or, one hundred and twenty grains (8·0 gm.) of tartar emetic in 2 pints of water *per os.* Half this dose of tartar emetic may be used together with eserine salicylate as described under (i).

(iii) Or, *Lentin* in doses of 4·0 to 8·0 cc. administered subcutaneously may be used instead of eserine, but the latter drug is probably of greater value.

(iv) Twelve of the following powders should be given at the rate of two daily:—

℞ Rad. Gentian ... . gm. 30·0,
Nuc. vom. .... gm. 4·0,
Ammon. carb. .... gm. 15·0,
M. f. pulv. .... Nr. XII.
Sig. two powders daily for a cow.

It is absolutely essential that the patients be given green food (oats, barley, lucerne) and ample water daily in order to prevent a recurrence of the paresis and impaction. To prevent paresis of the fore-stomachs and impaction of the omasum by the continuous ingestion of dry feeds, mature and seeded grasses or hay), green feed (green oats, barley, lucerne, etc.) should be added to the ration.

Summary.

In view of newly acquired knowledge concerning the treatment of colic in the horse, and hoven and paresis of the fore-stomachs in the bovine, recommendations are made as to the treatment of these maladies.

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A Note on the Occurence of the Ear Mite, *Otodectes cynotis*, Hering, in the Union of South Africa.

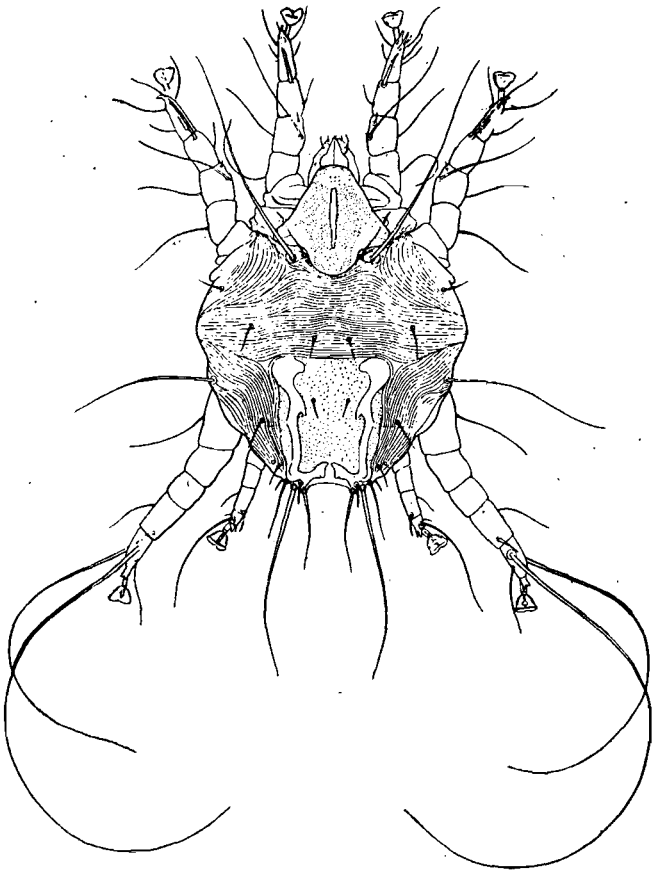
By C. F. B. HOFMEYR, Umtata, and R. DU TOIT, Onderstepoort.

The parasitic mite, *Otodectes cynotis,* Hering, belongs to the family *Sarcoptidae* and infests the ears of dogs and cats and causes intense irritation and inflammation with considerable inflammatory exudate. It has a wide distribution but, according to Bedford (1932), has not yet been recorded in the Union, although he states that it probably does occur here. Huber, (1861) says the species occurs frequently in the ears of London cats where it is responsible for an otitis generally spoken of as canker.

The genus *Otodectes,* Canestrini, 1894, contains a single species with only two known varieties. These occur in the ears of dogs and cats and one in the ears of ferrets in England. Hunting dogs in England are stated to be particularly liable to the form of otitis caused by *O.cynotis* var. *canis* which at times is believed to be responsible for a type of epileptiform-like fit. The constant shaking of the ears due to the irritation aggravates the inflammatory condition, and marked thickening and distortion, particularly of the ventral lobe of the ear, frequently results.

Otodectes cynotis is morphologically close to *Chorioptes* and, as in the latter genus, the suckers of the legs are borne on short unsegmented pedicels. In the female, tarsal suckers are present only on the first and second pairs of legs whereas in *Chorioptes* suckers are also present on the fourth pair. In the male *Otodectes,* the posterior lobes are much less salient than in *Chorioptes,* nor are the hairs upon them spatulate.

An ear scraping taken from a domestic cat during March 1940, by Mr. C. F. B. Hofmeyer, Veterinary Officer, Umtata, Transkei, showed the presence of large numbers of all stages of these small mites which in this



Otodectes cynotis, Hering. Dorsal view of male. *(After Hirst.)*

case were identified as *Otodectes cynotis* var. *felis.* This constitutes the first record of this parasite from the Union but I have little doubt that the species is probably of more common occurrence than this record indicates.

According to Hirst (1922) treatment consists of the thorough removal of all excretions from the ears and the introduction of a small quantity of

olive oil mixed with flowers of sulphur or one-tenth part of naphthol every few days until a cure is effected. In the case of dogs suffering from parasitic otitis, a warm solution of potassium sulphide (10 gms. per litre of water) should be poured twice daily into the ears. A cure generally results within a few days. Infected dogs should be isolated and the kennels disinfected.

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OBITUARY.

JOHN LEONARD MAINPRIZE.

The sudden death under tragic circumstances of John Leonard Mainprize on 1st May, 1940, came as a great shock to his many friends and colleagues. On that day he was a member of a shooting party in Sinoia district, Southern Rhodesia, and while he was sitting down resting, a gun lying by his side went off, accidentally, inflicting injuries to which he succumbed two hours later.

He was born on 1st June, 1914. After matriculating he took up the veterinary course and qualified at Onderstepoort in December, 1936. He subsequently served as Government Veterinary Officer in the Union Service for about 18 months, and in June, 1938, assumed duty as Veterinary Research Officer in Salisbury, S. Rhodesia.

Quiet and retiring by nature "Jacky" always proved himself a staunch friend. His reserve hid an inner kindness and warmth of heart and nobility of spirit. As a student he ranked amongst the foremost, combining a scholarly instinct with a practical nature.

To be in his company meant that one was entertained by a humorously critical attitude, an attitude that at times simulated despair towards the weaknesses of veterinary science, of veterinary teaching, and of general veterinary practice. It was just this attitude that hid his enthusiasm and love of work.

We feel keenly the loss of a good colleague and a loyal friend.

In September, 1939, he married Miss Mona Kewley, of Johannesburg, and our sincere sympathy is extended to her and to his parents.

The funeral took place in Johannesburg on the 17th May, and was attended by a large number of his colleagues.

A. de B.

BOOK REVIEWS.

Dr. E. A. Briggs is the author of the second laboratory manual on the dissection of the sheep's brain to be published within the last two years. His little book* is intended for junior students of zoology, but it is almost complete enough for students of elementary veterinary anatomy, from whom, on account of the backward state of veterinary neurology and its limited applications in practice, the detailed knowledge of the central nervous system, which is expected of medical students, has never been demanded. With good texts such as this and Wilkie's (1937) *Dissection and Study of the Sheep's Brain,* there is no reason why the sheep should not become the type species for the study of the brain in veterinary training. This would be an advantage, since the sheep's brain is, by comparison with that of the horse, readily obtainable and easily removed, and the brain of the average equine dissection subject is, as often as not, unsatisfactorily preserved for dissection purposes. Apart from the venous sinuses of the dura mater, the intracranial course of the cerebral nerves, and the systematic description of the sulci and gyri of the cerebral cortex, there is little omitted from Dr. Briggs' book that it is essential for the veterinary student to know about intracranial anatomy.

The author's style is not particularly original. Whole sentences or paragraphs very often have a too familiar ring and are found to have been closely paraphrased or even taken over intact from standard anatomical texts. This will not worry the student, but the reviewer confesses to having found the practice somewhat irritating.

The directions for dissection are clear and concise. The illustrations are of a high order, although more pictures of transverse sections of the brain could well have been included to supplement the descriptions. The proof-reading has evidently been carefully done, since no misprints were detected—a pleasant feature in present day scientific publication.

This book can be recommended to second and third year veterinary students as well as to students of medicine and zoology, and one is surprised that the author's modesty restrains him from doing so in his preface.

C.J.

* **The Anatomy of the Sheep's Brain** by E. A. Briggs, D.Sc., Asst. Professor of Zoology in the University of Sydney, Pp. XIII and 49, text figs. 4, pl.8. Sydney: Angus and Robertson, Ltd. Price 6/-. Obtainable at all booksellers.

Major Hamilton Kirk, who, in view of his wide practical experience, keen powers of observation and marked diagnostic ability, is probably better suited for the task than any other, has filled a big gap in veterinary literature by writing this book.†

This is a departure from the usual stereotyped class of text book in which diseases are described in a definite order (name, aetiology, symptoms and treatment). Hitherto the conscientious practitioner, when confronted with an uncommon condition, had perforce to wade through the symptomatology of a large number of diseases before being able to correlate the symptoms shown by the

† **Index of Diagnosis (Clinical and Radiological) for the Canine and Feline Surgeon with Treatment,** by HAMILTON KIRK, M.R.C.V.S. pp. viii. 572, 287 figures. Baillière, Tindall and Cox, London.

animal in question to the text, and even then was frequently left bewildered and uncertain.

The author in this work starts with the symptoms, which are taken in alphabetical order, and then briefly describes the different conditions which may produce such symptoms. The great variety of causes which may be concerned only becomes apparent on perusal of this index. Believe it or not, "abdominal distension" may be caused by no less than fourteen different conditions; there are ten other causes of "foul breath" besides pyorrhoea and gastritis; and "convulsions" may be produced by any one of seventeen different pathological states.

Explanations are also offered for many common abnormal phenomena or attitudes which are not really associated with disease, such as trembling, dogs running on three legs, barking abnormalities, etc.

The work is very well illustrated and contains about 300 reproductions of which 120 are radiographs. Under "X-ray diagnosis" are grouped all indications for radiography, and this section is a very complete treatise on X-ray technique, diagnosis and interpretation.

One feels that the author has perhaps attempted to make the work too comprehensive by including treatment, with the result that this aspect has suffered accordingly. Treatment is given for most conditions, but not for all, and there are serious omissions. For instance no mention is made of trypan blue in the treatment of canine piroplasmosis. In future editions — which are bound to follow soon — the author would do well to consider the desirability either of eliminating treatment completely or giving it much fuller consideration.

This criticism is not intended to detract in any way from the value of an excellent production that should occupy the premier place on the bookshelf — or rather in the pocket — of every student and practising veterinarian.

S. W. J. v. R.

THE ASSOCIATION.

Council Meeting held at Carlton Hotel, Johannesburg, Thursday 11th April, 1940.

Present — S. T. Amos (President), H. H. Curson, P. J. du Toit, D. G. Steyn, R. Alexander, A. D. Thomas, J. L. Dickson, A. C. Kirkpatrick, M. Sterne, J. H. Mason, V. Cooper, J. G. v. d. Wath, S. W. J. van Rensburg (Hon. Secretary).

Apologies for Absence — A. S. Canham, A. M. Diesel. These two members were represented by Dr. J. H. Mason and Mr. V. Cooper respectively. Approved.

1. *Minutes* of meeting held on 14.12.39 having been circulated were taken as read, and confirmed after attention had been drawn to the fact that Mr. Dickson's name had been inadvertently omitted from the list of those who attended.

2. *Arising from these Minutes* — Nil.

3. *Resignations* — Col. J. W. Rainey. This member had left South Africa, and it was accordingly decided to recommend acceptance of his resignation at the next General Meeting.

4. *International Veterinary Conference* — Dr. du Toit read a letter dated 1.11.39 from Prof. de Blieck, which stated that less than 25 per cent. of the expenses of the last Conference had been recovered. Decided to refer this to Finance Committee with authority to contribute £50 if this were permitted by the constitution.

5. *S.P.C.A. Prosecutions* — Correspondence with a member was read. It was decided that Council could not interfere with the contract of service between this member and the Society until after the expiry of the Contract. The Secretary was instructed to get a copy of the contract for the confidential information of Council before this matter could receive further consideration.

Dr. Curson suggested that the question of witness fees paid for professional evidence to veterinarians be again taken up. The Secretary was instructed to obtain the necessary information and to report to the next meeting.

6. *Cropping of Dogs' Ears* — The Secretary read a letter dated 5.3.40 from the secretary of the Dobermann Pinscher Club of S.A., asking Council's opinion on the Club's proposal to ask the S.A. Kennel Union to rescind the rule prohibiting the showing of dogs with cropped ears.

Considerable discussion ensued. Several members pointed out that cropping of ears seemed to be going out of fashion and that people were now used to Dobermanns with long ears. It was finally unanimously resolved that "In the opinion of this Council the cropping of dogs' ears should not be encouraged."

7. *Natal Anti-tuberculosis Association* — The President was unanimously re-elected as the S.A.V.M.A. representative on the above Association. In thanking the meeting, Mr. Amos gave an account of the excellent work done by the Anti-tuberculosis Association in combating tuberculosis.

8. *Annual General Meeting* — The opinion of Council was that it could not be definitely decided at this stage whether the General Meeting should be held or not, and the matter was left to Dr. du Toit and the Secretary.

9. *Lecturer in Anatomy* — Veterinary Faculty: The Secretary read his telegram of 1st April and letter of 3rd April, sent on the President's instructions to the Secretary for Agriculture.

At Dr. Alexander's request Dr. du Toit outlined the procedure in making appointments to the Faculty. Two channels are usually followed (a) Faculty recommends a candidate to Senate, Senate to the Council of the University, and the latter to the Minister of Education. (b) The Director of Veterinary Services approaches the Secretary for Agriculture who has to obtain the necessary funds for the lecturing allowance from the Public Service Commission.

After discussion on the case in question, Dr. Alexander proposed: "This Council of the S.A.V.M.A. confirms the request of its President to the Secretary for Agriculture to take whatever steps he considers necessary to delay the appointment of lecturer in anatomy in the Veterinary Faculty of the University of Pretoria, pending an enquiry by himself into the circumstances under which the recommendation of the Faculty was reversed by the Senate of the University."

Dr. Thomas seconded and the motion was carried.

Arising from this Dr. Alexander further proposed:— "This Council of the S.A.V.M.A. earnestly requests the Minister of Agriculture to consider the

advisability of appointing immediately a commission to enquire into veterinary education in South Africa with special reference to:—

(a) The relationship between the Veterinary Faculty and the Pretoria University.

(b) The relationship between the Veterinary Faculty and the Department of Agriculture.

(c) The relationship between the Veterinary Faculty and the Veterinary Profession."

Dr. Mason seconded, and after full discussion the motion was passed unanimously.

Dr. Alexander suggested that the Secretary should write to the two candidates concerned informing them that the discussions on these proposals were confined entirely to the priciples involved and took place without any personal consideration. Agreed.

The meeting concluded at 6.45 p.m.

S. W. J. van Rensburg,
HON. SEC.-TREAS., S.A.V.M.A.

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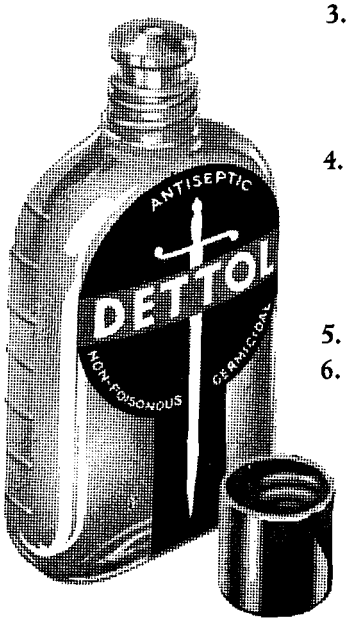

3. Dogs and other animals usually remain quiet during treatment with Dettol as the solutions neither irritate nor sting raw wounds.
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Jl. S.A.V.M.A.
XI(3) : 75 - 97.
1940.

Animals and Poison Gas Warfare.

By DOUW G. STEYN, Onderstepoort.

Contents.

In this article an attempt is made to review the literature on war gases. It is calculated that sixty-five to eighty million men participated in the 1914-1918 War. Of these, approximately ten millions were killed. Of the sixty-five to eighty million men, about one million were 'gassed', and of these, approximately one hundred thousand died.

It appears that, although war gases may not materially affect the result of a war, their effect should not be underestimated. Levinstein (1936) states that mustard gas was used with great success by the British when they stormed the Hindenburg Line in the Great War.

In this article particular attention is paid to poison gases, although it should be mentioned that in future wars incendiary bombs will probably be used on a large scale and as a result man and animal will be burnt by (1) pieces of phosphorus, which through the terrific explosions may be blown on to the skin and clothes, (2) superheated bomb fragments and (3) phosphoric acid. Keilholz (1937) recommends that such burns should be washed with a 5 per cent. solution of sodium bicarbonate or a 3 per cent. solution of sodium carbonate and then be treated with a 2.5 per cent.

tannic acid ointment prepared with vaseline. If a large part of the body or the whole body is contaminated, the entire animal should be washed with the above solutions of sodium bicarbonate or sodium carbonate. Detailed information concerning the treatment of burns is obtainable in the following publications: Bettman (1936), Blair *et al.* (1938), Branch (1938), Clark and Cruickshank (1935), Editorial (1930), Editorial (1938), Fantus and Dyniewicz (1937), Fasal (1937), Kissmeyer (1936), Russel (1936), Stewart (1937), Turner (1935), Wells (1933) and Wilson *et al.* (1936).

It should be kept in mind that in incendiary bomb explosions carbon monoxide is evolved, hence the danger of poisoning with this gas, especially in enclosed spaces.

I. — SMOKE SCREENS AND POISON GASES USED IN WARFARE.

The word 'gas' is really a misnomer as almost all the poisonous substances used in 'gas' warfare are either liquids or solids which pass into a state of very minute division or into a gaseous state at the moment of explosion of the shells.

In 1915 (Great War) the Germans released chlorine and hydrocyanic acid gas from cylinders in their trenches (Marshall, 1934). This method of gas warfare was soon dropped as (1) poisonous concentrations of the gases are very difficult to achieve in this way owing to rapid diffusion into the air, and (2) climatic conditions are frequently unfavourable. Dry weather and light winds blowing in the direction of the enemy are essential for the success of this method of gas warfare. Moreover, there was also the danger of the prevailing wind turning and directing the gas towards those who were using it. The result was that poison 'gases' were incorporated in high explosive shells and this method of gas warfare will most probably be continued in future wars. It is possible that some of the liquid poisons (mustard gas) will also be sprayed from aeroplanes.

According to Richters (1936) some fifty or more 'gases' were used in the Great War but it is unlikely that more than about a dozen will be used in future wars, as the majority are not suitable for this purpose. The use of poisons in warfare does not depend only on their degree of toxicity. Factors like specific gravity, volatility, solubility in fats and liquids, rate of diffusion, their capacity to adhere to surfaces, hydrolytic dissociation, and the toxicity of their products of dissociation play an important rôle in rendering poisons suitable as war 'gases'. In addition to this there are various technical points to be considered. The principal properties of an efficient war gas should therefore be (1) rapid action (most important), (2) great volatility and high specific gravity resulting in slow diffusion and tendency to keep to the ground level, (3) solubility in aqueous solutions, fats, and liquids, and (4) capability of being absorbed easily by the body. The action of a gas depends upon its concentration in the air and the duration

of its action, hence the coefficient of toxicity (introduced by Haber) is $W = c \times t$ (c = concentration in mg. per cubic metre of air and t = minimum time in minutes in which symptoms are caused). This formula has only a relative value and is not infallible as results with very highly lethal concentrations and very low concentrations of gases become irregular. In these extreme cases (very high and very low concentrations) a new factor enters, namely, that concerning the difference in the periods during which the poisons are detoxicated in the body. With highly lethal concentrations of gas in the air death occurs so rapidly that there is practically no time for the processes of detoxication (fixation, oxidation, reduction, etc.) in the body to exert their effects. On the other hand, with low concentrations of gas there is sufficient time to allow detoxication to protect the system from poisoning for a certain period at least, if not indefinitely, as in the case of non-toxic concentrations of gases. Hence, the introduction of a constant detoxicating factor *e* in Haber's formula renders the results of the calculation more reliable [$W = (c - e) \times t$].

A. Smoke Screens (Artificial fogs).

The ability of a substance to produce 'fogs' depends on its hygroscopic properties when suspended in a state of fine division in the air. Substances employed in the production of smoke screens in order to hide forces from view include preparations containing compounds of phosphorus, zinc, arsenic, antimony, tin, titanium, sulphur and ammonia (Kulesza, 1938). Gorniewicz (1938 a) states that two kinds of smoke 'candles' are used in military manoeuvres, namely, one containing lead chloride, chlorates, and compounds of nitrogen, and the other containing distilled pitch, saltpetre, and sulphur. Animals grazing in fields on which lead, arsenic, antimony, and zinc compounds have been used in the production of smoke screens may become poisoned by the vegetation they eat and the water they drink.

According to Richters (1936), chlorosulphonic acid and sulphur trioxide (forming fuming sulphuric acid in moist air), zinc dust (Berger's mixture — French) and zinc with carbon tetrachloride, and tin and silicon tetrachloride are used in the production of artificial fogs. On the whole no harm is done to animals by these fogs as they are rarely used in harmful concentrations.

B. Poison Gases.

Maass (1934) mentions sixty-two American, German, British, French, Italian, Austrian, and Russian war gases, and gives details about their chemical and toxicological properties. There have been rumours that German chemists have discovered an extremely toxic gas 'capable of wiping out communities at a time' (Editorial, 1935). On the other hand a number of authorities on war gases maintain that it is unlikely that poison gases will ever become a very formidable weapon as their success depends on a

large number of factors [specific gravity and volatility of the gases, climatic conditions (rain, wind, cloud, etc.)], and it is very difficult to obtain a set of circumstances all of which favour the achievement of toxic concentrations of gases in the atmosphere. *Wind and rain are the greatest enemies of gas warfare.* In addition to these limitations, protection against gases by means of gas masks and protective clothing has been developed to a considerable extent. Perhaps it is not out of place here to refer to a method of camouflage used by the Germans in the Great War. They fired shells which gave off voluminous smoke and a vile stench, but which did very little harm. As soon as they thought that the enemy had become accustomed to the smoke and stench and had become careless in the use of gas masks, they fired the same type of shell containing extremely toxic gases (Editorial, 1935).

According to Flury (1938), poison gases damage enzymes in the body and consequently must be regarded as cell poisons. It is not really the war gases as such, he states, which poison the cells but it is their breakdown products which are formed after entrance into the body. In addition to the preliminary toxic effects, harmful effects are exerted by the breakdown products, the so-called '*körpereigene*' (endogenous) substances of the cell and cell nucleus. For the formation and accumulation of these poisons oxygen-deficiency is responsible, hence poisoning with war gases is nothing more than asphyxia.

Hansel (1935) states that chronic bronchitis and its sequelae (emphysema and bronchiectasis), tuberculosis, and bronchial asthma (rare) may follow on poisoning with war gases.

Only those gases which are likely to be used again in the present conflict will be discussed.

(1) *True Poisons.*

Carbon monoxide and hydrocyanic acid are the only two poisons which need be considered here. It has already been mentioned that carbon monoxide develops in explosions and is dangerous in enclosed spaces. Since, due to very rapid diffusion, it is not easy to achieve highly toxic concentrations of these two gases in the air, it is improbable that they will be used alone. It is, however, possible that they will be used mixed with other gases.

Horses suffering from acute carbon monoxide poisoning show giddiness, incoordination of movement, and paralysis of the hind limbs. Animals which recover are liable to secondary pneumonia (Henry, 1935). As treatment, bleeding followed by blood transfusion is recommended; also oxygen inhalations (oxygen with 5 to 10 per cent. of carbon dioxide) and injections of lobelin, cardiazol, coramine, etc. Intravenous injections of " chromosmon " (0.25—1 per cent. methylene blue and 10 per cent. glucose solution) are recommended in poisoning with carbon monoxide (Deutsch und Weiss, 1934).

Five thousand parts of carbon monoxide per million parts of air (= 6.0 mg. per litre) cause death in man after 5 to 10 minutes inhalation, and 2,000 parts per million parts of air cause symptoms of poisoning in one-half to one hour (Maass, 1935).

Very high concentrations of hydrocyanic acid in the air cause almost instantaneous death from heart and respiratory failure. Smaller but toxic concentrations cause convulsions, spasms, and vertigo followed by stupor and paralysis and death from respiratory failure. On post-mortem examination pronounced general cyanosis and hyperaemia of the lungs are usually to be seen.

Air containing 200 parts of hydrocyanic acid per million has, when inhaled by man, fatal effects in 5 to 10 minutes, while 50 p.p.m. was found to exert no effects upon man when inhaled for half to one hour (Maass, 1935). In the Great War the French used hydrocyanic acid under the name of *vincennite.*

Intravenous injections of sodium thiosulphate are very effective in cases of hydrocyanic acid poisoning (10 to 15 g. of sodium thiosulphate in 20 per cent. solution intravenously for a horse).

Hydrogen phosphide was also used but was discarded for the same reasons as were carbon monoxide and hydrocyanic acid.

(2) *Asphyxiants (Suffocating gases).*

The gases described here are grouped under the Green Cross Group (German).

(a) Chlorine (Cl_2).—It is unlikely that it will be used owing to the rapidity of its diffusion.

(b) Phosgene ($COCl_2$). — Known as 'collongite' (French), 'C.G.' (English) or 'C.G. Stoff' (German). It is a colourless irritant gas damaging the alveolar walls (oedema of the lungs) and affecting, to a lesser extent, the eyes and upper air passages. It has the smell of musty hay.

Death ensues within 5 to 10 minutes when human beings inhale 50 parts of phosgene per million parts of air. One part of phosgene per million parts of air can be inhaled with impunity for from half to one hour (Maass, 1935). According to Herlant (1939), 125 parts of this gas per million parts of air (= 0·5 mg./litre or 0·5 g./cubic metre of air) is fatal within one minute.

Moisture decomposes phosgene into carbon dioxide and hydrochloric acid.

(c) Palite (Cl.C.OOCH.Cl, Monochlormethyl-acetic ester). — It is also known as 'C-stoff' and is very irritant to the eyes and respiratory organs (Maass, 1935).

(d) Surpalite ($Cl.C.OOC.Cl_3$).—Known as diphosgene or 'Perstoff'. Surpalite is an oily liquid that evaporates slowly and has a choking smell.

When brought into contact with water, it immediately decomposes, like phosgene, into hydrochloric acid and carbon dioxide.

(e) Chlorpicrin ($C.Cl_3NO_2$, trichlornitromethane). — Known as vomiting gas, 'Klopp' (German) and 'aquinite' (French). It is a colourless liquid which is very volatile and affects the eyes more than the lungs. It is soluble in organic solvents. Fifty parts of chlorpicrin per million parts of air (= 340 mg./ cubic metre) is fatal when inhaled for 10 minutes, whilst air containing 1 part of this substance per million parts is harmless (Maass, 1935). Chlorpicrin is stable and insoluble in water. After absorption it acts on the heart and central nervous system, and also causes haemolysis and methaemoglobinaemia. It causes fairly severe irritation of the eyes.

(f) Nitrogen derivatives (nitric and nitrous oxides) are very irritant, especially to the lungs (similar to phosgene). There may be nitrogen shock, dilatation of the heart, fall in blood pressure, and methaemoglobinaemia.

Symptoms of Poisoning with Asphyxiants.

Peracute poisoning: Animals fall to the ground and die immediately, or within an hour, from asphyxia.

Acute poisoning: In acute poisoning the symptoms and post-mortem appearance caused by the above asphyxiants are very similar and are as follows. Irritation of the mucous membrane of the nose, throat, larynx, and bronchi, and to a less degree of the eyes, and oedema of the lungs. When phosgene ($COCl_2$) is inhaled it is dissociated in the lungs into hydrochloric acid and carbon dioxide. The liberated hydrochloric acid produces irritation of the alveolar epithelium resulting in effusion of moisture and oedema of the lungs. Keilholz (1937) states that the $COCl_2$ (phosgene) molecule as such is approximately 800 times more poisonous than the hydrochloric acid molecule. There is spasm (constriction) of the bronchial muscles, and the lungs are enlarged about 5 or 6 times. Phosgene, chlorpicrin, and the organic arsenicals induce irritation of the nerve endings in the bronchi causing reflex vaso-dilatation, thrombosis and modification of permeability of certain parenchymatous cells of the lungs leading to oedema and eventually asphyxia (Cordier, 1937). The severe oedema of the lungs results in concentration of the blood, which has a tarry and chocolate-like appearance. There is also dilatation of the ventricles of the heart, raised blood pressure (after a preliminary fall), albuminuria, cylinderuria, glycosuria, and swollen spleen and liver.

Within one hour after the commencement of inhalation of toxic concentrations of the above gases, horses develop the following symptoms: restlessness; coughing; muscular tremors; cold sweats; pain; unsteady gait; apathy; head lowered and stretched forward; pronounced laboured respiration (dilated nostrils); copious, dirty, blood-tinged nasal discharge

which later forms scabs; oedema of the lungs; temperature 104-105°F.; injected conjunctivae; pulse 80 to 100 per minute, at first full and strong, later irregular and hardly perceptible; finally coma and death.

If lower concentrations of these gases are inhaled by the animals, symptoms commence with a dry cough, but the above symptoms may be absent or may appear in a mild form from within 6 to 24 hours after the gas attack. There may be slight oedema of the lungs and laboured respiration and degenerative changes (fatty) in the heart, liver, and kidneys. The most important complications are broncho-pneumonia and albuminuria.

According to Muntsch (1937) animals which inhale air containing quantities of phosgene under the toxic limit do not develop chronic poisoning.

Dildine (1939) states that horses are approximately as susceptible as man to lung irritants.

Cats and rabbits poisoned with diphosgene did not show a higher degree of susceptibility to anaesthesia with evipan sodium than normal animals. Whether the animals were anaesthetised or not, the mortality from diphosgene was the same (Hecksteden, 1937).

Dowgiatto (1939) states that severe poisoning of horses with phosgene is followed by definite changes in the blood which are valuable in the early diagnosis of poisoning with this gas. These blood changes are detectable in the latent stages of the disease, and in later stages the blood changes are of a nature compatible with the clinical symptoms. In the first phase (during the first hour after exposure to the gas) there is a decrease in haemoglobin (71 per cent.), a slight decrease in the number of erythrocytes (6.1 million per c.mm.) and also a decrease in the number of leucocytes. Blood sedimentation rate is accelerated and also the pulse and respiration rate. The temperature is slightly elevated. "In the 2nd stage (24 hours after exposure) there is an increase of haemoglobin (up to 117 per cent.), the number of erythrocytes is raised (up to 13.8 millions per c.mm.) and of leucocytes (up to 14,400 per c.mm.); the sedimentation rate attains 139 mm. in 24 hours, respiration 60 and pulse 80 per minute; temperature 2.3°C above normal. During the 3rd stage in animals that recover (6 days after exposure) there is a gradual return to the normal, except that the temperature remains a little high. During the illness neutrophiles are prevalent, whereas the number of eosinophiles and lymphocytes is markedly lower," (Dowgiatto, 1939). Experimenting upon mice, rats, guinea-pigs, and rabbits, Schmidt and Podloucky (1937) found no difference between the size of the red blood cells of normal animals and of those poisoned with phosgene.

Post-mortem Appearances (Horses).

Peracute cases: Pronounced congestion of the larynx, trachea, and bronchi. There is no oedema of the lungs.

Acute cases: Oedema and emphysema of the lungs due to irritation of the vagal nerve endings, subpleural haemorrhages, damage to alveolar epithelium and peri-alveolar capillaries (rapid transudation of plasma).

Treatment.

As soon as it is discovered that a gas attack has been made, gas-masks should be fitted on the animals [see Anti-gas Organisation (11)].

Treatment on the following lines should be applied immediately:—

(1) Rest is very important. However, it is obvious that animals should be removed from areas contaminated with gas. The moving should be done very slowly and along the easiest routes, as exertion means increased respiration, resulting in the inhalation of larger quantities of poison gas.

(2) Bleeding may be of value in removing some of the absorbed gas from the system and in preventing oedema.

(3) Oxygen inhalations are essential.

(4) Heart stimulants (caffeine, coramine, cardiazol, strophantine, intravenously, camphorated oil intramuscularly). It is stated (Keilholz, 1937 a) that digitalis and adrenalin are not suitable heart stimulants in poisoning with these asphyxiating gases.

(5) To allay coughing give Dover's powder, codeine phosphate, and warm inhalations of turpentine, balsamum peruvianum, menthol, tincture of benzoin, or weak alkalis. Avoid morphine and chloral hydrate as they depress the respiratory centre. Lobeline could be given to prevent respiratory paralysis.

(6) In cases of pneumonia, symptomatic treatment should be applied (atropine, etc.).

(7) Eyes—alkaline ointments as in cases of poisoning with tear gases (see 1 B_3).

(8) Since some asphyxiants may contain arsenic, the possibility of arsenical poisoning should also be considered. Intravenous injections of sodium thiosulphate should be given (horse — 5·0 to 10·0 g. in 10 to 20 per cent. sterile aqueous solution).

(9) Give intravenous injections of calcium borogluconate in 10—20 per cent. solution (horse, 30-60 g. in sterile water) to combat pulmonary oedema. In man intravenous injections of 1.0 g. of calcium bromide in 20 cc. of a 35 per cent. glucose solution are recommended (Leschke, 1932).

(10) Richters (1936) recommends the intravenous injection of 2 to 3 litres of a 20 per cent. solution of glucose for the horse.

(11) Diphosgene is decomposed by moisture, sodium bicarbonate, and ammonia water.

(12) Chlorpicrin is most easily rendered harmless by sulphurated potash (liver of sulphur—K_2S_3 + $K_2S_2O_3$) in soap solution.

(3) *Lachrymators* (*Tear gases*). (White Cross Group—German).

The most important poisons in this group are :—

(a) Bromobenzylcyanide ($C_6H_5CHBr\text{-}CN$) known as 'B.B.C.' and 'C.A. Stoff' (German) and 'camite' (French).—It is a brownish oily

liquid. In the horse 5 mg. of this liquid per cubic metre of air causes irritation of the eyes in 5 minutes; 5 mg. per cubic metre of air allowed to act for 1 hour causes slightly more severe irritation of the eyes but this disappears after a few hours; 500 mg. per cubic metre of air permitted to act for 15 minutes produces a pronounced flow of mucus from the nostrils, and laryngitis lasting for a few days. In the dog the symptoms are similar, (Andreoni, 1939). Bromobenzylcyanide is the most pronounced eye irritant of the lachrymators.

(b) Chloroacetophenone ($C_6H_5COCH_2Cl$) (phenacylchloride), a colourless crystalline substance known as 'C.A.P.' and 'C.N.'—With a concentration of 50 mg. of this substance per cubic metre of air there are signs of irritation in the eyes and nose in 5 minutes. If horses are allowed to remain for 1 hour in such air there are signs of fairly severe irritation of the eyes and nose which disappear after a few hours; 500 mg. per cubic metre of air allowed to act for 15 minutes cause similar symptoms of irritation and also laryngo-tracheitis and bronchitis which last for about a week, (Andreoni, 1939).

(c) Bromoacetone (CH_3COCH_2Br) and brominated ketones. — They are liquids. Bromoacetone is termed 'B-Stoff' by the Germans and 'martonite' by the French. One milligram of bromoacetone per cubic metre of air causes transient irritation of the eyes. If a concentration of 100 mg. per cubic metre of air is allowed to act on horses for half-an-hour, pronounced irritation of the nose and eyes sets in; there is also general excitement, and a severe laryngitis which lasts for about 14 days. The symptoms produced in the dog are similar but less severe, (Andreoni, 1939). If dogs are left in the latter cencentration (100 mg./cubic metre of air) for 1 hour, they develop acute laryngo-tracheitis or croupous tracheitis and pneumonia, and may die, (Andreoni, 1939). Andreoni states that bromobenzylcyanide, chloroacetophenone, and bromoacetone irritate the air-passages (especially the upper air-passages) of the horse and dog more than they irritate the eyes.

(d) Bromoxylol (Br. $H_2C.C_6H_4CH_3$).—It is a liquid and known as 'T-Stoff' by the Germans. It is a fairly severe irritant of the eyes but is not a very poisonous substance.

(e) Benzylbromide ($C_6H_5CH_2Br$).—It is a liquid and is known as 'cyclite' (French) and 'T-Stoff' (German). It causes irritation of all mucous membranes, but affects chiefly the eyes.

(f) Chlorpicrin (CCl_3NO_2) (Trichlornitromethane). — It is an irritant to mucous membranes and also an asphyxiant (see I B 2).

(g) Ethyliodoacetate ($C_2H_4I.OOC.CH_3$).—A brown liquid, like bromobenzylcyanide, with a high boiling point; vaporization is slow and consequently these two substances are fairly persistent (for several days). Ethyliodoacetate has a smell like that of 'pear drops' and is known as 'K.S.K'.

The above poisons are used mainly to disorganise the enemy troops, and

it is unlikely that they will be used in cities because, as a rule, they produce only transient irritation of the eyes and upper air-passages associated with pain in the affected organs, and pronounced lachrymation. Parree (1937) states that 'tear and nose-irritant gases appear to have very little effect on horses.' Further, Henry (1935) states that horses are affected only by very high concentrations of tear gases. According to Herlant (1931), tear gases have been used in a concentration of 0·3 g. per cubic metre of air.

Symptoms.

Under field conditions the tear 'gases' cause fairly severe irritation of the eyes, which is accompanied by piercing pains, lachrymation, and possibly also by irritation of the nose, larynx, and trachea.

It is only in very high concentrations in the air that they are lung irritants (congestion, oedema, pneumonia). In these concentrations they may also induce a tingling sensation in the skin. If their action on the eyes is prolonged, conjunctivitis is produced. These 'gases' are insoluble in water but are soluble in fats and fat solvents.

Treatment.

(1) Gas-masks should be fitted immediately and the animals removed from the affected areas.

(2) Irrigate the eyes and nose with a 2 or 3 per cent. aqueous solution of sodium bicarbonate. It is essential not to rub or wipe the eyes, but to dry them, after irrigation, by dabbing with absorbent cotton wool.

(3) If the eyes are very painful, instil 1.0 per cent. cocaine hydrochloride solution.

(4) In cases of chemical conjunctivitis, an alkaline ointment (2 per cent. sodium bicarbonate in vaseline) should be used. This ointment may also be used when the skin is irritated.

(5) For treatment of irritation of the lungs, see treatment of poisoning with asphyxiants (I B 2).

(4) *Vesicants (Skin irritants).*

'Mustard' gas and 'lewisite' and their derivatives are the most important poisons of this group of blistering gases, known by the Germans as the 'Yellow Cross Group'.

(a) Dichlorethylsulphide $[S \begin{matrix} -Cl_2 \\ =(C_2H_4)_2 \end{matrix}]$ known as 'mustard gas'. 'Yperite', 'Lost' (German). — Mustard gas is known as the 'king of gases'. It is a heavy oily or straw-coloured liquid in the pure form and a dark-brown liquid in the crude form. Mustard gas has the smell of garlic and to some extent also of mustard and onion. Unfortunately detection of poison gases by smell is rendered impossible as other gases (viz. tear

gases) are used as camouflage. The boiling point of 'mustard gas' is 417°F., hence it evaporates slowly and is very persistent. It is scarcely miscible with water, but is soluble in fats and lipoids. It is a very stable 'gas' and has tremendous powers of penetration. It does not, however, penetrate china, porcelain, glass, or metals, but easily finds its way into leather and rubber. It is a most severe irritant, a droplet the size of a pin's head causing a blister as large as, or larger than, a half-crown piece. It is a poison of a very insidious nature in that its effect may be delayed up to 24 hours. From experiments conducted on rabbits and guinea-pigs Hasskó and Fülöp (1938) suggest that a histamin-like substance possibly plays a rôle in the production of oedema in mustard-gas-poisoning. However, the action of this gas on cells, especially considering the latent period that elapses with low but toxic concentrations, is still a mystery. It is possible that the hydrochloric acid split off from mustard gas may be, at least partly, responsible for the lesions produced. Mustard gas, as such, has practically no effect upon the hydrolysing enzymes of proteins, fats, and carbohydrates, (Lang, 1938 and 1938 b).

Muntsch (1937) found that rats, compared with other animals, are fairly resistant to the effects of mustard gas. He established that rats exposed for 10 minutes to 0.005 mg. of this gas per litre of air showed quite pronounced lesions. According to Lustig (1935) 0.07 mg. of mustard gas per litre of air is fatal.

Wester (1937 a) states that mustard gas is harmful (serious conjunctivitis in man) in concentrations (0.001 mg./litre of air) which cannot be detected by smell. In concentrations of 0.0013 mg./litre of air this gas is detectable by its garlic or onion odour.

(b) Chlorovinyldichloroarsine ($CHCl{:}{:}CH.AsCl_2$) known as 'dew of death', 'Supergas' (German), and 'lewisite', It causes severe irritation of all the mucous membranes and of the skin. It acts more rapidly than mustard gas but the lesions heal in a shorter time. In the pure and fresh state it is a colourless, light oily liquid with an odour of geraniums. After a few days its colour darkens. It is quickly destroyed by oxidation. Alkaline solutions (washing soda) break it down almost instantly and in the presence of moisture it dissociates very rapidly. Because of these disadvantages it was eliminated from warfare, (Richters, 1936).

(c) Leipert (1938) states that new skin irritants are formylchloridoxime, cyanoformylchloridoxime, phosgenoxime and *n*-heptoylvanillyl (English pepper gas), and that probabilities are tetraethyl lead, diethyltelluride and nitrogentrioxyfluoride. The gas masks used at present are effective against all these poisons.

Symptoms of mustard gas poisoning.

Mustard gas is the most dangerous of all war gases and is a severe cell poison, especially of the epithelial cells, blood, capillaries, and nerves, and affects the skin, eyes and air-passages. The rate of coagulation of the

blood is accelerated and a preliminary rise in the red blood cell count and haemoglobin content is followed by a marked drop. There is pronounced neutropenia and lymphocytosis and slight eosinophilia, and disintegration of the nucleus of neutrophiles is frequently seen. Anaemia persists for long periods, (Drews, 1939). If the eyes are exposed to concentrated vapours there is no immediate severe irritation, except possibly lachrymation. Subsequently severe irritation sets in, accompanied by marked swelling of the eyelids and temporary blindness within 24 hours. The symptoms of mustard gas poisoning may be summed up as follows. Severe inflammation of the eyes; oedema of eyelids, conjunctivitis, keratitis, iritis, ulceration and perforation of cornea; violent itching and swelling and redness of the skin do not appear until about 2 hours after exposure and blisters appear only after 12 to 24 hours, in severe cases, the tender parts of the skin being first affected; these skin lesions may become severe only 5 to 6 days after the gas attack. Necrosis follows oedema of the skin. There may be moist eczemata. These lesions take a long time to heal (often weeks). It is stated that the horse's skin is 4 to 5 times more sensitive to mustard gas than that of man, cattle, sheep, and dogs (Richters, 1936; Andreoni, 1939b). In experiments carried out in Holland it was found that horses are at least as susceptible as man to the effects of mustard gas except that instead of causing blisters the gas produce necrosis of the skin (Parree, 1939). It is obvious that there is a difference in individual and breed susceptibility in animals. The skin of Negroes is less susceptible to mustard gas than that of Europeans. If contaminated food and water are ingested, blistering of the lips, mouth, pharynx, and oesophagus will occur, and gastro-enteritis will be produced. The air-passages do not respond immediately they are exposed to air contaminated with mustard gas, and symptoms of irritation may appear only 12 to 24 hours after exposure if the concentration of the gas is not very high ; severe irritation, with lesions (hyperaemia, blisters, ulceration, and necrosis) of the nasal mucous membrane down to the alveoli; purulent bilateral nasal discharge; enlargement of the submaxillary lymphatic glands; salivation; yawning; neighing; choking cough; laboured breathing; apathy; bronchitis; broncho-pneumonia; rapid rise in temperature; toxic hæmorrhages in the kidneys (urine may contain protein, blood, and leucocytes), gastro-intestinal canal, and brain; feeble and irregular pulse; anæmia may persist for years; unsteady gait; ataxy, paralysis of the extremities; muscular spasms; loss in condition; death may follow in 24 to 36 hours from heart-failure. There may be sudden deaths. The period of convalescence is very long.

Post-mortem appearances.

These are obvious from the symptoms described (ulceration and necrosis of mucosa of the air-passages, and gangrenous pneumonia). If death is due to the ingestion of contaminated food and water there will be ulceration and necrosis of the mucosa of the mouth, pharynx, larynx, and gastro-intestinal canal (ulcerative and necrotic gastro-enteritis).

Treatment.

Treatment should be commenced as soon as possible.

(1) Fit on gas-masks and do not move animals, or if necessary, do so very slowly. It is necessary, but admittedly very difficult, to protect the whole body of the animals (see II. B 3).

(2) Irrigate the eyes and mucous membranes freely with warm water, normal saline, or sodium bicarbonate (2.0 per cent. sterile aqueous solution). A few drops of liquid paraffin, cod liver oil, or castor oil (5 drops, 3 or 4 times a day) will prevent the lids from sticking together. For man, Bickerton (1940) recommends the use of 2 per cent. homatropine in oil in severe inflammation of the eye (4 drops, 3 times daily). He states (a) that one should not use cocaine, unless compelled, because the pain in the eyes causes a beneficial flow of tears which bathes the eye; and (b) that decicaine and procaine are less harmful than cocaine. No pads or bandages are to be placed over the eyes. In bad cases a 2.0 per cent. alkaline (sodium bicarbonate) ointment should be used.

(3) Wash the whole body with warm aqueous solution of sodium bicarbonate (3 to 4 per cent.), bleaching powder, soap, hydrogen peroxide or potassium permanganate (0·05 per cent.) or, in the absence of these preparations, spray liberally with warm water. Visible drops of mustard gas should be wiped off carefully with absorbent cotton wool, which must be burnt.

(4) Bronchitis and pneumonia should be treated as described under 'Asphyxiants'.

(5) Secondary sores should be treated with sulphur ointments (ichthyol, etc.), gentian violet, and radiotherapy.

(6) In gastro-enteritis, activated charcoal and sodium bicarbonate should be given internally.

According to Metelmann (1938), chamomile is not in any way superior to chloramine in the treatment of skin lesions caused by mustard gas.

Müller (1938) states that the enzymes and vitamins (A and B_2) of honey reduce the tendency to blistering by war-gas, and also the possibility of secondary infections. The enzymes and vitamins present in honey cleanse and protect the wounds. From experiments conducted upon horses it appears that (a) 2 per cent. mercurochrome in water, (b) lotio Leonardii (probably a tar preparation), and (c) 1 per cent. methylene blue in glycerine are practically of no value in the treatment of mustard gas wounds. The mercurochrome solution, however, reduced pain and, to a certain extent, prevented secondary infection. (Editorial, 1940).

(5) *Sternutators (Sneezing gases).*

These are chiefly arsenical compounds. They are, like the other war gases, filled into shells and the explosion renders the particles of the compound so small that they pass through gas masks not fitted with dust filters

(layers of cellulose). These 'gases' belong to the German 'Blue cross Group'.

The chief members of this group are:—

(a) Diphenylchloroarsine [$(C_6H_5)_2$ AsCl]. It is known as 'Clark I' and 'D A' and is a crystalline substance.

(b) Diphenylcyanoarsine [$(C_6H_5)_2$ AsCN]. It is a solid and is known as 'Clark II' or 'DC'.

(c) Diphenylaminochloroarsine [$(C_6H_5)_2$ N.AsCl]. It is known as 'DM' and 'Adamsite' and is a yellow crystalline substance.

(d) Ethyldichloroarsine ($C_2H_5AsCl_2$). It is a liquid known as 'Dick.'

Symptoms of poisoning.

The above poisons were used more to put man and horse out of action than to poison them fatally. They produce vomiting and pronounced irritation of the eyes, nose, and also of the air-passages if they are highly concentrated in the air. Consequently men were forced to remove their gas masks and were then exposed to the effects of more poisonous gases. The present gas-masks prevent the passing of these irritants through the filters. The irritation is maximum in from 15 to 20 minutes after exposure and then usually slowly subsides. The effects are rarely lasting and usually pass off within 24 hours. Animals are less susceptible to these poisons than man, nevertheless the effects, even in low concentrations, on animals can be very serious and may appear after a few minutes, (Richters, 1936). There is profuse lachrymation (conjunctivitis), salivation, spasmodic cough, distress, watery nasal discharge, laboured respiration, followed by itching of the skin, eruptions especially on the lips and nose (of the horse), frequent neighing, oedema of the lungs, and pneumonia in some cases. In bad cases there may be rapid emaciation, prostration, profuse haemorrhagic diarrhoea, heart-weakness, and death in a few weeks due to arsenical poisoning. These latter symptoms, or acute deaths, may also follow the ingestion of food and water contaminated with these poisons.

Post-mortem Appearances.

If death has been caused by the above arsenical compounds, autopsy may reveal congestion and oedema of the lungs and/or pneumonia and hæmorrhagic gastro-enteritis.

Treatment.

(1) Gas-masks should be fitted on and the animals immediately removed from the poisoned area. The lungs may be affected, hence care should be exercised as explained under treatment for 'Asphyxiants.'

(2) Mouth and nose and also the skin should be irrigated with a 2 per cent. aqueous solution of sodium bicarbonate, chloramine T., (2·0 per cent.

solution for wounds and 0·5 per cent. as mouth wash and for eyes), 1 : 4000 potassium permanganate, or weak solutions of chlorinated lime. Gebele (1933) states that inhalations of chlorinated lime gas give immediate relief.

(3) Pneumonia, heart-weakness, and affections of the eyes should be treated as prescribed under 'Asphyxiants.'

(4) In cases where animals are suffering from arsenical poisoning, sodium thiosulphate should be administered *per os* and intravenously [horse, 20 – 30 g. *per os* and at the same time 5 – 10 g. (in 10 – 20 per cent. sterile aqueous solution) intravenously. These quantities can be repeated once daily if necessary].

(5) Contaminated harness, blankets, etc., must be removed from the horses immediately and washed in alkaline solutions (5 per cent. sodium bicarbonate) or in a solution of chlorinated lime.

(6) *Labyrinthic Poisons.*

In addition to the above groups of war poisons Herlant (1939) mentions 'labyrinthic poisons' which disturb mobility and cause falling down. He quotes dichloromethyl ether ($Cl\ H_2C-O-CH_2Cl$) as an example.

(7) *Food Value of Animals Poisoned by War Gases.*

Gorniewicz (1938) states that meat kept in places contaminated with 'lewisite' absorbs the poison and must be condemned. In such meat he found from 0·5 to 24 mg. of arsenic per 100 g. In animals poisoned with 'lewisite' he found the following quantities of arsenic per 100 g. of tissue: 3·12 mg. in the liver, 1·56 mg. in the kidneys, and 1·9 mg. in the lungs.

According to Szablowski (1939), the flesh of animals suffering from mustard gas poisoning could be consumed if they are killed within 24 hours of contamination. The lungs should be discarded.

In cases where animals poisoned with mustard gas are slaughtered for human consumption the skins should be removed immediately. This should be done very carefully in order to avoid the skin coming into contact with the flesh of the carcass. The skins may be used only after thoroughly soaking them in solutions of chlorinated lime and then drying them in the sun. Individuals handling materials contaminated with mustard gas should wear gas-masks and protective clothing.

It is advisable that the meat of poisoned animals should be inspected by a qualified veterinarian. Special care should be exercised in the inspection of meat of animals poisoned with arsenic-containing war gases.

The following references may be found useful to those interested in gas warfare:—

Maass (1934 and 1935), Richters (1935 and 1939), Keilholz (1937a and 1937b), Andreoni (1939a, 1939b, 1939c), Marshall (1936), Dildine (1939), Clayton (1937), Henry (1935), Lustig (1935), Wester (1937), Kämpf (1936a and 1936b), Muntsch (1937), Leipert (1938), Herlant (1939), Flury (1938),

Lang (1938a and 1938b), Flury and Zernik (1931), Aizzi (1938), Hansel (1935), Schmidt und Podloucky (1937), Gebele (1933), Cordier (1938), Maier (1938), Szablowski (1939a and 1939b)), Dowgiatto (1939), Parree (1939), Crowden (1938), Budelman (1938), Bickerton (1940), Kling (1937), Deutsch und Weiss (1934), Editorial (1934), Beulich (1934), Leschke (1932), Metelmann (1938), Hecksteden (1937), Müller (1938), Hasskó und Fülöp (1938), Drews (1939), Editorial (1940).

II. — ANTI-GAS ORGANISATION.

A. Decontamination.

It is highly probable that mustard gas and arsenical compounds will be used extensively in the present war and it is advisable to adopt the following procedure in contaminated areas after a 'gas' attack.

(1) Destroy (burn) foodstuffs intended for stock. Foodstuffs should be stored in gas-proof rooms (which should be on elevated areas) or should be covered with cellophane, tarpaulins (heavily tarred), or tarred linen. Phosgene and diphosgene are easily absorbed by wet foods (liquids, meat) and are immediately decomposed into hydrochloric acid and carbon dioxide. Such meat can be rendered edible by washing it in dilute sodium bicarbonate, which may also remove some of the arsenical poisons if such are present (Plücker, 1934).

Plücker states that chlorpicrin does not affect the baking quality of grain or flour, although the germinating capacity of grain is lowered.

As far as mustard gas is concerned it is useless to cover foodstuffs with paper or cardboard (cardboard discs on milk bottles) as such coverings are easily penetrated by this poison.

(2) Contaminated areas should be marked off with flags. Richters (1933a) states that it is frequently difficult to know which areas (pastures) are contaminated with war gases as even after fairly heavy contamination plants show the effects (yellowish-grey discolouration and drying of the leaves) only after 2 to 3 days. Chlorinated lime should be scattered over small areas (foot paths, roads, eac.) beginning on the edges. The chemical reaction between chlorinated lime and mustard gas is violent and produces very high temperatures. It is therefore advisable to mix the chlorinated lime with soil before scattering it. If no chlorinated lime is available the affected areas may be ploughed up and watered repeatedly. Shell-holes should be treated with chlorinated lime and filled up with soil. Dry grass pastures should be burnt. As a rule the detoxication of pastures poisoned with mustard gas must be left to wind and rain.

Leeuwenkuyl (1936) states that mustard gas can remain dangerous for months in woods and bushes. In moist weather, mustard gas is broken up in from one to two days, while in cool, dry, and windless weather it takes much longer, (Richters, 1933).

(3) In poisoned areas stagnant water should not be used for drinking purposes. Water contaminated with mustard gas may be drunk after boiling

it for 30 to 60 minutes, but arsenic-contaminated water remains unaffected by boiling.

(4) It is best to break up and burn contaminated wooden floors. Other floors should be covered with chlorinated lime (bleaching powder) (*ca.* 0·5 kg. per square metre). Cotton wool and rags used for wiping off contaminated articles should also be burned or buried very deep.

(5) Clothing, blankets, rugs, and harness should be washed repeatedly in an aqueous solution of chlorinated lime (cheap and effective) or, failing this, in a 5 per cent. aqueous solution of sodium bicarbonate or a solution of potassium permanganate. After treatment, leather articles should be oiled or smeared with fats.

(6) Detoxication of stables: After a gas attack all animals, harness, utensils, etc., should be removed from the stables and treated as prescribed above. Animals should be moved to elevated areas. They should not be returned to the stables until at least three hours after thorough detoxication of the latter. In the case of poisons belonging to the 'green cross group,' ventilation by means of fans is sufficient; regarding the 'blue cross group,' ventilation and spreading of chlorinated lime should be combined, and in the case of the 'yellow cross group' (mustard gas), the stables should be well ventilated and the roofs, walls, doors, and windows sprayed with a solution of chlorinated lime or sodium bicarbonate. Chlorinated lime should be scattered on the floor.

(7) It is esential that those individuals concerned in the detoxication of poisoned areas should wear gas-masks and protective clothing. (See II B, 'Prevention of poisoning with war gases.')

B. — Prevention of Poisoning by War Gases.

It is essential that an effective anti-gas organisation should be available. This should be constituted and organised as follows:

(1) Specially trained medical doctors, veterinarians, chemists and first-aid assistants, who should perform their duties in collaboration with the local authorities in cities and towns. The chemist's duty is to identify the gas or gases as soon as possible and to prescribe methods of detoxicating them. Information concerning the identification of poison gases is to be found in the following publications:— Chambon (1939), Crowden (1939), Editorial (1937), Gigon and Noverraz (1939), Lichtenberg (1937), May (1936), Mohler und Polya (1936), Polya (1937), Shimizu (1939), Sorley (1939), van Eck en van Marle (1940), (Weber (1937), Wester (1937, 1937a and 1937b), Edwards (1940).

According to Richters (1937), the dog may possibly be used as a gas detector. This method may have disastrous results, because the effects of some war poisons (*e.g.* mustard gas) may be delayed for a day or longer and consequently dangerous areas may be considered safe.

(2) Provision should be made for rapid and efficient detoxication of animals, contaminated rooms, stables, etc. Large quantities of the following should be available: water, warm water, soap, chlorinated lime, sodium bicarbonate, sodium carbonate, potassium permanganate, fat and oil for treating leather articles, vaseline, raw linseed oil, and drugs required for treatment [tannic acid, heart and respiratory stimulants (caffeine, lobeline, atropine, digitalis, cardiazol, coramine, etc.), sodium thiosulphate, cough mixtures (codeine phosphate, heroin, menthol, oil of turpentine, oil of peppermint, benzoin, Peru and Tolu balsam), local anæsthetics (cocaine, novocaine)]; and buckets, watering-cans, spades, carts, and wagons for scattering chlorinated lime; cotton wool, rags, gas-masks, and protective clothing.

There should be mobile first-aid parties, fire-extinguishing squads, dressing-stations, ambulance services, and pamphlets containing details concerning the duties to be performed.

Individuals engaged upon detoxication work should also detoxicate the protective clothing and the utensils used in the course of their duties. When taking off contaminated clothing care should be exercised that the external surface of the clothing does not come into contact with the body.

(3) Protective clothing:— Protective clothing should be (a) light, (b) cheap, (c) easily handled, stored, and transported, (d) durable and resistant to climatic conditions and (e) able to withstand at least 5 disinfections (Gersons and Keilholz, 1938). Protective clothing is usually made of oil-cloth or oil-silk. Crowden (1938) states that oil-skin, oil-cloth, and oil-silk cause an increase in the body heat and consequently exhaustion during heavy work, and that if moistened cloth is worn over these types of protective clothing evaporation of the moisture cools the body.

In the Great War the Americans attempted to protect the hoofs of the horses by fitting between the hoof and shoe a plate of sheet iron embedded in composition rubber which covered the sole and frog. The legs from the knee or hock downwards, including the hoofs, were enveloped in satin boots. The wear and tear on these satin boots was heavy and their adjustment and maintenance by no means easy. It appears that at present no acceptable protective covering, which could be applied to animals on a large scale, is known.

(4) Gas-masks: Gas-masks for horses should (a) be durable (strong), (b) be cheap, (c) be light, (d) be easy to fit on, remove and pack, (e) not interfere with the normal play of the reins, (f) protect against all war gases, (g) be easily recharged, (h) permit use at all paces.

It is obviously a very difficult task to construct a gas mask which would satisfy all these requirements.

The filters in the human gas-mask act in the following ways:—

(a) mechanical action of the layers of cellulose which prevent the

passing through of dusts (arsenical dusts) and smokes;

(b) physical absorption of the poisons on large surfaces as constituted by the layer of activated wood charcoal;

(c) chemical absorption (silicate);

(d) changing the poisons into non-toxic substances — by impregnating the activated charcoal with solutions of sodium carbonate, zinc sulphate, and caustic soda. Caustic soda renders hydrocyanic acid harmless (NaCN). The gas-masks in use at present for man afford protection against all poisonous war gases except carbon monoxide. Gersons and Keilholz (1938) state that mask G renders approximately 48 g. of chlorpicrin and 24 g. of phosgene harmless. In the case of phosgene this means that with a concentration of 1 g. per cubic metre of air this mask would afford protection for approximately 100 hours, calculated on a basis of the human being inhaling 10 litres of air per minute. Details concerning the cleaning, refilling, and storing of masks are described by Gersons and Keilholz (1938). When the concentration of the poison gases in the air is very high, it is essential that an ogygen apparatus be available. This is now fitted to gas-masks. The life of the filters depends upon the concentration of the poison gases in the air.

The horse has an enormous respiratory capacity which according to Richters (1936) is as follows (per minute): at rest — 40 to 60 litres, at the trot — 90 to 150 litres, at a fast trot — 220 to 250 litres, at the canter — 300 to 400 litres, and at the gallop — 500 litres.

In the Great War, animal respirators of the nose-bag type were used where both inspiration and expiration were effected through layers of cloth impregnated with detoxicating chemicals and moisture.

These respirators proved very unsatisfactory. The Russian type of mask covered the horse's head and had mica eye-pieces. The greatest drawbacks of respirators for horses are that they interfere with respiration and the play of the reins, and do not protect against all war gases. Another serious disadvantage is the difficulty of knowing when the masks lose efficacy. They dry very rapidly in summer and freeze in winter. Henry (1935) reports that the French are using the 'Model Decaux' mask and they state that horses can work comfortably at any pace. The author was unable to find details of this mask in the literature consulted and an attempt is being made to obtain details from the War Office, London.

It is therefore clear that it is by no means an easy matter to evolve a satisfactory mask for the horse and mule. The donkey and ox would probably be sufficiently protected by the humid filter respirator used for the horse in the Great War. The pronounced difference in the size and shape of the heads of the different breeds of dogs adds to the existing difficulties regarding gas-masks for animals. Carrier pigeons could be kept and transported in portable gas-proof cases made to carry four birds each.

The following references are given for the benfit of those interested in anti-gas organization:

Clayton (1937); Richters (1933, 1933a, 1936, 1937, 1939); Henry (1935); Leeuwenkuyl (1936); Plücker (1934); van Giffen and van Bronkhorst (1937); Keilholz (1937a); Gersons and Keilholz (1938); Steiner (1939); Editorial (1936); Bangert (1938); Jousma (1940); Hyde and Falkiner (1937); Wester (1937, 1937a and 1937b); Wooldridge (1940).

III. — SUMMARY.

A detailed review is given of chemical substances, which have been and are still being used as smoke screens (artificial fogs) and 'poison gases' in warfare. The chemical nature, symptomatology, post-mortem appearances, treatment, and prevention of poisoning with the different groups of war gases are discussed. The food value of animals poisoned by war gases, and anti-gas organisations have also been considered.

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The Susceptibility of the Ferret to Heartwater.

By J. H. MASON and R. A. ALEXANDER, Onderstepoort.

In a previous article we (Mason and Alexander, 1938) mentioned that all our attempts to infect small laboratory animals with heartwater had failed. Investigation into this phase of the work has been going on since 1931, and we believe that we have every justification for stating that the rickettsia of heartwater cannot easily be induced to develop in guinea-pigs, rats, rabbits, or mice. During the course of the work, we have used as infective material, blood, intima scrapings of jugular veins, and brain of infected sheep, brain of infected cattle, and infected ticks (*Amblyomma hebræum*), either engorged or not engorged. Inoculations have been made intraperitoneally (guinea-pig, rabbit, rat, mouse), intracerebrally, (guinea-pig, rabbit, rat, mouse), intraocularly (rabbit), and intratesticularly (guinea-pig and rabbit). In one series of experiments, intra-uterine injection of infected material into guinea-pig embryos was tried without success. The natural resistance of guinea-pigs was attacked with deep X-ray therapy, and by feeding them on vitamin-deficient or vitamin-free diets. Blockade of the reticulo-endothelial system with India ink did not render guinea-pigs or mice susceptible. No attempt was abandoned until 3, and often 10 or more, passages had been carried out. On two occasions there was a translocation of the virus to the brain and its survival therein, once in a guinea-pig and once in a rat. In addition, all attempts to infect the developing chick embryo failed, in spite of modifying the technique in many ways and in spite of using fertile eggs from hens living on a vitamin-deficient diet.

Eventually the susceptibility of the ferret was demonstrated and it is the purpose of this paper to record in some detail the methods used, and the grounds on which the claim is based.

At the outset we must point out that the work has been hampered to a considerable extent by a shortage of ferrets. Before 1937, there were no ferrets in South Africa. In September of that year, Dr. Gilles de Kock kindly consented to bring with him from England 5 females and 1 male. During the voyage, one bitch gave birth to a litter of pups, and our present stock of about 80 ferrets is the progeny of that importation.

The Susceptibility of the Ferret.

We have made three attempts to infect ferrets, series 1 in 1938, series 2 in 1939, and series 3, at present in progress, in 1940. The continuity of the first two series was governed to a large extent by the supply of ferrets.

Series 1: The general procedure was to give the ferrets an intraperitoneal injection of heartwater-infected material, and to keep them under observation until they showed a temperature or other clinical reaction. If a reaction occurred, the brain and/or spleen was subinoculated into other ferrets and the infectiousness of the passage material controlled by injecting it into susceptible sheep.

The inoculum for the first two ferrets was a saline emulsion of intima scrapings of the jugular veins of a heartwater-infected sheep (51263). A total of 17 ferrets were used and 10 ferret-to-ferret passages were carried out. A number of the ferrets showed mild febrile reactions after an incubation period of from 6 to 10 days, but none were clinically ill. Definite fever reactions were produced in all the sheep that received ferret brain or spleen intravenously. An example is given in chart I. (sheep 52649 that received brain material from a ferret of generation 2).

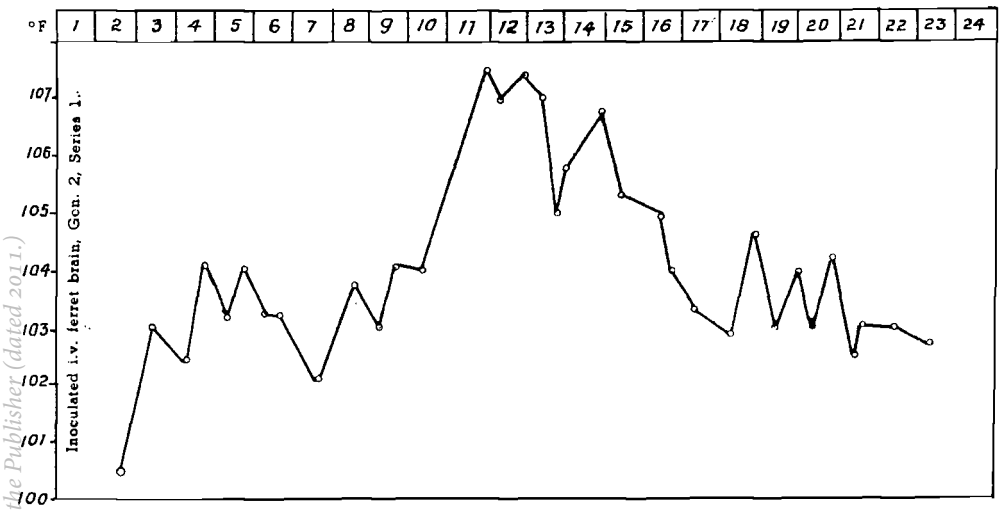


CHART 1.

Temperature chart of sheep 52649.

With one exception (sheep 51855, see below), all the sheep recovered, and on subsequent immunity test were susceptible to heartwater. The exception (sheep 51855) received an injection of spleen emulsion from the first generation ferrets; a typical heartwater reaction followed and, after death, rickettsias were demonstrated in jugular intima smears. At the height of the heartwater reaction 10 cc. of blood was subinoculated into heartwater-immune sheep (41835, 51632, 52126) and into a normal sheep (51938). The heartwater-immune sheep did not react, the susceptible sheep reacted

and died. After death, rickettsias were found in smears prepared from the jugular intima. A graphic representation of the history of sheep 51855 is given in Chart 2.

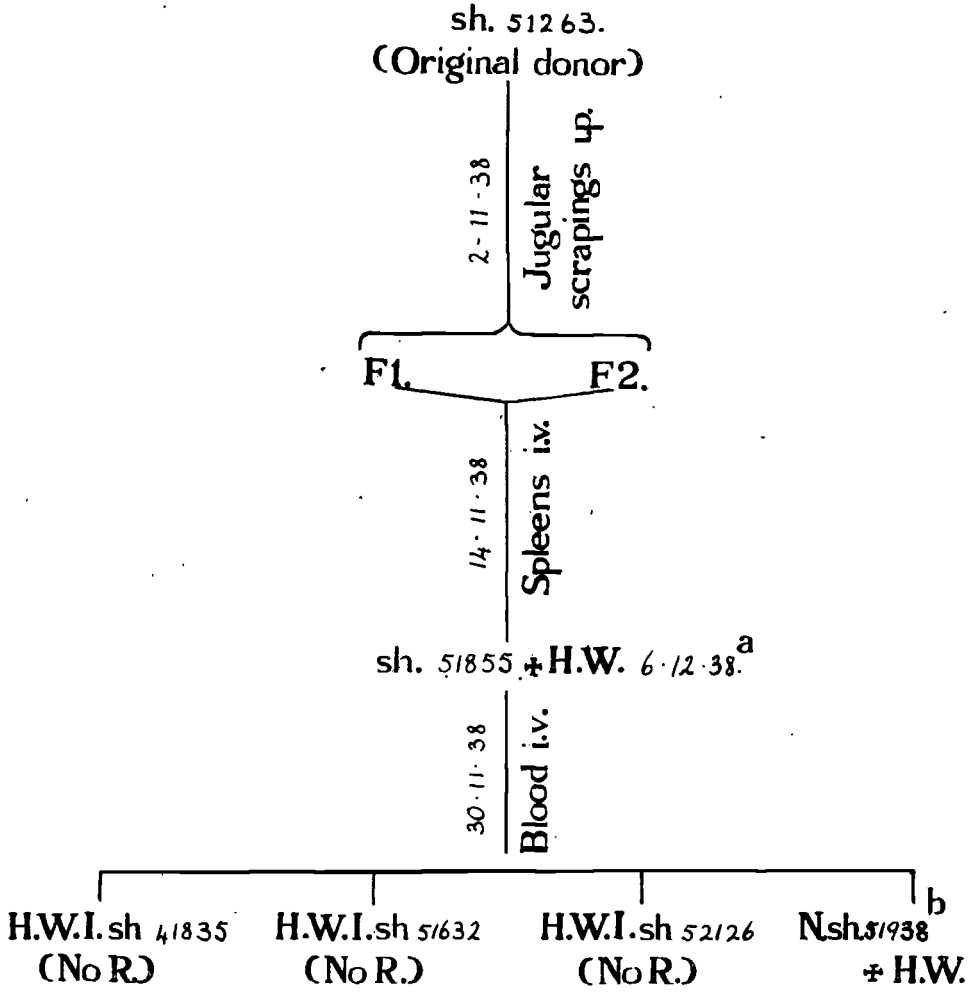


CHART 2.

INFECTION OF SHEEP 51855 WITH HEARTWATER FROM FERRETS.

(i.p. = intraperitoneally; i.v. = intravenously; + = died; H.W. = heartwater; H.W.I. = heartwater-immune; N = normal; sh. = sheep; No R. = no reaction; F = ferret; a = rickettsias demonstrated in brain; b = rickettsias demonstrated in jugular intima smears).

Conclusions: An undetermined, almost inapparent infection of unknown origin was carried from ferret to ferret for 10 passages as proved by the temperature reactions produced in the sheep. This infection was also passed to guinea-pigs and from them back to sheep, but it was obvious that it

was not heartwater, because none of the sheep developed immunity to this disease. The heartwater infection transmitted to the one sheep (51855) must therefore be considered to have resulted from survival of the original inoculum in the ferrets.

At this stage, the work was discontinued in order to build up our supply of ferrets.

Series 2: We used as primary inoculum a saline emulsion of the brain of an infected sheep. Otherwise the general procedure was similar to that followed in series 1. Except that in the first generation ferrets there was no indication of even survival of the heartwater virus, the results obtained were similar to those of series 1. Temperature charts of the control sheep could be superimposed upon that recorded in Chart 1. No other clinical deviation from normal health was noticed. All the sheep recovered and none developed immunity to heartwater.

The infection was carried through 6 generations of ferrets only. On the 7th passage it died out and was not regained by further ferret-to-ferret subinoculations. The infection was carried from a ferret to guinea-pigs for one passage only; mice and rabbits appeared to be insusceptible.

Comment: Since all blood, brain, and spleen material of the passage ferrets was bacteriologically sterile, we assumed that the cause of the temperature reactions in the sheep was a virus. The most reasonable assumption appeared to be that our stock of ferrets carried this virus and that they were suffering from an inapparent infection. In an attempt to clear up this point two ferrets from stock were killed and an emulsion of the pooled brains was injected into two sheep. No reaction was produced. It is admitted that this single test was inadequate because, to reproduce similar conditions, it would have been necessary to have injected blood or brain from a healthy sheep into a ferret, and then to have carried out several brain-to-peritoneum passages in ferrets before returning to sheep. Unfortunately the number of ferrets available was too small to allow of this.

From this series of experiments we again concluded that the ferret was insusceptible to heartwater.

Series 3: In this series of experiments, at present in progress, we have definitely succeeded in transmitting heartwater to ferrets and in maintaining it in them for a period of approximately 3 months. Unfortunately we have again encountered an additional infection similar to, or identical with, that described in series 1 and 2. Although this complicates the work, and makes the interpretation of results somewhat difficult, it in no way invalidates our final conclusions.

It has been shown, (Alexander, 1931), that the virus of heartwater is concentrated in or on the leucocytes and erythrocytes and is absent from the blood serum of reacting sheep. Therefore we decided to use as primary inoculum a saline suspension of the buffy coat obtained by spinning infected

sheep blood at 1500 r.p.m. for 5 minutes. This material was injected intraperitoneally into two ferrets.

On the 13th day a slight febrile reaction that lasted for four days occurred. Clinically the ferrets appeared well, but they were killed by ether anæsthesia and an emulsion of the pooled brains injected intraperitoneally into two ferrets and intravenously into one sheep. Both ferrets and the sheep subsequently showed febrile reactions. Since then, 7 further ferret passages have been carried out and the details are given in chart 4 (discussed below). Probably as the result of adaptation of the virus to the ferret, the period of incubation has shortened and with passage much more definite temperature reactions have been obtained. This is illustrated in Chart 3, where the temperature charts of one ferret of each of generations 1, 4, and 7 are depicted.

It will be noticed that ferret 1 (gen. 1) showed a very slight thermal rise commencing on day 13; ferret 8 (gen. 4) a much more marked reaction beginning on day 10; ferret 13 (gen. 7) an equally good reaction commencing on day 7. It would appear that the saddleback type of reaction is characteristic because, with the exception of the 2 ferrets of generation 1, 10 of the remaining 12 have reacted in this manner. Only one ferret was clinically ill, viz. ferret 8, generation 4. It was dull, and showed nervous symptoms and incoordination of movement. The only constant post-mortem change has been an enlargement and darkening of the spleen. Rickettsias, indistinguishable from *Rickettsia ruminantium*, have been demonstrated in sections in endothelial cells of the kidney of ferret 8 and in smears of intima scrapings of the vena cava of ferret 9. Although a careful search of sections or smears of the brain, kidney, spleen, liver, lung, pleura, and peritoneum of the other ferrets was made, rickettsias were not found.

In Chart 4 the main data proving that heartwater has been transmitted to, and maintained in, ferrets is given. At the present time we have reached the 8th ferret-to-ferret passage, but as the results of the subinoculations into sheep of generations 6, 7 and 8 are not yet available, we have terminated the chart at generation 5.

A consideration of the chart shows that every heartwater-susceptible sheep that received ferret brain reacted. Sheep 55182 (gen. 1) and 57020 (gen. 2) recovered and subsequently were shown to be immune to heartwater; sheep 57618 (gen. 2), 56413 (gen. 3) were killed while showing the nervous symptoms associated with heartwater, and rickettsias were demonstrated in large numbers in jugular intima smears; sheep 56433 (gen. 3), 57181 and 57219 (gen. 4) and 55644 (gen. 5) died after showing symptoms typical of heartwater, and, except in the case of 57219, rickettsias were demonstrated in sections of the brain or in intima smears. Subinoculation of blood from sheep 57020 (gen. 2) produced typical heartwater in two sheep (56423 and 56391). At post-mortem examination, all sheep that were killed or died showed lesions typical of heartwater. No heartwater-immune

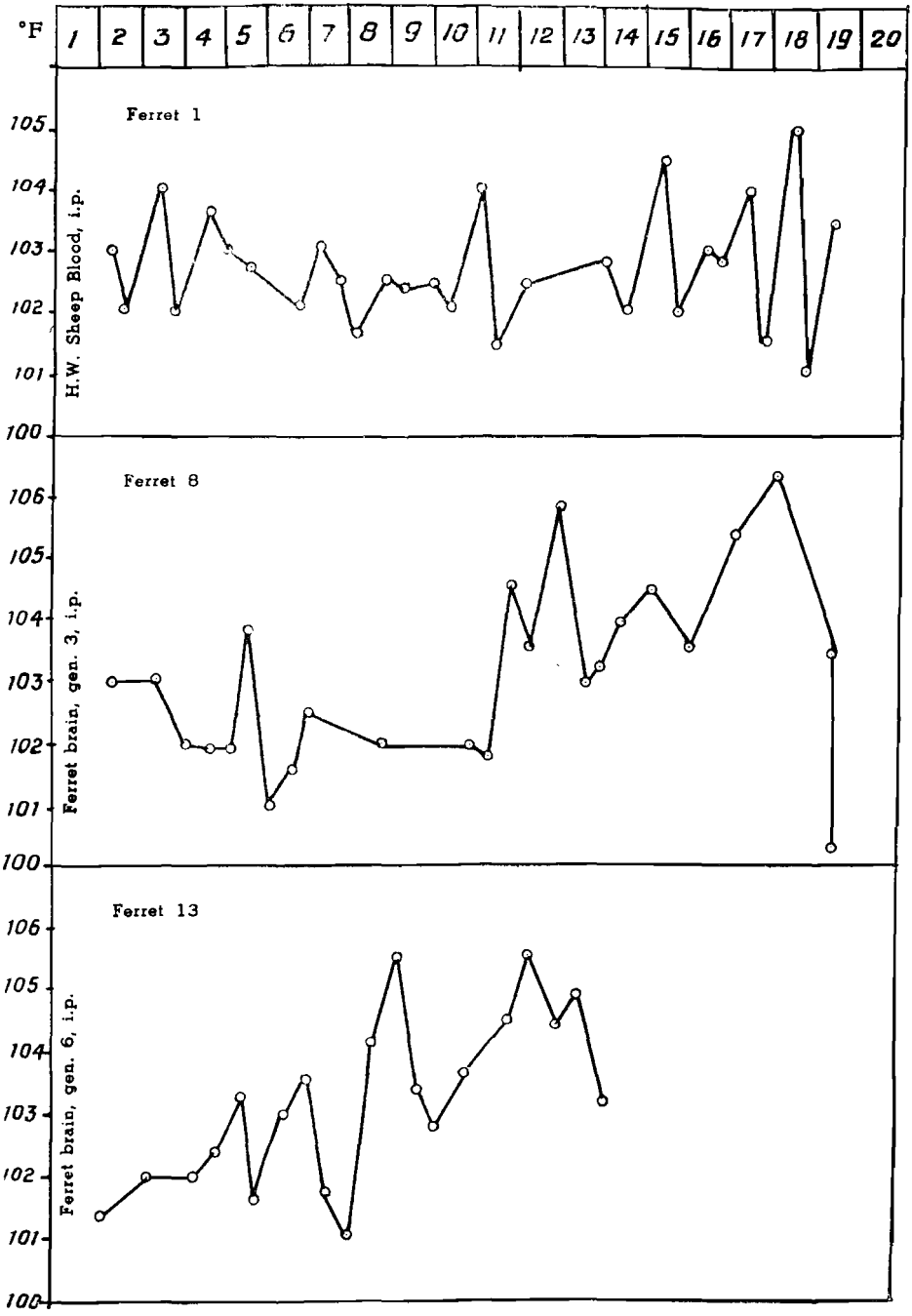


CHART 3.

Temperature Reactions in Ferrets.

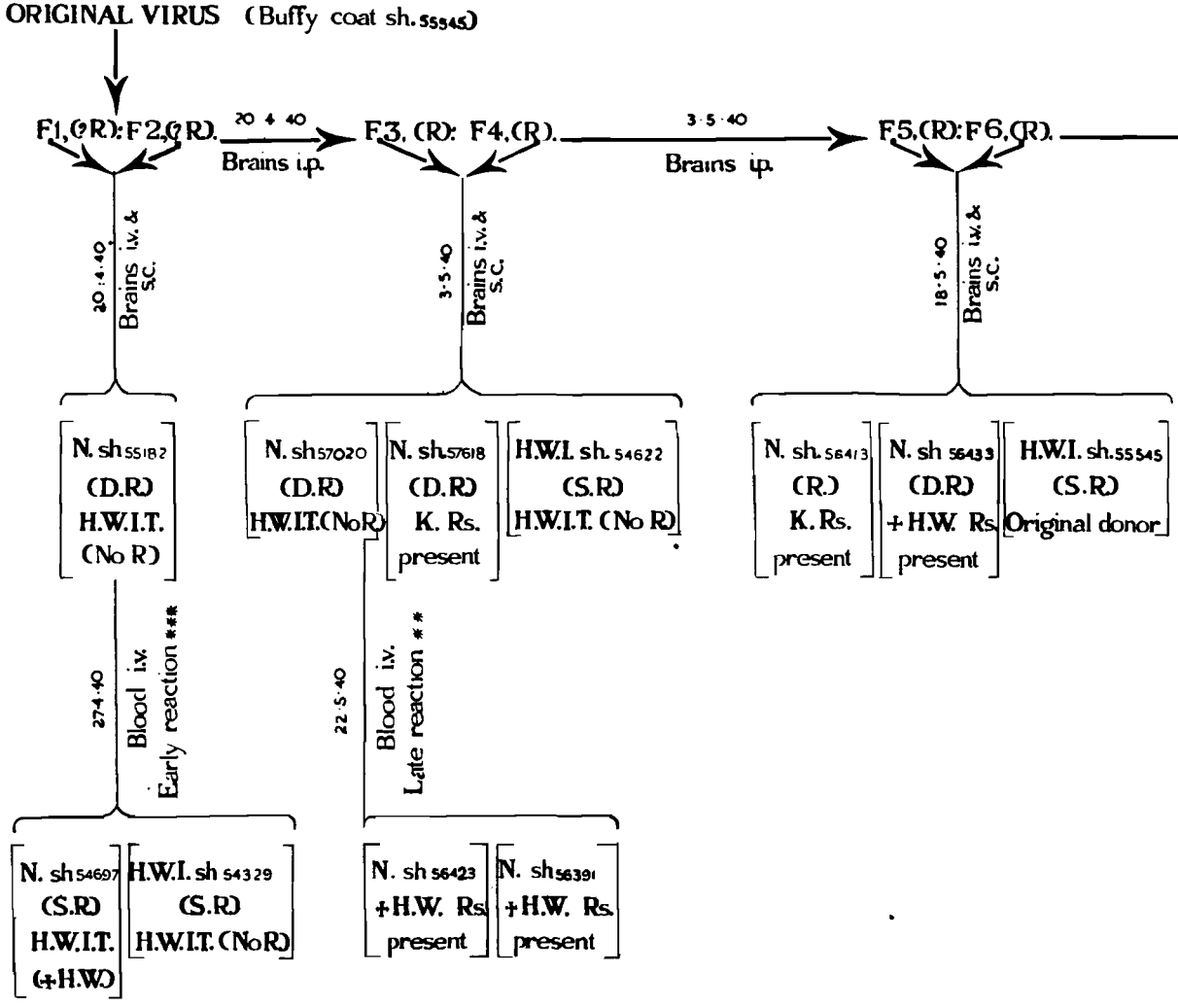
sheep (54622 gen. 2, 55545 gen. 3, 54280 gen. 4, 55887 gen. 5) became clinically sick or died as a result of the injection of ferret brain, but each showed a temperature reaction. One sheep, 54622 (gens. 2 and 4), that had recovered from both heartwater and the reaction produced by ferret brain showed no reaction when tested again with ferret brain.

We stated earlier that we had again encountered an infection in ferrets similar to that of series 1 and 2. This could be demonstrated only by the reactions produced in sheep. All the heartwater-immune sheep that received ferret brain responded with a febrile reaction that began on or about the 6th day and lasted for from four to seven days. A typical example is given in Chart 5 (55887, a heartwater-immune sheep that received ferret brain, gen. 5). The normal sheep on the other hand showed two separate and distinct reactions (Chart 5, sheep 55644), although on some occasions the two reactions were merged into one prolonged elevation (Chart 5, sheep 57181). The second rise commenced between the 10th and the 16th day after injection or from 2 to 4 days after the primary rise. No illness was noticed in sheep throughout the course of the first temperature reaction. The symptoms of heartwater always occurred towards the end of the second reaction.

The relationship of the virus causing the first or early reaction to the rickettsia of heartwater is not clear. There is evidence that it is quite unrelated. Blood from sheep 55182 (gen. 1) taken at the height of the early reaction was injected intravenously into a normal (54697) and into a heartwater-immune (54329) sheep. Both reacted, but showed only a single rise; sheep 54697 subsequently proved susceptible to heartwater. On the other hand, blood of sheep 57020 (gen. 2) taken during the second reaction produced typical heartwater when inoculated into two normal sheep (56423 and 56391). Finally the reactions produced in sheep by the brain of ferret 7 (gen. 4) are of importance; two heartwater-susceptible sheep (57181 and 57219) showed, respectively, a prolonged and a double reaction, and both died of heartwater; a heartwater-immune sheep (54280) showed only a single early reaction and survived; sheep 54622, immune to both heartwater and the 'ferret virus,' did not react in any way.

Fortunately we were able to show that the 'ferret virus' did not originate in the donor sheep that provided the infective inoculum for the first two ferrets. This sheep (55545) survived the heartwater reaction and, tested later with brain material from passage ferrets 5 and 6 (gen. 3), responded with a single early reaction.

This double infection has complicated this investigation to some extent. At one time we suspected that a coccus isolated in pure culture from the brain of the ferrets of generation 2 might have been the cause. However, a heavy suspension of this coccus produced no reaction when inoculated intravenously into a sheep and the sheep was subsequently shown to be susceptible to heartwater. Moreover, all ferret passage material after



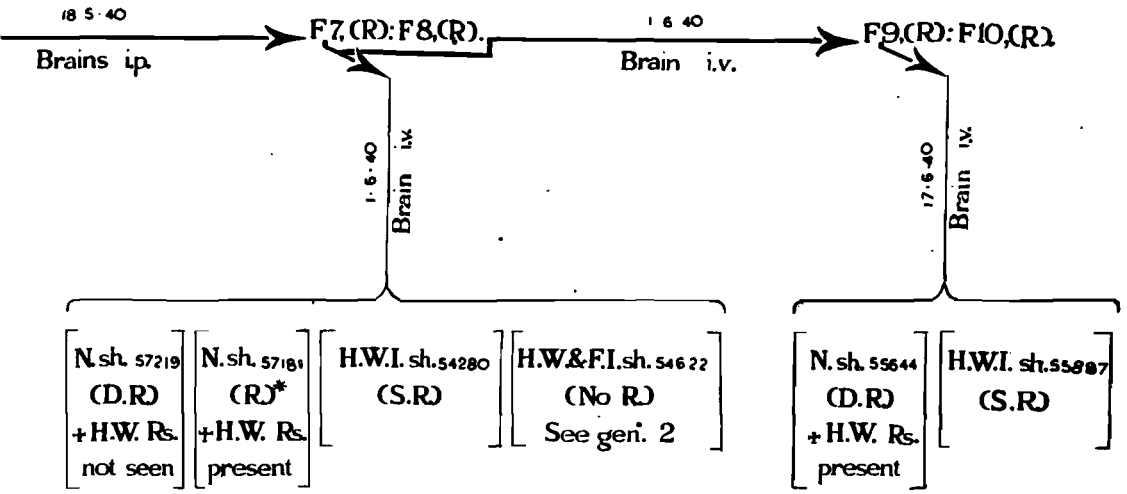


CHART 4.

MAINTENANCE OF HEARTWATER IN FERRETS.

(i.p. = intraperitoneally; i.v. = intravenously; s.c. = subcutaneously; N. sh. = normal sheep; H.W.I.sh. = heartwater-immune sheep; H.W. & F.I. sh. = heartwater-and-'ferret-virus'-immune sheep; H.W.I.T. = heartwater immunity test; (D.R.) = double temperature reaction; (S.R.) = single temperature reaction; (R.)* = single, but prolonged temperature reaction; (No R.) = no reaction; + = died; Rs. = rickettsias; K. = killed; **, ***, see text).

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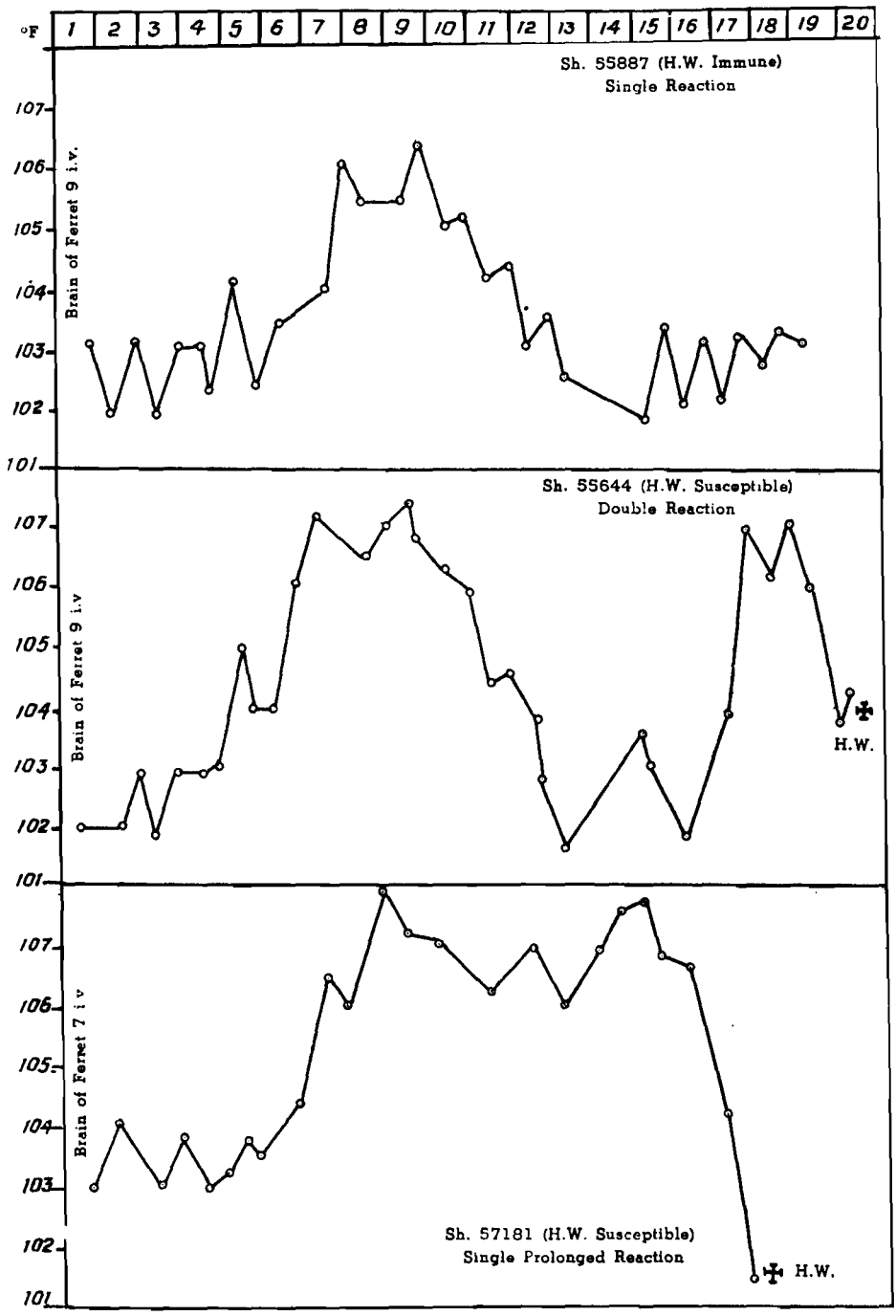

CHART 5.

Temperature Reactions in Sheep inoculated with Ferret Brain.

generation 2 has been bacteriologically sterile. At the present moment we are exploring methods of separating the two infections. Brain material from ferrets 9 and 10 of generation 5 produced a febrile reaction in guinea-pigs, and it has been possible to maintain this infection in guinea-pigs by brain-peritoneum passage. This guinea-pig brain material has produced a febrile disease in sheep, but at present we do not know if the disease transmitted is 'ferret disease,' heartwater, or both. In addition we are attempting to propagate the viruses in the developing chick embryo, but the results are not encouraging.

Discussion.

On each of 3 occasions when an attempt was made to infect ferrets with *R. ruminantium*, a virus infection was picked up and maintained in ferrets by brain-peritoneum passage. This virus caused a febrile reaction in sheep, but never death; the disease bore no resemblance to, and did not immunize against, heartwater. The origin of this infection or its association with the strain of heartwater that is maintained by serial passage in sheep at Onderstepoort is not known. In spite of the lack of direct experimental evidence on the point, we believe that the virus exists as an inapparent infection in our stock of ferrets.

On the third attempt, *R. ruminantium* was propagated and maintained in ferrets by serial passage. It appears somewhat remarkable that a rickettsia, previously supposed to be pathogenic only for ruminants, should now be found capable of multiplying in a carnivore, the ferret. The finding opens up a wide avenue for research that is now being explored. Until either the virus disease or the heartwater rickettsia is propagated separately, we cannot be certain which is responsible for the temperature reaction in the ferret.

Conclusions.

1. On three successive occasions a virus, that produces a mild febrile disease in sheep, has been picked up in ferrets.

2. This virus produces no immunity against heartwater.

3. The origin of this virus is obscure, but it is believed to exist in our ferrets as an inapparent infection.

4. In addition, *R. ruminantium* has been shown capable of multiplying in ferrets.

5. After five passages in ferrets there was no modification in virulence of the ferret passage rickettsia for sheep.

6. From the ferrets an infectious agent has been passed to guinea-pigs, but at present it is not known if this is heartwater or the ferret virus.

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ADDENDUM.

Since going to press, we have evidence that the heartwater infection has died out in ferrets (gen. 5).

The ferrets of succeeding generations show no temperature reactions, but the inoculation of their brains into sheep produces a thermal rise. Thus it would appear that the heartwater infection was the cause of the reaction in the ferret.

We have also shown that it was the ferret virus and not heartwater that was transmitted from ferrets to guinea-pigs.

The Avian Tuberculin Test.

By A. S. CANHAM, Allerton Laboratory, Pietermaritzburg,
and L. C. BLOMFIELD, Greytown.

Tuberculin testing of poultry does not fall to the lot of many veterinarians in South Africa, and for this reason we thought that a report on such a test carried out recently by us might be of interest.

Tuberculosis had several times been diagnosed in live and dead fowls sent by a certain farmer to Allerton Laboratory for examination. Just before we did the test this farmer had consigned about 20 birds to the local market-master and, on arrival, four were dead in the crates. Post-mortem examination revealed advanced generalized tuberculosis. At the request of the Medical Officer of Health, eight emaciated birds were removed and the remainder were allowed to be exposed for sale. A tuberculin test of these eight gave six positive and two negative reactions. On post-mortem examination, seven birds showed advanced lesions and one was negative. One bird that gave a negative reaction to the test was affected with advanced generalized tuberculosis.

The flock to be tested comprised 429 birds, of which 239 were Leghorns, 180 Rhode Island Reds, and 10 Australorps. These were kept on the semi-intensive system and were housed in 18 different pens.

Several interesting points were noted in connection with the actual testing. In some cases the wattles showed a marked difference in size, and before the actual injection was done a note was made of this peculiarity, and also of the wattle used for testing. Other birds showed a shrunken wattle on one side, due possibly to previous wounds. Some wattles felt more fleshy than others; this should be specially noted in connection with the interpretation of test results involving only very slight oedema. The wattles of the Leghorns were in most cases larger than those of the Rhode Island Reds. It was much easier to carry out the intradermal test on the wattles of the cockerels than of the hens. The site used for injection was about 3 or 4 mm. above the lower edge of the wattle, and the dose of tuberculin was about 0·1 cc. Results were read after 48 hours.

The result of the test was as follows —

Leghorns — Negative 201, positive 38 hens.
Rhodes — Negative 120, positive 53 hens, 7 cockerels.
Australorps — Negative 10, positive 0.

Most of the reactions were typical. The affected wattle, particularly at the edge, showed a marked oedema. There was little or no pain evinced

on handling the swollen wattle, and heat was not observed. In some of the reactors the oedema extended across the throat to the opposite wattle, while in one case the face on the side injected was swollen, causing the eye to be partly closed. Some birds showed only a very slight oedema and, in order to appreciate this, both wattles had to be palpated carefully.

Birds with only slight oedema of the wattle were condemned, because previous tests — confirmed by post-mortem examination — showed that such reactions were usually indicative of extensive tuberculosis. This was also the experience of Feldman (1938), who found that a bird in which the lesions were minimal might give a pronounced tuberculin reaction, whereas one that was extensively affected might give a small, moderate, or even unrecognisable tuberculin reaction. The degree of oedema varied markedly in different birds and Feldman rightly states that, since the intensity of the local tissue reaction varies in different chickens, considerable experience is necessary for a proper interpretation of the reaction. Some wattles showed a small bluish discolouration at the site of injection, but this was not accompanied by oedema. It is thought that this was caused by puncturing small blood vessls while injecting the tuberculin.

No reaction was recorded as doubtful. It was interesting that none of the birds at the time of testing appeared ill, and that no condemned bird showed any systematic reaction to the test. Oedema of the injected wattle was the only result of the test. A number of the reactors were under-weight for their size, and many of them showed crooked breast bones, while the pectoral muscles were frequently partially atrophied. More reactors were found among the Rhode Island Reds, although they lived under the same conditions as the Leghorns. Fifty-one of the reactors were slaughtered and examined the day after the reading of the test and the remainder four days later. One bird died between the two killings and was not examined. On arrival at the farm, it was found that many of these birds had been killed by chopping off their heads, so that it was impossible to correlate the degree of oedema with the extent of the lesions of tuberculosis present.

The results were as follows.

TABLE 1.

Breed.	*No Lesions.*	*Localized.*	*Localized but Extensive.*	*Generalized.*
Leghorns	5	26	2	4
R.I.R. (Hens)	1	33	1	18
R.I.R. (Cockerels)	3	4	–	–
TOTAL	9	63	3	22

In considering these figures the no-lesion reactors are of interest. Earlier in this paper reference was made to the presence of only a very

slight oedema of the injected wattle of some condemned birds. These cases numbered about 12 or 13 in all, and were confined to the Leghorns. It is possible that in the five no-lesion reactors among the Leghorns our interpretation was wrong. The reactions in the positive Rhode Island Red cockerels were, however, typical, although no macroscopic lesions of tuberculosis were found.

In this connection Feldman (1938) says: 'It is my opinion that the failure to find lesions of tuberculosis in birds that have reacted in a characteristic manner to the intracutaneous injection of tuberculin does not necessarily impugn the reliability of the test as a means of separating tuberculous from non-tuberculous chickens. It must be recognised that the demonstration of lesions of tuberculosis is not always simple. Frequently, tissues devoid of lesions of macroscopic dimensions contain definite and numerous microscopic lesions.'

During the course of the examination of the carcases, a record was made of the infection in various organs.

Liver	70 birds
Spleen	42 birds
Intestine and gizzard	28 birds
Lungs	15 birds
Thyroid Gland	12 birds

The lesions in the liver varied from pinpoint-sized whitish or greyish areas, dotted over the surface and in the substance of the organ, to areas about 6 mm. in diameter. In the spleen the lesions were not so numerous, but were generally larger than those found in the liver. They were mainly confined to the surface of the organ, and were approximately 12 mm. in diameter. The spleens were much enlarged. The intestines and gizzard contained small hard irregular nodules of various sizes. On sectioning some of these, no caseous material was found. The intestines were not opened. In the lungs, the tuberculous lesions differed from those found elsewhere and took the form of large, diffuse, greyish-yellow areas deep in the substance of the organ. The thyroid gland showed lesions in varying stages.

An interesting observation was made during the course of these examinations. In some of the markedly generalized cases emaciation was absent; in fact, the pectoral muscles were quite well developed, and there was a fair amount of fat in the fat depôts. In some generalized cases, lesions were observed in the oviduct, and disintegrating egg material was present. No examination of the bone marrow was made. Eleven out of 48 birds were rejected for human consumption in the first batch, and 14 out of 46 in the second batch. The lesions in the other fowls were so slight that the carcases, excluding the viscera, were passed.

The question may be asked, 'How many positive cases were missed?' The possibility of having missed some cases of tuberculosis among these

birds is readily admitted. Van Es and Schalk (1914) claimed that about 8·4% were missed in testing, while Van Leeuwen (1915) claimed that about 7% failed to react. Laurberg (1927) in a test of 584 chickens found lesions in two birds that gave no reactions.

On the 18th December, six weeks from the time of the first test, a second intradermal test was carried out on the remaining 302 birds, and 35 birds reacted. The results were as follows:—

TABLE 2.

Breed.	*Positive.*	*Negative.*	*Total.*
Leghorns	7	179	186
Australorps	–	10	10
R.I.R.	28	78	106
TOTAL	35	267	302

These positive cases were slaughtered and the post-mortem examination showed the following findings —

TABLE 3.

Breed.	*No Lesions.*	*Localized.*	*Generalized.*
Leghorns	2	5	–
R.I.R.	2	22	4
TOTAL	4	27	4

No further tests were conducted, as the owner killed all the fowls left over.

Conclusions.

429 fowls were tuberculin-tested and 98 reacted. One reactor was not examined; of the remaining 97 reactors, 88 showed macroscopic lesions. Six weeks later 302 of the remaining, and supposedly clean, birds were retested and 35 reacted, but 4 of these showed no macroscopic lesions.

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VAN LEEUWEN, J. F. H. L. (1915). Die intrakutane Tuberkulination bei Huhnern. *Centralbl. f. Bakt.* **76**: 275-288.

LAURBERG (1927). Quoted by Feldman (1938).

Reduction of Haemorrhage in Dehorning of Cattle.

By J. M. FOURIE, Government Veterinary Officer, Hoopstad.

When adult cattle are dehorned there is always a considerable amount of hæmorrhage, which, although not considered dangerous, is objectionable.

A simple, practical, and effective method of reducing hæmorrhage to a minimum is to tie a ligature, consisting of a fairly strong piece of string, around the bases of the horn-cores in the way illustrated in the accompanying figure. The ligature should lie across the skin well away from the cornu-

..... = Outline of bony skull.
A. = Crest of frontal bone
B. = Zygomatic process of frontal bone } bounding temporal tossa D.
C. = „ arch.
—— = Arteries involved.
1 = Artery supplying horn core.
2 = Superficial temporal artery.
X = Cornual nerve.

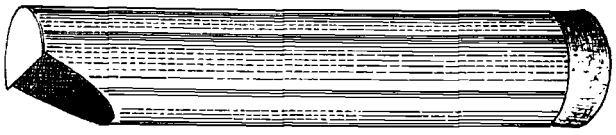
cutaneal junction. If tied as illustrated across the middle of the frontal crest it is easily tightened and will occlude the main source of hæmorrhage, viz. the large branch of the superficial temporal artery, which passes around the outer side of the base of the horn-core and supplies the corium of the horn.

The effect of the ligature will be obvious if it is removed immediately after the operation. It may be removed after four to six hours, but can be left on until the next day.

A Collector for Skin Scrapings.

By F. W. LAZENBY, Stock Inspector, Port Elizabeth.

All who have had the experience of trying to take skin scrapings with a knife in the open air will appreciate the difficulty of collecting the samples before they are blown away. In windy weather the process is most unsatisfactory and at times almost impossible. For this reason I have devised an instrument, " The Lazenby Scraper Collector," with which I have been able



The Lazenby Scraper Collector

to collect scrapings with a minimum of trouble under all weather conditions. The collector is a piece of metal tubing, one end plugged with a rubber stopper and the other provided with a sloping aperture and a scraping edge (see sketch). In the collection of scrapings the hair is clipped from a small area of skin and the aperture and scraping edge of the collector pressed against this area. By moving the instrument up and down scrapings are removed and are retained in the barrel. They may then be collected by removing the rubber stopper.

CORRESPONDENCE.

ROARING AND WHISTLING IN THOROUGHBRED HORSES

Johannesburg.

The Editor,
Journal, South African Veterinary Medical Association,
P.O. Onderstepoort.

Dear Sir,

An article in the December number of the S.A.V.M.A. Journal prompts me to write this. Some time ago I brought a proposal before the S.A.V.M.A. about the importation of unsound horses into this country. This proposal, after some opposition, was carried, but although it was intended for the Government I cannot find that it reached this destination, although the Government derives a substantial sum per annum from the tax on imported race horses. This tax applies only to horses imported for racing, those for breeding come in tax free.

The man who imports and pays tax wants to win races and therefore makes some provision to see that the animals are sound. The breeder who imports duty free, brings in any unsound animal, because, as Drs. Quinlan and Steyn remark, they can be obtained at a much reduced price.

Sound animals to race; unsound ones to breed from, what a noble effort to improve the breed of horses in this country!

Dr. Quinlan states that roaring is not increasing in South Africa and that it is probably not hereditary. I have had nearly 40 years' experience with thoroughbreds both in this country and in England, and I feel I cannot let this statement go unchallenged. Roaring is on the increase. Thirty years ago I knew of only one South African bred horse a roarer and this was a galloway named 'Elphinstone' sired by a bad roarer. To-day we can find them on any race course; fourteen have been treated at Onderstepoort, but this does not comprise all the roarers in all our centres. The increase in the number of roarers was very apparent to me when I returned to this country in 1933, after ten years' absence. It is true that more horses are being bred, but the roarers are out of proportion to the number bred.

At one time it was thought that one could not breed roarers in South Africa. Since the influx of roaring sires we have bred them, and plenty of them. During the last twenty years, fifty per cent. of the stallions imported have been wrong in their wind. May I ask why we should import unsound animals, no matter what their breeding and turf performances, when sound animals can be obtained? It is a question affecting the whole horse breeding industry of the country and not merely the breeders' pockets. The breeders, to quote from the article in the Journal of the S.A.V.M.A., were forced "to take a chance." A chance of what? Filling the country with unsound horses?

It is, however, pleasing that there are still some breeders who will not have a roarer, and we have NON-roarers just as well-bred and performed as the roarers, but not of course bought at a roaring price, which in many cases is what one likes to offer, for roarers have no value in the country of their origin and no other country wants them.

The following horses developed roaring without any previous illness:— 'Floros,' 'Herne Bay,' 'Stormont,' 'Primus,' all sons of 'Kerasos' (roarer); 'On the Square,' son of 'Rampart' (roarer). These horses, according to the information given to me by their trainers, "just turned roarers," some at three, some at four, and some at five years. These are all Johannesburg-trained horses. What other centres of racing have experienced I do not know. 'Ponell,' when he won the Natal Derby and Guineas, was a whistler and his wind got worse. He won races after his operation. 'Stormont' won the S.A. Derby on an objection, *then* sound in his wind; subsequently, for no apparent reason, he developed roaring. He was operated on, put into training, but turned out useless.

'Waterproof' became useless for racing and was given away as a hack.

The examples given of the four sons of the roaring 'Kerasos,' all roaring for no apparent reason, will surely give supporters of the non-hereditary viewpoint some food for thought. There was no history of previous illness; then why did they roar, if their roaring sires had nothing to do with it? I can only come to one conclusion —— they inherited it from one or perhaps from both parents. I will readily admit that after strangles and biliary fever some, but not all, horses roar. Those with roaring ancestors are invariably the sufferers, and the roaring ancestor may be two generations back. Roaring stallions in some cases have not produced many roarers, but their sons whose

wind was sound have been roarer producers. Those who deny the hereditability of roaring are forever quoting the roaring mare 'Pocahontas' who produced 'Stockwell' to bolster up their theory. If they had troubled to look a little deeper into the stud record of 'Pocahantas' they would have found that her first foal, a colt by 'Camel' was a roarer. A glimpse into the attitude of some countries (and these countries breed good horses) to roaring stallions may be of interest. In February 1939, a conference was held in the Agricultural Hall, Islington, to discuss various statements which had appeared in the press stating that roaring was not a hereditary disease. Dr. Adair Dighton, F.R.C.V.S. said it was non-hereditary. The President of the Clydesdale Horse Society said, "Roaring in horses is hereditary and they should not be bred from." One may assume this gentleman had some experience of horse breeding to make so definite a statement.

Lord Rosebery, President of the Thoroughbred Breeders Association said, "From my study of the pedigree of horses, I agree with the opinion of the National Veterinary Medical Association that roaring is hereditary." This from a man, a keen student of breeding, who bred a Derby winner. Dr. Bullock, Secretary, Royal College of Veterinary Surgeons, "I beg to inform you that in 1888 this College was asked to supply information as to what were considered to be hereditary diseases of horses to the Royal Commission on Horse Breeding. Circulars were issued to all Veterinary Surgeons and the replies tabulated. As a result it was found that there was a consensus of opinion that roaring must be classified as a hereditary disease. The rules as to the licensing of stallions in France also provide that a stallion suffering from chronic roaring is not eligible for a licence. This was settled by the law of August 14th 1885, supplemented by the law of March 8th 1923. Stallions are not bought by the State Stud Administration if they suffer from chronic roaring, and if a stallion belonging to the stud becomes a roarer he is either castrated or destroyed."

Very good for France. I might mention that tubed horses are not allowed to compete in races in France. I cannot say whether this rule also applies to horses operated on for roaring.

Mr. J. B. Robertson, M.R.C.V.S., 'Mankato' of the 'Sporting Chronicle,' one of the greatest authorities on Thoroughbred horse breeding in the world, said: "I have noticed in the press recently certain irresponsible and illogical expressions of opinion that roaring in horses is not hereditary. Not one of the writers observes the first principle in the grammar of genetics, namely, whether or not the incidence of roarers and whistlers is larger in the descendants of roarers and whistlers than in the off-spring of horses and mares not affected with the lesion, or in the cases of horses in general. After collecting data for over sixty years in the case of Thoroughbreds and examining hundreds of horses and mares from the hereditary aspect in Thoroughbreds, and also very many horses for the Ministry of Agriculture under the Horse Breeding Act of 1919, the evidence at my command is overwhelming that paralytic roaring and whistling are due to hereditary factors..... There is no better illustration than 'Lily Agnes' (roarer), her son 'Ormonde' (roarer), his son 'Goldfinch' (roarer), his daughter 'Chelandry' (roarer." [Mr. Robertson could have added 'Chelandry's' son 'Traquair' (roarer)]. Mr. Robertson goes on to say, "I doubt if those who claim that roaring is purely an acquired defect know anything about the ancestry and history of roaring Thoroughbreds. I have not noticed that they advance any statistical data in support of their pathological and Weismann theories."

There would have been more interest, from the hereditary point of view, in the list of animals operated on at Onderstepoort, if suggestions had been

made as to why these animals roared, e.g. strangles, biliary fever, roaring parents. We are told so many horses roared, and that roaring is not hereditary. I want a much more convincing statement before I follow the Onderstepoort authorities.

Some of the horses operated on are got by roarers, and others have roaring strains on the dam's side. I should, however, like the non-believers in the hereditability theory to explain why 'Boy Cherry' roared. He was by 'Rampart' (roarer), out of 'Cherry Red' and own sister to the roaring 'Camelot' also operated upon at Onderstepoort. 'Cherry Red' and 'Camelot' were both by 'Kerasos' (roarer). This is in-breeding to roarers with a vengeance.

In Tattersall's sale catalogue is the following:—'Caffyns' by 'Coronach' out of 'Buck Barn' (Hobdayed), 'Pike Barn' by 'Papyrus' out of 'Buck Barn' (Hobdayed) ('Hobdayed' is another term for the operation for roaring). These horses are by different sires but out of the same dam and they may have inherited roaring from 'Buck Barn,' a daughter of 'Buchan,' who has a roaring son at stud in this country.

I await with anxiety an explanation of why 'Boy Cherry,' 'On the Square,' both sons of the roaring 'Rampart,' and also why 'Floros,' 'Herne Bay,' 'Stormont,' 'Prusus,' four sons of the roaring 'Kerasos,' were all roarers.

I am,

Yours faithfully,

BEN RUNCIMAN.

Memorial to the late Capt. W. E. Footner, *M.C.*, M.R.C.V.S.

As a mark of their esteem, a number of the veterinary friends and colleagues of the late Capt. W. E. Footner have subscribed to the erection of a headstone in his memory. Mrs. Footner, his widow, expressed her deep appreciation, both of the memorial itself, and of the thought which prompted its erection.

A. W. D.

GOLF.

The annual bogey competition for the du Toit Trophy was played off on the 1st May at the Murray Farm Golf and Country Club. The recent rains produced just the necessary improvement in the course to make the conditions perfect. All participants agreed that the catering, the standard of golf, and the keen competitive spirit shown resulted in a most enjoyable day's golf.

The winner of the Trophy was R. du Toit with I. P. Marais as runner-up.

The afternoon was devoted to a four-ball-better-ball bogey competition which resulted in a win for R. A. Alexander and I. P. Marais; second place was taken by the dark-horse combine of D. B. Smuts and G. B. Lawrence.

V. Cooper of the Field Staff came over specially from Johannesburg to compete. The organising committee would welcome other competitors from the Field Staff. Applications should be forwarded to the Secretary of the S.A.V.M.A. before the 1st of April of each year.

B. S. P.

BOOK REVIEWS.

This monograph* contains a chapter on Biological Therapeutics by Adolph Eichhorn. Milks has introduced numerous changes into this edition, and sections on the autonomic nervous system and sex hormones have been added. The section on vitamins has been brought up to date.

A large number of drugs, including nicotinic acid, benzedrine, lentin, mandelic acid, and sulphanilamide preparations, have been added to those discussed in the third addition.

I have to repeat here what I said in the review of the 3rd edition of this book about the therapeutic doses of carbon tetrachloride, namely, that the doses prescribed for cattle and sheep are dangerously high and will in many cases cause mortality. D. G. S.

* **Practical Veterinary Pharmacology Materia Medica and Therapeutics.** By H. J. Milks, D.V.M., Professor of Therapeutics and Director of the Small Animal Clinic, New York State Veterinary College at Cornell University, Ithaca, N.Y. 4th Edition. 1940. pp. 581. Baillière, Tindall & Cox, London. Price, 35/-.

THE ASSOCIATION.

Secretary's Report for the Year Ending 31st March 1940.

Membership. — The membership on 31st March 1940 was 176 (170 ordinary members, 4 life members, and 2 Honorary Life Vice-Presidents). The death of two members (W. G. Barnes, and W. A. Simson) during the year is recorded with regret. Six new members were elected at the last General Meeting and at the same meeting Mr. F. J. Carless was elected Honorary Life Vice-President.

Council. — Four meetings of Council were held.

Veterinary Officer: S.A. Permanent Force. — After a struggle of approximately eight years the Association has at last succeeded in obtaining a substantial professional allowance for the above post. The success which attended our endeavours in this connection is reflected not only in the allowance obtained for the post in question but also in the salary and professional allowance now paid to those of our members who are being called up for full-time service in the S.A. Veterinary Corps. The veterinary profession in the S.A. Force to-day occupies a status equal to that enjoyed by any other profession.

Hormone Treatment. — A special meeting of Council was held to consider the question of hormone treatment of race horses, because of the serious view taken by the Jockey Club of South Africa on this method of treatment. A special Committee was appointed to consider this question from every angle, and this Committee has issued a very comprehensive report which should serve as a useful guide to interested bodies.

Prosecutions. — Successful legal action was undertaken against an unregistered person who was alleged to have issued a Veterinary Certificate.

Book Fund Prize. — The S.A.V.M.A. Bood Fund Prize for 1940 was awarded to Mr. F. J. Veldman.

General Meetings. — On account of the war, the scientific portion of the Annual General Meeting was cancelled and only a business meeting was held on 15th November 1939. A Special General Meeting to consider the question of the utilization of Association funds for war charities was held on 14th December 1939.

Finance. — The appended financial statements reveal a very satisfactory position with regard to all the funds controlled by the Association. The only disquieting feature is the large amount outstanding under "Arrear Subscriptions." This no doubt is due, in a large measure, to the unsettled conditions that followed the outbreak of war. Those responsible for conducting the affairs of the Association, and particularly the Editorial Committee of the Journal, have no wish to relax their efforts in any way, as it is realised that the profession in South Africa through the Association and its Journal has within recent years obtained world-wide recognition. An appeal is therefore made to all members to fulfil their obligations towards the Association. Unless this is done the Council will be obliged to refer the names of several delinquents to the next General Meeting for consideration.

It is desired to draw special attention to the Benevolent Fund which, started only six years ago, has now a credit balance of £459 2s. 9d. There were no calls on this fund during the past year. Unfortunately dependants of deceased colleagues seem to lose contact with the Association in the course of time so that there may be cases in which assistance is badly needed but is not given because they are not brought to our notice. Members are therefore requested to inform the Secretary, or any member of Council, of any such cases which come to their notice in their respective districts.

The year under review has been a most difficult one as a result of the international upheaval, and, as can be expected in a country peculiarly subjected to political passions, situations arose which required delicate handling. The great tact, patience, and loyalty displayed by our President have, however, inspired every member to place the interests of the profession above personal considerations. Further, we owe him a great debt of gratitude for his leadership during this period and for the many sacrifices made by him in order to promote the interests and welfare of the Association.

Our thanks are also due to all those members who have helped in various capacities to carry out the functions of the Association.

S. W. J. van Rensburg,
HON. SEC.-TREAS., S.A.V.M.A.

Report of Finance Committee for 1939-40.

Members of Committee: H. H. Curson, B. S. Parkin, A. D. Thomas and J. G. van der Wath.

The books were audited by the Association's Auditor and the financial position of the Association is shown by the appended statements.

A. Cash Statement of Income and Expenditure 1939-40.

Receipts.				*Expenditure.*			
Credit Balance on 1/4/39	£98	2	7	Printing of Journal	£205	4	6
Members Subscriptions	314	2	6	Stationery	7	11	6
Donations: Benevolent Fund	12	12	0	Clerical Assistance	38	0	0
Book Fund	359	9	7	To Benevolent Fund	49	7	0
Subscriptions to Journal	30	9	0	Book Fund	359	5	7
Sale of Reprints		7	10	Travelling Expenses: President	10	2	0
Advertisements	29	17	8	Book Fund Prize (1939 and 1940)	20	0	0
Donations: Footner Fund	6	0	0	Footner Memorial	11	5	0
Interest	20	0	0	Natal Branch	10	5	0
				Auditing	5	5	0
				Legal Expenses	11	3	0
				Council Meeting Expenses	2	2	0
				Wreath		11	0
				Railage	1	2	9
				Petty Cash (Postage, Phones, Typing, etc.)	30	0	0
				Bank Charges	4	3	0
				Credit Balance on 31/3/40	105	13	10
	£871	1	2		£871	1	2

B. Balance Sheet, 1939-40.

Assets.				*Liabilities.*			
Union Loan Certificates	£750	8	0	Caxton Printing Works	£44	0	0
United Building Society	500	0	0	Subscriptions Paid in Advance	39	18	0
Arrear Subscriptions	194	1	0	Natal Branch	8	17	0
Cash in Bank 31/3/40	105	13	10	Credit Balance on 31/3/40	1,461	13	5
Cash in Hand 31/3/40	4	5	7				
	£1,554	8	5		£1,554	8	5

C. Benevolent Fund, 1939-40.

Credit Balance on 1/4/39	£379	5	6	Expenses	Nil		
From General Account	36	15	0	Credit Balance on 31/3/40	£459	2	9
Donations	12	12	0				
From Group Insurance Account	15	14	9				
Interest	14	15	6				
	£459	2	9		£459	2	9

D. GROUP INSURANCE ACCOUNT.

Credit Balance on 1/4/39	£15	14	9	Premiums Paid	£725	17	1
Premiums Collected	747	13	11	To Benevolent Fund	15	14	9
				Refund: Amount Overpaid		5	0
				Bank Charges....	1	12	9
				Credit Balance on 31/3/40	19	19	1
	£763	8	8		£763	8	8

S. W. J. van Rensburg,
HON. SEC.-TREAS., S.A.V.M.A.

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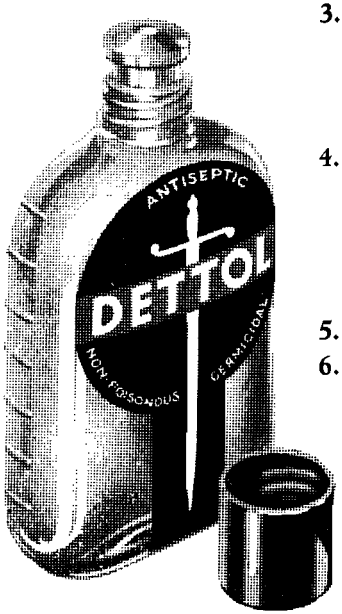
The striking success of Dettol for medical and surgical purposes soon led to a large number of enquiries and enthusiastic comments on its advantages from veterinary surgeons. This was not surprising, because the valuable properties of Dettol make it eminently suitable for veterinary purposes. The principal advantages are :—

1. Undiluted Dettol is non-toxic even when accidentally swallowed in considerable quantities.
2. Dettol can be employed on wounds at far higher germicidal strength than the older types of disinfectant, without interfering with the natural process of tissue repair.



3. Dogs and other animals usually remain quiet during treatment with Dettol as the solutions neither irritate nor sting raw wounds.
4. It has been shown that the film of Dettol which is left when solutions of it dry on the skin or wounds possesses antiseptic protective properties for several hours subsequently.
5. Dettol has an agreeable odour.
6. Many observers have commented on the fact that dogs show little tendency to lick off Dettol dressings, although, if they did so, it would not harm them.
7. Clinical reports indicate that flies are kept from wounds treated with Dettol.
8. Dettol does not stain either the skin or fabrics.
9. Dettol has considerable cleansing action apart from antiseptic properties.

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Jl. S.A.V.M.A.
XI (4) : 123-137.
1940.

Some Physiological Aspects of Mammary Function with particular reference to the "Drying off" of Dairy Cows and Chronic Bovine Mastitis.

By H. P. STEYN, Onderstepoort.

Chronic bovine mastitis is not a new disease, but the tremendous economic importance which is attributed to it is a new phenomenon. The incidence of chronic mastitis in South Africa has not been thoroughly investigated but there is some evidence that the disease is as prevalent in this country as elsewhere [van Rensburg (1939)]. There are undoubtedly a number of factors which have contributed towards the greatly increased prevalence of chronic mastitis, but only a few of these factors, which are not commonly stressed, will be discussed in this article. Factors such as improper dairy hygiene, ignorance on the part of dairymen, etc., will not be dealt with, but certain methods of dairy management which can be considerably improved and which may play a very important part in the development of mastitis will be discussed. In the "drying off" of dairy cows, particularly, I suggest that improved methods should be adopted and the greater part of the article will be devoted to reviewing those aspects of the physiology of mammary function which may assist in finding a method of "drying off" which is least harmful to the udder.

Before, however, undertaking the main theme of this paper, namely the physiological functions of the mammary gland which have a direct bearing on "drying off", and the possible significance of incorrect "drying off" in the etiology of mastitis, I cannot resist mentioning some of the modern tendencies in dairies and stud establishments which assist in the development and spread of mastitis. The greatly intensified methods of modern commercial dairying have probably contributed much towards creating the present mastitis problem. These intensified methods include, *inter alia,* an increased amount of stabling, feeding for continuous maximum production, and a reduction of the period of non-lactation to a minimum. Stabling unquestionably results in the spread of infection and this effect is seen not only with mastitis but also with certain infectious diseases of the genitalia, tuberculosis, etc. Under South African conditions there appears to be no reason for stabling except to milk and feed. Several dairymen have been advised to discontinue stabling and their experience has been that once the cows are accustomed to staying out at night no appreciable difference in the milk yield is noticed, even during the coldest part of the year. Some dairymen have expressed the opinion that slightly more feed may be required for a short period during winter months to maintain the same level of milk production. If cows are run in a sheltered paddock increased feeding will probably not be necessary.

The continuous forcing of dairy cows for the purpose of maintaining maximum production is probably a shortsighted policy. The dairy cow, once her productive life has started, becomes merely a mechanical unit and the limits of physiological production are forgotten. Indeed this tendency is not only apparent in dairies but it controls the breeding policy of all stud breeders. There must be a limit to physiological production and that limit has probably already been exceeded. There is some evidence that mastitis is more prevalent in high milk-yielders than in lower producers [Munch-Petersen (1938, p. 10)], and this may to a large extent explain the veterinary problems encountered almost exclusively in dairy herds. As examples of these problems one must mention infertility and chronic mastitis which are much more rife in dairy cattle than in other bovines or other animal species. I do not wish to suggest that feeding for maximum production should be discouraged, but a longer rest period between lactations must be considered. This, of course, would mean that the milk yield from each cow would be reduced slightly, but other advantages may be gained which would more than compensate for this loss. In addition I should have no hesitation in advising a breeder to aim at breeding cows with a good robust constitution and with only moderately high production, rather than breeding for high production only. One hesitates at being too dogmatic when discussing these broader aspects of the mastitis problem because at present there is very little concrete evidence to guide one, but nevertheless these are important aspects of disease control in dairy cattle which deserve more attention than has been given them.

The advice that a longer period of rest should be allowed cows between lactations is supported by sound physiological reasons. The period of rest is the period during which body reserves are built up and tissue repair in the mammary glands can be accomplished. High milk production drains the body reserves more than pregnancy does, and a period of rest from lactation during at least the last three months of pregnancy is considered necessary. The non-functioning mammary gland undergoes the most active preparation for lactation during the last two to three months of pregnancy, and if a cow could be dried off immediately before this period, enough time would probably be allowed for mammary repair.

The reason for recommending a period of rest to allow for mammary repair may not be obvious. It is based on the observation that injuries to mammary tissue, with resultant inflammatory changes and without infection do occur and it is extremely likely that in many cases proper tissue repair does not take place in the actively functioning gland. It must be remembered that the mammary gland was developed to suckle the young during a comparatively brief period which was followed by a relatively long period of rest before the next lactation. The highly developed mammary gland which functions so efficiently and persistently in our dairy cattle is actually an abnormal organ produced by artificial selection. The possible

significance of the aseptic inflammatory changes referred to will be more fully discussed under the pathogenesis of mastitis.

The advice that a longer "dry period" should be allowed dairy cows brings us to our main problem, the importance of which has not been sufficiently stressed, namely how lactation should be stopped or how the dairy cow should be "dried off." It is my opinion that there is no really safe method of interrupting lactation, and that the popular practice of incomplete or intermittent milking is probably the worst that could be adopted. A discussion of those aspects of the physiology of mammary function which are involved when the mammary gland changes over from the lactating to the non-lactating state will disclose my reasons for making this statement.

The Secretion of Milk.

The modern explanation of many important phenomena of lactation has made untenable a number of old theories about the causes of mammary disease. The first important fact explained is that milk secretion is definitely a continuous process [Espe (1938), pp. 72-77; Hammond (1936); Swett, Miller and Graves (1932)]. Before the work of Swett and his associates in 1932 the incorrect theory that the secretion of milk took place at the time of milking was popular. The milk which is, however, secreted uninterruptedly is stored in the complicated system of ducts and cisterns in the udder. Espe (1938), in his very excellent monograph on the secretion of milk, states that the rate of secretion is most rapid immediately after milking, when intramammary pressure is at a minimum. This means that within a very short period after milking, milk would once more have accumulated in the teat and lactiferous sinuses. Finally, even though a cow has been thoroughly milked, only about 80-85 per cent of the milk present in the udder at the time of milking is removed [Hammond (1913), Espe (1938), p. 69].

These facts make one doubt that thorough stripping is as essential to the control of mastitis as it has been claimed to be. Woodward (1936) describes an experiment in which he investigated the effect of incomplete milking in relation to milk production and udder troubles, and he concluded that leaving a small quantity of milk in the udder had no effect on milk production, nor did it cause mastitis. Very little work has been done to determine the influence of incomplete or other systems of milking on the udder, and the problem needs more thorough investigation. The effects of incomplete and intermittent milking in relation to "drying off" will be more fully discussed later. Anyone desirous of obtaining more detailed information about the physiology of milk secretion and mammary function should read the works of Hammond (1936) and Espe (1938).

The Effect of the Mechanical Stimulation of Suckling or Milking on Mammary Function.

The stimulation caused by milking has a very definite effect on lactation. Hammond and Marshall (1925) showed in experiments on rabbits that " suckling keeps the mammary gland active, whereas failure to suckle, which results in an accumulation of milk in the alveoli, leads to cessation of function and atrophy." Wayne *et al.* (1933) stopped milking a cow completely for 11·5 days; they then milked the left half of the udder at regular 12 hourly intervals and found that the milk yield from this half returned to normal in quantity and composition. In the right half of the udder, which was not milked, milk secretion was not re-initiated. Turner (1931) is quoted by Espe (1938) as having initiated lactation in a non-pregnant cow by the mechanical stimulation of milking.

It is obvious, therefore, that continued milking of a cow which is being " dried off " should be avoided if possible. Nelson (1936), in his review of the literature on the subject, indicates that in small laboratory animals milk secretion may be maintained by suckling even after ligation of the galactophores. Incomplete and intermittent milking, the systems most popular in " drying off " dairy cows, therefore actually result in a repeated stimulation of the mammary gland and must lengthen the period of " drying off."

The Effect of Intramammary Pressure on Milk Secretion.

The effect of intramammary pressure on milk secretion has been fully discussed by Hammond (1936) and Espe (1938). The intramammary pressure gradually increases from one milking to the next, and immediately before milking the pressure at the base of the teat usually averages from 25 to 35 mm. Hg. There are however wide variations (Espe p. 70). If milking is stopped this pressure gradually increases up to the 16th to 30th hour, when the maximum pressure is reached [Garrison and Turner (1936)], after which milk secretion is stopped or checked and the composition of the milk in the udder begins to change. This interval will vary somewhat depending upon individual factors, but is always relatively short. The rate at which milk is secreted decreases rapidly as the intramammary pressure increases between milkings [Ragsdale, Brody and Turner (1924); Bartlett (1929)].

The mechanism by which milk secretion is slowed down and finally stopped by the steadily increasing intramammary pressure is explained by Hammond (1936) as being purely mechanical — the mammary alveoli become distended, the secretory cells stretched and compressed, and finally atrophied. Finally regression of the alveoli and ducts takes place and during the next pregnancy they regenerate.

The pressure which is attained in the udder as a result of abrupt cessation of milking apparently starts decreasing relatively rapidly, if other

factors which stimulate milk secretion (e.g. milking or suckling, feeding of concentrates, etc.), are removed or reduced to a minimum. The effect of milking or suckling on mammary function has been discussed, the effect of feed will be discussed later. Wayne, Eckles and Petersen (1933) reported that sudden cessation of milking resulted in considerable distension of the udder. The marked tension, however, started to subside from the second to the third day and resulted in much more rapid "drying off" than intermittent or incomplete milking. When intermittent milking is practised in "drying off" cows it is the general rule that the period between milkings is gradually increased until the cow is milked three times and then twice a week, etc. From the foregoing discussion it is apparent that milk secretion usually stops approximately 20 hours after milking and is not re-initiated until the intramammary pressure is reduced by the removal of the milk accumulated in the udder. Milking a cow every second or third day therefore re-initiates lactation and must result in a prolonged period of "drying off."

There can be no doubt that "drying off" will be more rapid following abrupt cessation of milking, but the nature of the changes in the fluid which is retained in the udder, the effect on the resistance of the udder to infection, and a number of factors which may affect the behaviour of any latent infection present in the udder have not been examined sufficiently critically. Thus Espe (p. 178) states that there is a preliminary increase in the number of bacteria present in the udder, but that their activity is checked as soon as the fluid in the gland has changed so as to resemble blood-serum more than milk. (The fluid in the udder soon becomes rich in serum-albumin and serum-globulin). Little (1939), however, states that changes in pH, chlorine and cell content of milk may adversely influence its bacteriostatic value. This aspect of the problem should be more thoroughly investigated. Whatever the correct interpretation may be, it is obvious that intermittent and incomplete milking must lengthen the period of increased bacterial activity in the udder. It has been proved [Wayne and Macy (933)] that there is a period of increased bacterial and cell content of the fluid in the mammary gland with both methods of "drying off." It would therefore be an advantage if this period could be made as brief as possible.

Espe (1938) advances the theory that intermittent intramammary pressure may be an essential factor in the maintenance of lactation. His evidence in support of this theory is really based on the fact that high intramammary pressure adversely influences milk secretion in that it slows down the rate of secretion and interferes with the secretion of fat. Espe argues that one would therefore expect that inserting a catheter in a teat so that the milk would flow away continuously should overcome this disadvantage. Actually, however, it results in a greatly lowered milk yield. Similarly he points out that when there is a milk fistula in a teat, so that

the milk flows away uninterruptedly, that quarter soon stops secreting milk. From these observations he claims that intermittent intramammary pressure is essential for the proper functioning of the mammary gland. This theory cannot easily be disproved, but I doubt its validity. To me it seems that the decreased milk yield and cessation of secretion noted by Espe are actually due to increased intra-alveolar pressure in the udder. Bearing in mind the now generally accepted fact [Hammond (1936) thoroughly reviews this subject] that the "letting down" of milk is an active process, it is obvious that in the absence of a stimulus to bring about this "letting down" a large amount of milk must remain trapped in the alveoli and smaller ducts of the mammary gland. The pressure in these alveoli and ducts must increase, although it probably would not become as high as during the normal interval between milkings. If this argument is correct the manipulation and milking of a cow after catheters have been inserted in her teats for a 24 hour period should result in the retained portion of the usual yield being obtained. A small experiment is being planned for the purpose of testing this theory. Whichever theory is correct neither is a reason for objecting to the abrupt cessation of milking when drying off a cow, but both could be used as objections against any method of milking during the "drying off" period.

The Influence of Feed on Milk Production.

The effect of feed on the quantity and quality of milk is so thoroughly appreciated that its lengthy discussion is unnecessary. The significance of feed cannot be over-emphasised and one fully appreciates the hyperbole of Hammond (1936) when he says: "We consider that, since the efficiency of feeding for high milk production has risen to such a high level, further progress in obtaining larger yields of milk and butter fat is now being limited more by milk pressure than by any other factor." It is, therefore, sufficient to stress that prior to "drying off" a cow, by any method, her feed must be properly controlled. Espe (1938) p. 178 advises: "For cows giving over 20 lb. of milk a day the ration should be reduced until production falls or the cow is no longer gaining in weight." He gives no details about the actual way in which the ration should be reduced but general usage and experience indicate that the best results are obtained by withholding all concentrates and green feed and, if necessary, limiting the amount of water consumed. When a cow is in advanced lactation this change in her ration causes a marked reduction in milk yield. During the early stages of lactation a change in feed does not exercise as marked an effect. I am not aware of any work which describes any more scientific method of controlling feed than the method described.

There are certain particular constituents in feeds which are essential to the production of milk of the best quality, and which also influence the quantity of milk secreted, but none of these appear to be of any practical

significance in the " drying off " of cows and one is obliged to follow the somewhat general rules indicated.

The Hormonal Control of Mammary Function.

The hormonal control of mammary function has been the subject of many treatises and a great deal of invaluable work has been done to explain mammary development and function. In the human this work has resulted in a very satisfactory method of interrupting milk secretion during the early stages of lactation, by the use of oestrin or synthetic oestrogenous substances [Foss Philips (1938), Anon (1938)]. The effect of oestrin on lactation in the cow has not been investigated, but there is little likelihood of success following its use because it would have to be used during advanced lactation. There is actually an objection to its use in advanced lactation, as most cows are pregnant at this time, and Folley, Watson and Scott (1939) have reported that abortion followed the use of stilboestrol dipropitionate as an inunction. There is no record of the successful interruption of lactation by hormonal treatment, except the instances cited in woman, and at present there seems to be little likelihood of finding a satisfactory method of applying hormonal treatment to stop milk secretion in the bovine.

The Significance of Rapid Milking, thorough Stripping, Incomplete and Intermittent Milking, etc., in the development of Udder Troubles.

If the maximum milk yield is to be obtained rapid and efficient milking is essential. This is because the " letting down " of milk is an active process resulting from an " erection " of the mammary tissue when milking is commenced. If milking is not rapidly carried out the state of " erection " passes off and a large proportion of milk is retained in the alveolar and duct system of the mammary gland. The effect of intramammary pressure on milk secretion has been discussed and as a result of this discussion the recommendation was made that milking should be stopped abruptly to end lactation. The converse, it should be pointed out here, is true too: *viz.* that to obtain a higher milk yield more frequent milking should be adopted. The explanation for the higher milk yield obtained when milking at shorter intervals (e.g. six or eight, instead of twelve hourly intervals) is that the intramammary pressure is not allowed to rise so high between milkings as it does when longer intervals are allowed. As explained earlier, the intramammary pressure rises fairly rapidly from one milking to the next, and milk secretion slows down as the intramammary pressure rises [Hammond (1936), Espe (1938)] The advantage of milking at short intervals is seen only in high producers in which milk secretion is so rapid that the udder cannot accommodate all the milk produced between milkings. By combining rapid and efficient milking with milking at 6-8 hourly intervals in the high milk producers, the intramammary pressure is probably pre-

vented from becoming excessively high, and this would reduce the danger of injury and mastitis.

The intramammary pressure which inevitably develops during " drying off " may be an important etiological factor in the development of chronic mastitis, but there appears to be no way of overcoming this danger and the best procedure would therefore be to try to reduce the period of danger as much as possible. This can only be done by adopting the method of complete cessation of milking combined with properly controlled feeding. The possible significance of high intramammary pressure will be more fully dealt with under the heading of the pathogenesis of mastitis.

The significance of thorough stripping has been briefly discussed and we concluded that " thorough stripping " is never really attained as only 80-85% of the milk present in the udder is removed by the most efficient methods of milking [Hammond (1913)]. The most important advantage to be gained by thorough stripping is probably purely economic. Woodward (1936) investigated the problem of incomplete milking in relation to udder troubles and milk production and he found that milking by machine left an average of 1·2 lb. (minimum 0·8 lb., maximum 2·1 lb.) of milk in the udder. He concluded: " Incomplete milking did not increase either the number of cells, the number of total bacteria, or the number of streptococci in the milk when the averages of all lactations are considered," and that " Since leaving a pound or two of milk in the udder at each milking does not affect the percentage of fat in the milk, the normality of the milk, the persistency of lactation, nor the health of the cow, the only questions having a bearing on whether or not stripping should be practised on a dairy farm are the value of the milk obtained by stripping, the cost of stripping, and the sanitation of the product." Maurer and Steidle (1932), quoted by Woodward, similarly reported that incomplete milking for four months did not cause mastitis in cows with healthy udders nor did it cause the development of acute mastitis in an udder with latent infection. Wayne and Macy (1933) found that intermittent milking and complete cessation of milking caused a higher bacterial and cell content of the milk during " drying off," but that neither of these methods of treatment had any marked effect in the subsequent lactation. They found that the effect of both methods on the udder was essentially the same, but that " drying off " was much more rapid following complete cessation of milking. However they did not compare the duration of the period of increased bacterial and cell content of the udder following the different methods of " drying off," nor did they make a critical examination of mammary tissue to ascertain what the effects of the various methods of treatment were. In fact, there appears to have been no really critical work done on the effect of any method of " drying off " on the mammary tissue, in which histological appearances as well as changes in the fluid content of the udder have been thoroughly examined.

The few experiments noted are the only instances which could be found in which an effort has been made to disprove the generally accepted theory that incomplete milking is a direct cause of udder troubles. However, I am inclined to accept the conclusions of the few investigators reported because their conclusions seem to be logical when the modern interpretation of the physiology of milk secretion is borne in mind. It must however be clearly understood that the type of incomplete milking referred to is defined as that method of incomplete milking in which only a small amount of milk is left in the udder. When a large amount of milk is left in the udder the result would probably be similar to intermittent milking in which there is a great increase of intramammary pressure, a fairly prolonged period of increased bacterial activity, and a higher cell content of the milk [Wayne, Eckles and Peterson (1933), Wayne and Macy (1933), Woodward (1936)]. One would therefore anticipate that leaving a large amount of milk in the udder might cause the exacerbation of udder infections, clinical mastitis, and other complications. This aspect of the problem has not been investigated.

The old belief, which has been handed down from generation to generation of stockmen, is that incomplete milking does lead to udder troubles and a large number of prominent authorities have supported this theory, either as a result of general observation or of experiment [Klimmer and Haupt (1934), Schmidt-Hoensdorf and Schmidt (1932), Ernst, Schmidt-Hoensdorf and Schmidt (1931), Udall and Johnson (1933), and Steck (1921)]. However, the experiments undertaken to prove the ill-effects of incomplete milking are not convincing and are few in number. Thus Ernst *et al.* and Schmidt-Hoensdorf and Schmidt conducted experiments in which no record was made of the approximate amount of milk left in the udder, and in addition they artificially infected their cows immediately before practising incomplete milking — i.e. they practised incomplete milking during the acute stage of mastitis following artificial infection. The only conclusion one can legitimately make from their work is that incomplete milking during the acute stage of mastitis following artificial infection may cause a more severe mastitis of longer duration than when thorough stripping is practised.

It is interesting to note that Klimmer and Haupt (1934) seem to attribute the ill-effects of incomplete stripping to the greatly increased intramammary pressure which results. They consider that an accumulation of milk in the udder (Milchstauung) is one of the principal predisposing causes of mastitis and hence recommend milking all high milk yielders at regular 8 hourly intervals or even more frequently. To Klimmer and Haupt, therefore, incomplete milking meant leaving a relatively large amount of milk in the udder and as explained above this would be similar to intermittent milking.

Discussion.

From the preceding discussion of the literature referring to that part of the physiology of mammary function which is affected during "drying off," it is obvious that the method of artificially stopping lactation which is most in harmony with the functions of the mammary gland is the abrupt cessation of milking. It has however been clearly pointed out that no known method of interrupting lactation can be considered entirely safe. The chief danger in interrupting lactation is that there is a great increase in intramammary pressure and increased bacterial activity; but when intermittent milking is practised during the period of "drying off" the duration of these ill-effects is extended over a period of two to three weeks, whereas following abrupt cessation of milking the dangerous period is probably limited to less than a week. The lesser of two evils is therefore chosen when abrupt cessation of milking is recommended. From the discussion of the epidemiology and pathogenesis of mastitis which follows, it will become clear that the process of "drying off" probably plays a significant part in the spread of chronic mastitis and consequently it is important that we should exercise great care in making recommendations about the safest method of ending a lactation period.

Apart from the physiological and experimental evidence which has been cited in support of the recommended innovation in "drying off" dairy cows, the evidence of general clinical observation in justification of this method is worth mentioning. Most of us are familiar with the course of events when a cow freshens and one of her teat ducts is obstructed. In these cases a marked distension of the affected quarter takes place, but it atrophies and "dries up" in a comparatively short period without any complications, provided no surgical interference has been attempted. It was this observation which first caused me to doubt the correctness of the generally accepted methods of "drying off" cows and resulted in a search for experimental evidence in support of abrupt cessation of milking as a practical method of ending lactation. In addition Hammond (1936) states: "It is well known that the easiest way to dry a cow off is to leave milk in the udder and seal up the nipples."

Wayne and his associates (1933) sent a circular questionnaire to American dairy farmers and received 236 replies, from which they found that 16 per cent stopped milking suddenly, 76 per cent practised intermittent milking, and 8 per cent incomplete milking when drying their cows. No comments are made about the results of the various methods.

Abrupt cessation of milking is therefore not a new method, but it is comparatively unknown.

Epidemiology and Pathogenesis of Chronic Mastitis.

From a study of the literature in which an effort is made to explain the pathogenesis of chronic streptococcal mastitis, a few important facts are

found about which the majority of investigators agree. These facts are: firstly that infection definitely takes place via the teat duct (the galactogenous route); secondly that artificial infection by any method except direct introduction of fairly large doses of infective material into the lactiferous sinus (teat sinus) is extremely difficult and uncertain [Klimmer and Haupt (1934), Little (1937), Christiansen and Nielsen (1934), Munch-Petersen (1938)]. From these observations it became apparent that, for natural infection to occur and spread, a combination of factors which predispose to infection must be present. The list of predisposing factors which has been suggested is a very comprehensive one [Bendixen (1935), Christiansen and Nielsen (1934), Jones and Little cited by Little (1937), Klimmer and Haupt (1934), Peterson and Hastings (1939), Munch-Petersen (1938), Meigs *et al.* (1938)] but Munch-Petersen (p. 61) appears to be the only author who suggests that " drying off " may be an important factor in the establishment or the exacerbation of latent streptococcal infection.

A study of the epidemiology of streptococcal mastitis similarly discloses certain important observations about which there seems to be fairly general agreement. These observations are, that the incidence of chronic mastitis in dairy cows increases with age, i.e. that the lowest percentage of infected cows is found in cows passing through their first and second lactation period, and that, from the third lactation onward, the percentage of animals infected increases rapidly. The highest percentage of infected cows is found in animals at the eighth and ninth lactations. There is less agreement about the stage of lactation during which most cases of streptococcal mastitis occur, but the majority of investigators seem to agree that the most dangerous period is during the first few months of lactation. Stylianopoulos (1934) found that out of 117 cases studied 28 developed infection during the first 14 days following parturition, and that 31 became infected from the drying-off period until the next parturition. The remainder (58) became infected during the remaining part of the lactation period. Minett, Stableforth and Edwards (1933) similarly state that among ten old cows naturally infected with *Streptococcus agalactiae* in their experiment, they observed that clinical symptoms of mastitis became more obvious during drying off or after calving. The drying-off period and early lactation therefore appear to be critical times. The fairly generally accepted statement that the incidence of chronic mastitis increases with age justifies the suggestion that this is a result of the prolonged subjection of dairy cows to a combination of circumstances (or predisposing influences) which renders ultimate streptococcal infection, in infected herds, more or less certain. It is the purpose of this article to indicate that one almost constant factor in this combination of predisposing influences is an artificial method of terminating lactation in dairy cows, and I wish to suggest that before the effects of normal lactation and various methods of drying off on the physiology, pathology, and structure of the mammary gland have

been thoroughly studied, one cannot expect to solve the mastitis problem efficiently. It has been proved that a dairy herd can be maintained free from *Streptococcus agalactiae* infection [Minett, Stableforth and Edwards (1933), Plastridge *et al.* (1936)], and a study such as has been suggested, should therefore be possible. These authors also point out that since it is possible to maintain a herd free from *Streptococcus agalactiae* it would be possible to obtain more reliable information about other infections and to establish sound standards for normal milk in such an uninfected herd.

When considering the pathogenesis of streptococcal mastitis and the importance which is attached to predisposing influences in the establishment and exacerbation of infection, one is surprised to find that aseptic inflammatory conditions of the mammary gland are not mentioned. Yet there can be no doubt that aseptic inflammatory conditions which predispose to infectious mastitis, and indeed may even cause difficulties in diagnosis, must be common in a sensitive organ such as the udder which is so exposed to injuries, cold floors, and unsympathetic handling. Peterson and Hastings (1939) refer to what they describe as a "non-specific mastitis" which they suggest may be caused by a "virus," and they briefly review the literature dealing with this type of mastitis. They suggest the interesting hypothesis that "non-specific mastitis" is really the primary disease in all cases of chronic mastitis, and that it is followed by streptococcal invasion secondarily. Burkey *et al.* (1938) reported that they found that large numbers of leucocytes in the milk indicated an injury to the udder but not necessarily the presence of infection: i.e. they recognised the occurrence of aseptic inflammatory conditions. Minett and his associates (1933) similarly mention having found changes in milk without the presence of any suspicious organisms. When one considers the possible prevalence of aseptic inflammatory conditions of the udder during the critical drying-off and freshening periods, when the udder — due to circulatory disturbances, lymph extravasations, and high intramammary pressure — must be in an exceptionally vulnerable state, one readily appreciates that the condition which Peterson and Hastings describe as "non-specific mastitis," and which is here considered as probably a simple aseptic inflammatory state, must play an important part in the development of infectious mastitis. The paucity, if not the entire absence, of information about the "non-specific" and aseptic inflammatory conditions of the udder and their possible connection with streptococcal mastitis is striking, and it would be interesting to know to what degree indurations, and structural and secretory changes of the mammary gland may occur as a direct result of chronic aseptic inflammatory conditions. In concluding this part of the discussion it may be stated that there is a notable lack of knowledge about the pathology and pathological physiology of the mammary gland, particularly as so much emphasis is laid on udder troubles in dairy cattle. The result is that veterinarians are being forced, probably against their will, to undertake the

investigation of chronic infectious mastitis from the most difficult angle, and as a direct result empirical methods of treatment, control, etc., are being evolved and perpetuated. Those aspects of mammary function, histology, and pathology mentioned in this paper must be thoroughly investigated before more rational methods of control can be hoped for.

Summary.

The recommendation that drying off should be carried out by the abrupt cessation of milking is unquestionably based on sound physiological grounds, but the importance of controlling the feed properly before milking is stopped cannot be over emphasized. However it must be clearly understood that any veterinarian adopting this advice in practice should proceed with caution until more experience and knowledge have been acquired. A few cows have been dried off by this method at Onderstepoort, with excellent results, but only after the effect of repeated lactations followed by this method of ending lactation has been observed can reliable conclusions be drawn. As an example of the importance of experience of the method, one must indicate that the effect of abrupt cessation of milking may lead to more discomfort and greater intramammary pressure in the comparatively low milk yielder than in the heavier producer. This is attributable to the variation in udder capacity. The low yielders frequently have more fleshy and less elastic udders than the high yielders and this results in a more rapid and more marked distension of the udder in the former type. Similarly it is impossible to lay down any rule such as that suggested by Espe that any cow giving less than two gallons of milk per day may be dried off by this method. The low yielder may be producing very nearly her maximum amount of milk when giving two gallons of milk per day. It would be safer to say that a cow may be dried off when her milk yield has dropped to half the maximum yield or less, provided she is not giving more than two gallons per day. This would be a safe guide on which to work until more experience has been gained. However, in emergencies it does not appear necessary to adhere to any rule about the milk yield at the time of stopping lactation, and it is only with the object of minimizing temporary discomfort and any injuries which may result when the method is adopted as a routine that care must be exercised. It must be remembered that our sole object is to find a method of ending lactation which is safer than the old method, and that the ideal is to find an entirely safe method.

The old methods of drying off have nothing more than tradition to justify their application, whereas the newer knowledge arises from critical physiological investigation.

The practices founded on tradition in dairying are actually found to be contradictory when critically examined, as we are told that during lactation incomplete milking and irregular milking are harmful, whereas during drying off these are actually advocated in an extreme form.

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Sterilization of Bone Meal.

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Until recently the Union of South Africa's regulations for the importation of bone meal and similar animal products demanded a certificate of sterilization from the consigners stating that the bone meal had been exposed to 115°C steam for fifteen minutes or 140°C dry heat for three hours. In addition samples were taken from consignments at the Ports of Entry and sent to this laboratory for bacteriological examination. This examination enabled a watch to be kept on exporters to this country.

Towards the end of 1936 samples were obtained that contained large numbers of putrefactive anaerobes and that killed guinea-pigs on subcutaneous inoculation. During 1937 still more such samples were found, and a number of farmers reported anthrax outbreaks that they thought were due to bone-meal feeding. The samples of bone meal sent to us by these farmers were badly contaminated with putrefactive organisms although we could not isolate *B. anthracis.*

We were perturbed by the increasing number of samples that contained putrefactive anaerobes as we considered their presence an indication of insufficient sterilization. In 1938 (18.1.38) a lot of bone-meal submitted by a farmer who suspected anthrax contamination was found to contain *B. anthracis.* This was the first time *B. anthracis* had been found in bone meal examined at Onderstepoort. Shortly afterwards (4.3.38) *B. anthracis* was found in a consignment at a Port of Entry. During the following year eight more bone-meal samples were found containing anthrax; four sent in by farmers and four from the Ports of Entry. Thus between 18.1.39 and 2.2.39 ten samples were found to contain *B. anthracis.*

All the anthrax-infected bone meal intercepted at the Ports of Entry as well as most of the consignments found badly contaminated with putrefactive organisms were from overseas. Exporters blamed extraneous contamination (holds of ships, South African warehouses). This contention will be discussed later. On the evidence, however, we believed that in some way, probably innocent, unsterilized carcass material was being mixed with the bone meal.

Testing of Bone-meal Samples.

The test used here is as follows: thirty gm. of bone meal is thoroughly mixed with 35 cc. physiological saline. The mixture is heated at

65°C for two hours. It is then stirred and allowed to settle for five minutes. Two guinea-pigs then each receive two cc. of the supernatant. If both guinea-pigs survive, the sample is considered clean. If either or both guinea-pigs die without showing a specific disease (anthrax or gas-gangrene), the test is repeated and may be repeated several times. Appropriate cultural and serological examinations are also done. When, however, the bone meal consistently kills guinea-pigs in 24 to 48 hours and causes marked local lesions resembling gas-gangrene, even when the dose is reduced, it is considered " dirty."

This test is designed to detect specific infection and also gross contamination with putrefactive bacteria. We consider the latter indicative of insufficient sterilization or of serious admixture with unsterilized material. Obviously if bone meal contains large numbers of putrefactive organisms there can be no real guarantee of freedom from anthrax or other specific disease.

Certain manufacturers have contested this view and asserted that the putrefaction developed after sterilization and was thus no indication of under-sterilization, and that no bone meal could succeed in passing our test anyway.

Our experience, however, shows that this is a reasonable test and that most bone meals pass it with ease. The following observations bear on this point:—

1. (a) Bone meal was made (in this laboratory) from *unsterilized* dry abattoir bones. This passed our test. Unsterilized non-putrid bones, therefore, did not produce a " dirty " bone meal.

(b) Bone meals made from unsterilized thoroughly putrid bones failed to pass our test, but were not more lethal than imported " dirty " bone meals. Therefore the degree of contamination of the imported " dirty " bone meal was of the order of that of unsterilized putrid bone meal.

2. (a) Bones artificially infected with *B. anthracis* (spores forced into the medulla and cancellated substance) were processed under our control at two South African bone meal factories. Both the steam- and the naphtha-extraction process produced complete sterilization.

(b) Putrid bones processed under these conditions were also sterilized. The ordinary products from 13 South African factories using the steam-extraction process, and one factory using the naphtha process were clean by our test.

3. Consignments of " dirty " or anthrax-infected bone meal did not contaminate clean products carried in the same holds and housed in the same sheds.

4. Contamination was never found in specially processed bone meal such as degelatinized bone meal.

5. The imported contaminated bone meal was dry and showed no spoilage such as would be expected had putrefaction occurred *en route.*

A consideration of these observations shows the unlikelihood of either the *B. anthracis* or the other contaminations being due to casual contamination in transit.

Since these points were brought to the notice of exporters no anthrax contaminated bone meals have been found from 2.2.39 to the present (14.2.40) as compared with ten in the previous year; and the finding of bone meals full of putrefactive organisms has become a rarity.

The Manufacture and Sterilization of Bone Meal in South Africa.

It was found experimentally that both the steam-extraction process and the naphtha-extraction process of preparing bone meal sterilized bones efficiently. The former uses steam at 40 to 60 lb. pressure for from 5-8 hours. Obviously this will be effective even if the minutiae of sterilization technique (removal of air, saturation of steam) are not rigidly observed, as the safety margin is unlikely to be overstepped. In both these processes, the steam and the naphtha, the sterilization is secondary to a fat extraction for commercial purposes. Thus *incidentally* sterilization is ensured.

Some manufacturers, however, are primarily interested in making bone meal and naturally wish to employ the shortest sterilization time compatible with efficiency.

We therefore carried out experiments to see how long it took to sterilize bones in the steam digester of a bone meal factory.

Anthrax spores were forced into the medullary cavity and cancellated substance of bovine bones. These bones were placed in the middle of charges of bone in a four-ton steam digester. This was run at 40 lb. per square inch and the time was reckoned from the moment this pressure was reached. (This took about 15 minutes if the digester was hot). The bones were exposed to the steam for various times from 15 minutes to two hours. Care was taken that all air was blown out of the digester and that the steam was saturated. Under these controlled conditions *B. anthracis* still survived in bones exposed for one hour.

Regulations for the Importation of Bone Meal.

It is not advisable to frame regulations about the details of the sterilization procedure to be followed. A certain technique may be efficient and yet fail in particular instances owing to neglect, oversight, or accident. For example all the bad consignments of bone meal sent to South Africa were ostensibly sterilized by an unimpeachable process. Therefore we intend framing regulations insisting that the consigner guarantees "That the bone meal has been sterilized and is free from (1) *Bacillus anthracis* and (2) organisms of the gas-gangrene type." We are satisfied that these condi-

tions can easily be fulfilled commercially and are not onerous. Our tests will continue to control imported bone meal.

Regulations for Manufacture of Bone Meal in South Africa.

In this country, naturally, we are expected to indicate the limits of safe sterilization.

The new regulations will require the bone meal to have been sterilized

(i) by subjection to saturated steam under pressure of not less than 40 lb. per square inch maintained for a period of not less than two hours in a digester of not more than 4 tons capacity; or

(ii) by treatment of the bones, after being broken up, with the vapour of benzene boiling between 95°C and 115°C for not less than *four hours,* live steam to be thereafter admitted at a pressure of 80 lb. per square inch for two hours; or

(iii) by treatment of the bones, after being broken, up with the vapour of benzene boiling between 95°C and 115°C for *eight hours;* or

(iv) by any other treatment which may be approved in writing by the Minister of Agriculture and Forestry.

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Blowfly Strike in Sheep — A New Line of Treatment and Prevention (Preliminary Note).

By H. O. MÖNNIG, Onderstepoort.

Our present knowledge of the conditions that lead to blowfly strike in sheep can be summarised as follows:—

On the sheep there must be an area which is attractive to the pregnant female blowflies and suitable for the development of the maggots. Attractiveness has been analysed and defined by various authors and it would appear to be based generally on the following set of circumstances: The suint is hygroscopic and therefore draws water from its surroundings — moist atmosphere or wool moistened by diarrhoeic faeces or urine [Holdaway (1932), Holdaway and Mulhearn (1934), Belschner (1937), Beveridge (1935) and others] — and produces a moist area on the skin. The epithelial debris present in the moist mass of suint and wool grease decomposes, probably always under the influence of bacteria which are present. At a later stage skin exudate joins in the process; volatile decomposition products are liberated and attract flies to the spot where oviposition occurs. It is generally agreed that suint plays a very important part in this process and Holdaway and Mulhearn (1934) went so far as to correlate susceptibility of sheep with the amount of suint they produced.

Much has been written on soiling of the breech by diarrhoeic faeces and urine and on the importance of folds in the breech. These factors, however, appear mainly to aggravate or intensify the conditions set out above, in that soiling by faeces or urine provides moisture [Holdaway (1932)] and the folds provide recesses in which suint and skin debris accumulate, in addition to promoting soiling on account of their prominence. Bull (1931) and others have studied the so-called "scalding" of the skin caused by urine and diarrhoeic faeces and state that such scalded areas may directly lead to strike, but this is probably not a common cause of strike since it is found in practice that the strike occurs, as a rule, not on but immediately next to the soiled area. Holdaway (1932) states that the blowfly eggs are frequently deposited 1 to 2 inches away from the soiled area and that young maggots crawl away from a urine-soiled patch, since urine is harmful to them. In the case of body-strike or head-strike in rams, faeces and urine play no part and the process as described above operates in all cases.

A second point of great importance is that an area is attractive to blowflies and also suitable for the development of young maggots only when it is alkaline. These facts have emerged from the work of several

investigators, and Hobson (1935) has shown that alkalinity is an essential condition even when pure chemicals are tested for attractiveness to female blowflies.

The above picture of the conditions required for the production of successful strike led the present writer to consider the possibility of reducing the susceptibility of sheep and of wounds by treatment with substances which would reduce suint secretion on the healthy skin and at the same time produce an acid reaction.

The object of this note is to record briefly the results obtained by such methods in preliminary laboratory tests.

1. Skin treatments to reduce suint secretion.

A large area was closely shorn on both sides of the body of a number of sheep and in each case the right side was treated while the left was kept for control purposes. The sheep were treated by thoroughly moistening the shorn areas with 5 to 10 per cent solutions of one of the following: alum, formalin, tannic acid, salicylic acid, and copper sulphate. The treatments were repeated at intervals of two weeks in some cases and four weeks in others. After each period of four weeks the wool was clipped from each side and quantitative analyses for suint and wool grease were made. The method did not, however, give reliable results, because the substances applied to the skin were contained in the water-soluble extract, and it was therefore abandoned. The skin was not visibly affected in any of these sheep.

2. Skin reaction tests.

In order to determine the harmfulness of such applications to the skin, a number of sheep were again shorn and one of the following solutions was massaged into each:—

Wattlebark extract 12·5% and 25%.
Tannic acid 25% and 50%.
Formalin 10% and 50%.
Alum 10%.

Only the sheep treated with 50% formalin (20% formaldehyde) showed a transitory reddening of the skin which passed off within four days.

3. Protective tests.

Four sheep with long wool were now treated on the right side of the body over a large, marked-off area, by opening the wool in successive strips and moistening the skin of each with 10% solutions of one of the following: alum, formalin, tannic acid and wattlebark extract (pH — 4·7). Five weeks after this treatment the protection afforded by it was tested by means of artificial strikes, while control strikes were made on the opposite

side of the body. The strikes were made as described by Freney, Mackerras and Mackerras (1935), i.e. the wool was opened, a drop of water placed on the skin and well rubbed by means of a finger, then a good number of first stage *Lucilia cuprina* larvae were pipetted on to a moist cotton wool swab and placed on the prepared spot, over which the wool was then tied together. This method has proved to be completely reliable in a large number of strikes so far made. The results after 24 hours were as follows:—

Drug used	*Treated area*	*Control area*
Formalin	+	+
Alum	(—)	+
Tannic acid	—	—
Wattlebark extract	—	—

In the alum-treated area the larvae had grown a little before they died; protection was therefore not complete. While the various solutions were being applied to their right sides, the sheep had been lying on their left sides and some of the fluid evidently flowed across the backs and wetted the left sides also. In the case of the wattlebark extract the reddish-brown colour of this substance could clearly be seen in the wool on the left side where the control strike was made; the tannic acid probably also flowed round and for this reason the controls were negative in both cases.

Six weeks after treatment the sheep were again tested, except the formalin-treated case, and the control strikes were now made on the necks which could not have been soiled by the solutions. The results were as follows:—

Drug used	*Treated area*	*Control area*
Alum	+	+
Tannic acid	(—)	+
Wattlebark extract	—	+

In the tannic acid treated case the larvae had increased in size before they died.

Eight weeks after treatment the following result was obtained:—

Drug used	*Treated area*	*Control area*
Tannic acid	+	+
Wattlebark extract	—	+

The wattlebark extract (henceforth written W.E.) case was tested again 13 weeks after treatment. By this time the growth of new wool had lifted the stain caused by the W.E. away from the skin for about 15 mm. Two artificial strikes made in the usual way were both successful. It may therefore be that the skin itself does not remain unsuitable for strike for

a long period, but that it is protected by the extract in the wool as long as this is sufficiently close to the skin.

The test was repeated and extended by treating a patch about 20 cm. in diameter on each of the following sheep:—

Sheep No.	*Drug used*	*Site treated*
57129	W.E. 10%	Rump
57067	W.E. 10%	Withers
57029	Tannic acid 10%	Rump
56991	Tannic acid 10%	Withers
57008	Picric acid 1%	Rump
57002	Picric acid 1%	Withers
57051	Magnesium chloride 10%	Rump
57047	Magnesium chloride 10%	Withers

The control test area was the withers or the rump, whichever had not been treated in the particular sheep. No reaction to the treatment was noted in any of these cases. Artificial strike tests showed that the picric acid and the magnesium chloride gave no protection after four weeks, the tannic acid protected after four but not after six weeks and the W.E. protected after six weeks, but nine weeks after treatment a few (about 2 per cent) of the larvae applied were still alive after 24 hours and had slightly increased in size. Since new wool had grown in the nine weeks after treatment and had lifted the W.E. stain about 8 mm. away from the skin, it was decided to test whether there was anything on the skin itself to protect it, or whether protection was due to the substance in the wool, as in the case of jetting with arsenical mixtures. Over an area about 5 cm. in diameter the wool was clipped off close to the skin; in the centre of this area an artificial strike was made in the usual way and the moist swab covering the larvae was covered and surrounded with non-absorbent cotton wool, so as to keep the rest of the wool away from it. Over this the wool was then tied together. Again about 2% of the larvae were found alive and slightly larger in size after 24 hours, indicating that conditions on the skin may still have been to some degree unsuitable for strike. This matter requires further investigation.

The results obtained with the different chemicals are interesting. One might have expected that picric acid and tannic acid would readily be neutralized by the alkaline components of the suint and therefore be inactivated; moreover picric acid would tend to penetrate into the wool fibres and the skin and to become fixed there. The tannic acid, however, retained its efficacy for a fair period. If protection were due to a hardening of the skin and reduced suint secretion, one might have expected more from the formalin and alum than they were able to do. The wattlebark extract, which consists mainly of catechol tannate and which would therefore probably not be so readily inactivated by any ingredients of the suint, is strongly

acid as well as astringent and was expected to be the most effective and for the longest period.

Prophylactic treatment under natural conditions is about to be tested in the field on a few hundred susceptible sheep and will take the form of wetting the breech with W.E. solutions (10% and 20%) and noting the incidence of strike in them and in sheep that have been crutched only. This solution can be applied immediately after crutching and will promote healing of any shearing wounds present.

4. Therapeutic tests.

(a) Third stage *Lucilia cuprina* larvae were immersed in a 10 per cent. W.E. solution without any injury resulting to them.

(b) It was found that petrol and benzol could be emulsified in the 10 per cent W.E. solution without the addition of an emulsifier. The emulsions are rather coarse but they keep well. Such emulsions were tested on full-grown third stage *Lucilia cuprina* and *L. sericata* maggots as well as on natural and artificial strikes. The petrol emulsion proved to be rather ineffective in killing the maggots, but benzol was satisfactory.

In vitro tests were carried out as follows: The maggots were counted into a glass specimen tube and the emulsion was poured over them. A piece of fine-mesh wire gauze was placed over the mouth of the tube and five seconds before the time was up, as shown by a stop-watch, the tube was inverted over a sink and shaken so as to remove the fluid. The maggots were then immediately dropped on to clean sand in a jar of suitable size, so that they reached the sand at about just the end of the test period. The jars were covered with muslin and placed in a warm room. After sufficient time had been allowed for pupation and emergence of the flies and the latter had all died, the contents of each jar were sieved and the dead larvae, dead pupae, and dead flies were counted. The following table gives the results of such tests with emulsions of different concentrations:—

Date.	*Emulsion.*	*Immersion Time (min.)*	*Dead larvae.*	*pupae.*	*flies.*	*Total.*
1.8.40	10% W.E. 50, Petrol 50	1	5	4	16	25
,,	,, ...	2	5	7	13	,,
,,	,, ...	3	8	5	12	,,
,,	10% W.E. 70, Petrol 30	1	0	0	25	,,
,,	,, ...	2	0	0	25	,,
,,	,, ...	3	1	3	21	,,
,,	5% W.E. 50, Petrol 50	1	11	5	9	,,
2.8.40	,, ...	2	7	4	14	,,
,,	,, ...	3	7	2	16	,,
,,	5% W.E. 70, Petrol 30	1	0	2	23	,,
,,	,, ...	2	0	4	21	,,
,,	,, ...	3	0	4	21	,,

Date.	*Emulsion*	*Immersion Time (min.)*	*Dead larvae.*	*pupae.*	*flies.*	*Total.*
7.8.40	10% W.E. 50, Benzol 50	1	39	1	0	40
,,	,, ...	2	38	2	0	,,
,,	,, ...	3	40	0	0	40
,,	5% W.E. 50, Benzol 50	1	37	3	0	,,
,,	,, ...	2	40	0	0	,,
,,	,, ...	3	40	0	0	,,
8.8.40	10% W.E. 70, Benzol 30	1	14	26	0	,,
,,	,, ...	2	21	19	0	,,
,,	,, ...	3	35	5	0	,,
,,	5% W.E. 70, Benzol 30	1	13	27	0	,,
,,	,, ...	2	32	8	0	,,
,,	,, ...	3	30	10	0	,,
21.8.40	20% W.E. 50, Benzol 50	1	50	0	0	50
,,	,, ...	2	50	0	0	,,
,,	,, ...	3	50	0	0	,,
,,	20% W.E. 70, Benzol 30	1	49	1	0	,,
,,	,, ...	2	50	0	0	50
,,	,, ...	3	50	0	0	,,

A small number of sheep with natural strikes were treated with the benzol emulsion and the wounds healed rapidly under a thin, dry scab, without any re-strikes occurring. These tests were, however, made during the winter when strikes did not occur frequently. Some of the sheep showed slight uneasiness immediately after treatment, but this passed off quickly and was probably due to the cold sensation produced by the evaporation of the benzol. In cases treated with benzol only the same uneasiness was observed.

At a later stage of the work it was found that the emulsion corrodes metal and turns dark blue when kept in a tin. Apparently the tannates combine with the metal to form an ink and this would reduce the efficacy of the mixture. It was accordingly decided to alter the procedure of treatment as follows: The struck area was first sprinkled with benzol in order to kill the maggots and then the wool clipped off over and around it. This has the advantages that the benzol is used in a concentrated form and kills the maggots rapidly, it can be used sparingly and no maggots escape when the wool is clipped off. Then the affected part is moistened with a liberal quantity of the W.E. solution. (Benzol costs about 3s. 3d. per gallon in the Union and a gallon of 20 per cent W.E. would cost 4d.). If necessary the W.E. solution could be made more concentrated and if it should be found that the crust formed is too brittle, an emollient could be added to the solution. At a later stage 5 per cent gum acacia was added for this purpose. These aspects require further investigation. * The W.E. could also be used for prophylactic treatment of wounds, e.g. after cutting lambs'

* Promising results were later obtained with 30 per cent W.E. and 5 per cent boric acid solution.

tails when bleeding has stopped, and it would probably assist materially in stopping haemorrhage on account of its strong astringent action.

Large scale field tests are about to be carried out to test the efficacy of this treatment on screw-worm in cattle as well.

Artificial strikes were prepared to test the treatment. A difficulty was experienced in finding a suitable method of making re-strikes. The W.E. coagulates the protein exudate on the wound, producing a thin crust which dries very rapidly. The dryness of the wound alone would tend to make it unsuitable for re-strike, apart from the presence of the W.E. which also deters the larvae from attacking. If the larvae are applied with moisture the test is therefore not quite fair. If the wound is rubbed before application of the larvae the crust is broken and exposes the raw surface. Some re-strikes were made by moistening the crust, without rubbing, then applying the larvae by pipetting them on to the spot in a small quantity of water and then tying the wool over without a cotton wool swab to cover the larvae. Two re-strikes made on wounds treated with benzol only were positive; of seven re-strikes made on wounds treated with 10 per cent W.E. and benzol three were positive and four negative. Probably too much water was used on the wounds, because they were swamped and water flowed off, tending to wash away some of the W.E. which should protect the wounds. Further re-strikes were made by pipetting the larvae on to a small moist swab and placing this on the untouched wound, then tying the wool over it. Of eleven re-strikes made on wounds treated with benzol only, all were positive, while all of 13 re-strikes made on wounds treated with 10 per cent W.E. and benzol emulsion were negative, as well as all of 12 re-strikes on wounds treated, first with benzol and then with 20 per cent W.E.

In making artificial strikes for these tests about 200 to 300 larvae were placed on the skin in each case and left there for two to three days before treatment was applied. Re-strike tests were made 2 to 7 days after treatment. It was not considered necessary to test after a longer period, because after 7 days the wounds had healed so well that re-strike under natural conditions would be highly improbable. The real test of this treatment will, however, have to be carried out under natural conditions of strike.

In the case of maggot infected wounds which have produced a large amount of exudate the latter, which is strongly alkaline and contains some iron pigments, neutralises a part of the W.E. solution and also produces a dark-blue colour in the wool. In order that a sufficient quantity of W.E. should remain to protect the wound, it was decided to use a 20 per cent solution instead of the 10 per cent used at the beginning. It may even be desirable to increase the concentration still further. The blue colour in the wool was found to wash out satisfactorily in the ordinary washing process.

(c) *Healing of wounds and their unsuitability to re-strike.* Several authors, writing on the role of maggots in the treatment of wounds in man [Baer (1931), Messer and McClellan (1935), Robinson and Baker (1939) and others] have pointed out that the maggots produce a strong alkaline reaction in the wound which promotes healing. Robinson and others have accordingly recommended that slow-healing wounds should be treated with ammonium carbonate in order to produce an alkaline reaction and so to speed up the healing process.

According to this information an alkaline wound, which is attractive to blowflies and suitable for re-strike, is a fast-healing wound, while one made acid in reaction by treatment and therefore less attractive and unsuitable for re-strike would heal slowly. Laake and Smith (1938), however, found that although the wounds are strongly alkaline while the maggots are present the reaction swings to the acid side as soon as the maggots have left and then healing occurs. They conclude that slow-healing wounds are alkaline, while normally healing wounds are acid and that wounds should be made acid to promote healing. It is possible that a change from alkaline to acid reaction, or vice versa, may stimulate healing, depending on the reaction of a wound to start with. In regard to maggot-infected wounds the writer was able to confirm the findings of Laake and Smith and found that after the removal of the maggots, wounds kept acid healed more rapidly than those kept alkaline by treatment.

The wattlebark extract appears to have a very good healing effect on wounds and it may be useful in the treatment of ordinary wounds and abrasions which are attacked by flies.

While investigating the possibilities of *Tagetes* oil as a repellent the writer [Mönnig (1936)] eventually came to the conclusion that it would be far better to get the wounds to dry and heal rapidly and also to make them unattractive and unsuitable for re-strike in other ways than by means of repellents. Tagetes oil appears to be the strongest blowfly repellent known to-day, as tests made according to Hobson's (1937) method indicated, but even this substance was unable to prevent re-strike during a bad wave of blowfly attack.

It is probable that field tests will show that further modifications of the indicated treatments are required and better substances than wattlebark extract may have to be found before a completely satisfactory method is attained, but the line of treatment indicated in this paper is certainly worth further intensive investigation, as it is founded on basic principles and promises to give useful results.

SUMMARY.

The use of wattlebark extract for preventive and therapeutic treatment of blowfly strike in sheep is described. The method is based on our knowledge of the factors which cause attractiveness and suitability of sheep to strike.

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Destruction of Meercats by means of Exhaust Gases from a Car Engine.

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According to information gathered from farmers meercats * can be destroyed if the colonies are fumigated with the exhaust gases from an internal combustion engine. As several outbreaks of rabies had occurred on the Hoopstad Commonage the local Municipality decided to use this method to destroy the vectors of the disease in the immediate vicinity of the village.

The procedure adopted was in principle the same as described by Thomas (1936) and Snyman and Thomas (1938) for fumigating the colonies with cyanide dust by means of hand-operated pumps. The latter authors also describe the intricate system of burrows which constitute a colony and which may have an important bearing on the efficacy of gassing.

The colonies were either gassed early in the morning before the meercats had left them or later in the day, when the meercats were driven into the colonies by people walking in formation over the veld. An old six cylinder motor car, which produced blue smoky exhaust fumes, was used for fumigation. The exhaust pipe of the motor car was connected with one of the entrances of the colony by an old petrol bowser hose-pipe. The colonies were gassed with the engine running at a speed corresponding to about fifty miles per hour on top gear.

By racing the engine in this way and using a connection with a fairly wide inside diameter (about 1½ in.) it was found that the gasses were forced through the underground passages very rapidly. The exhaust fumes, which were, on account of the smoky appearance, always easily visible, appeared from the entrances so rapidly that six to ten natives could barely keep pace closing these with spades. Only very seldom was it necessary to fumigate a colony for a period longer than two minutes or to fumigate from more than one entrance.

The object of forcing the gas as rapidly as possible through the colony was to prevent the occupants from entering any blind burrows which sometimes exist. It is assumed that the meercats, unless unduly disturbed, do not readily enter these blind tunnels, where the gas cannot reach them. In one instance the gas was forced out, within less than a minute, at an entrance ninety feet from the opening where it was being introduced.

After fumigation, a few colonies were excavated while others were observed for a day or two. Some of the results obtained are given in tabular form below.

* Meercats referred to are *Cynictis penicillata, Suricata suricatta,* and *Geosciurus capensis.*

Description of Colony	Treatment.	Results.	Remarks.
15 holes	5.2.40 gassed for about two min. from one hole and excavated	5 dead. *Cynictis* found.	5 *Cynictis* seen to enter
17 x 20 yd. 73 entrances	5.2.40 gassed for about 5 min. from one entrance	6.2.40 closed 7.2.40 ,, 11.2.40 ,,	one *Cynictis* seen to enter
14 yd. in diameter	5.2.40 gassed for one min. from one entrance	6.2.40 closed 7.2.40 ,, 11.2.40 ,,	two *Cynictis* seen to enter
12 yd. in diameter	5.2.40 gassed for one min. from one hole	6.2.40 closed 7.2.40 ,, 11.2.40 ,,	one *Cynictis* seen to enter
4 big colonies in "trassie" bushes. (*Acacia stolonifera*).	5.2.40 all, except one partly dilapidated colony, gassed from one hole	6.2.40 closed 7.2.40 ,, 11.2.40 ,, and odour of decomposing carcass material from 2 colonies	were inhabited
28 x 10 yd. with 20 entrances	6.2.40 gassed for one min. from one entrance and excavated	5 dead. *Cynictis* found.	5 *Cynictis* seen to enter
small colony with 6 entrances	6.2.40 gassed and excavated	1 dead *Cynictis* found.	one *Cynictis* seen to enter
colony in "trassie" bush. 14 x 10 yd. 40 entrances	6.2.40 gassed 1½ min. from 1 hole	7.2.40 closed after 24 hrs.	definitely inhabited but no meercats were seen to enter
Colony in a "trassie" bush. 14 x 9 yd. 53 entrances	6.2.40 gassed for 2 min. from one entrance	7.2.40 closed after 24 hrs.	inhabited; no meercats were seen to enter
30 x 25 yd. 35 holes	6.2.40 gassed for 2 min. from one hole	7.2.40 3 entrances opened. Later marked odour of decomposing carcass material from this colony	inhabited by *Cynictis* and squirrels. Apparently opened by squirrels from nearby colony not gased
10 yd. in diameter with many entrances	7.2.40 gassed from one hole	9.2.40 still closed; putrid odour when opened	about 20 *suricata* were seen to enter
two separate warrens with 2 entrances each	each gassed for a few secs. and dug up	found a dead *Cynictis* in one warren and a dead *Geosciurus* in the other	the dead animals found were seen to enter
warren with 2 entrances 15 feet apart	gassed for a few secs. and dug up	2 dead young jackals found	jackal were suspected to inhabit the warren

The above results are not from colonies specially selected but from those situated more or less in the centre of the area where the meercats were exterminated. Other colonies situated towards the boundary of the area of operations and which were thus more liable to be opened by migrating meercats are not included.

Although suffocation may have played a part, the carbon monoxide contained in the exhaust gasses is thought to be the toxic principle responsible for the death of the animals. In other countries carbon monoxide has been used in various ways, on account of its efficacy and low cost of production, for the destruction of vermin. It is calculated that about one pint of petrol was used for the fumigation of an average sized colony. The cost of petrol consumed was therefore about 3d. per colony.

The advantages of using an old car are its low initial cost, general availability, easy operation, requirement of little extra equipment, and the fact that the unit is self-transported. More importance is attached, however, to its distinct advantage over the manually operated pump in rapidly delivering a large volume of gas.

The results from the few colonies excavated show that the fumigation killed all the occupants. The inhabitants of the other colonies were probably also exterminated because hardly any colonies were reopened within the next couple of days and because of the odour of decomposing carcass material detected in many.

It would appear therefore that the method employed is as effective as fumigation with cyanogas and the hand-pumps available for the purpose. The results warrant further investigation on a bigger scale, and such investigations should consider the economic aspects of the problem.

Conclusions.

(1) The use of the exhaust fumes from an internal combustion engine to destroy meercats is described.

(2) The results show that the method is effective for the purpose.

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Tuberculosis in a Turkey.

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This case was considered worth recording because tuberculosis of turkeys is not common in Natal. As far as I know, no case has previously been reported from this province.

Continental authorities seem agreed that turkeys are not commonly affected with tuberculosis. Eber (1897) at Leipzig, found only ten cases among 257 turkeys examined during a period of 24 years. Klimmer (1930) noted the disease in 11% of 56 turkeys examined during 22 years. Proscholdt obtained positive reactions to tuberculin in 18% of 127 turkeys. On the other hand it appears that tuberculosis of turkeys is fairly prevalent in the United States. Hinshaw, Niemann and Busic (1932) reported that of 88 turkeys obtained from four different farms in California and Nevada, 45 were infected with tuberculosis.

In September 1939, a farmer obtained five young turkeys and one adult from the Newcastle district. Of these two died, two were sold to the butcher, and one remained on the farm in the Klip River district. The last, a sick bird was sent in a very emaciated condition to Allerton Laboratory in December 1939. The accompanying letter stated that the liver was possibly the site of the disease and that turkeys as well as fowls had died from the same condition since November 1939. The symptoms were lameness progressing to partial paralysis, and persistent diarrhoea.

The bird was killed and at post mortem lesions were observed on the parietal peritoneum lining the sternum. These lesions consisted of three raised nodules the size of small peas. The liver, although normal in size and colour, was speckled with minute, pin-point, whitish foci, which were distributed throughout the parenchyma. Two small greyish-white nodules, about one mm. in diameter, were located on the anterior surface of the liver. They projected above the surface and so gave the liver a somewhat bosselated appearance. The spleen was not enlarged, but showed two whitish lesions roughly 2 mm. in diameter. Four lesions in the lungs were irregularly spherical discrete nodules sharply demarcated from the normal pulmonary tissue. Both surfaces of the mesentery were studded with numerous small oval and elongated nodules, arranged in chain-like fashion. The most prominent lesion was in the gizzard. Its muscular wall bulged on one side. On section, the greater part of the wall was found to be replaced by a hard, dry, caseous mass that projected into the cavity of the gizzard. The duodenum, the rest of the small intestine, and the caecum all showed nodular lesions throughout. The remaining organs of

the body showed no changes. Smears made from lesions in the various organs showed acid-fast organisms.

It would be of interest to know whether the disease originated in the fowls and spread to the turkeys, or whether the turkeys brought the infection to the farm.

SUMMARY.

An account has been given of the symptoms and post-mortem appearances found in a turkey. The lesions contained acid-fast bacteria. It is highly probable that the bird was suffering from avian tuberculosis.

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The Bleeding of Pigeons.

By D. A. HAIG, Allerton Laboratory, Pietermaritzburg.

As a result of an outbreak of *S.typhimurium* in a loft of about 150 valuable pigeons, it was decided to eliminate carriers of the infection by means of the agglutination test.

Pigeon fanciers are generally rather fastidious about the way their birds are handled, and consequently a technique for the abstraction of blood had to be evolved which would not injure the birds and which would obviate the soiling of their plumage with blood after the operation was completed. The object of this note is to describe how these difficulties were overcome, and to indicate a new site for the easy bleeding of pigeons.

Equipment.

(a) Sterilized glass tubes, 8 cm. x 8mm., corked at both ends and containing 0·5 c.c. of 5% boracic acid solution, which acts as a preservative and anti-haemolytic agent.

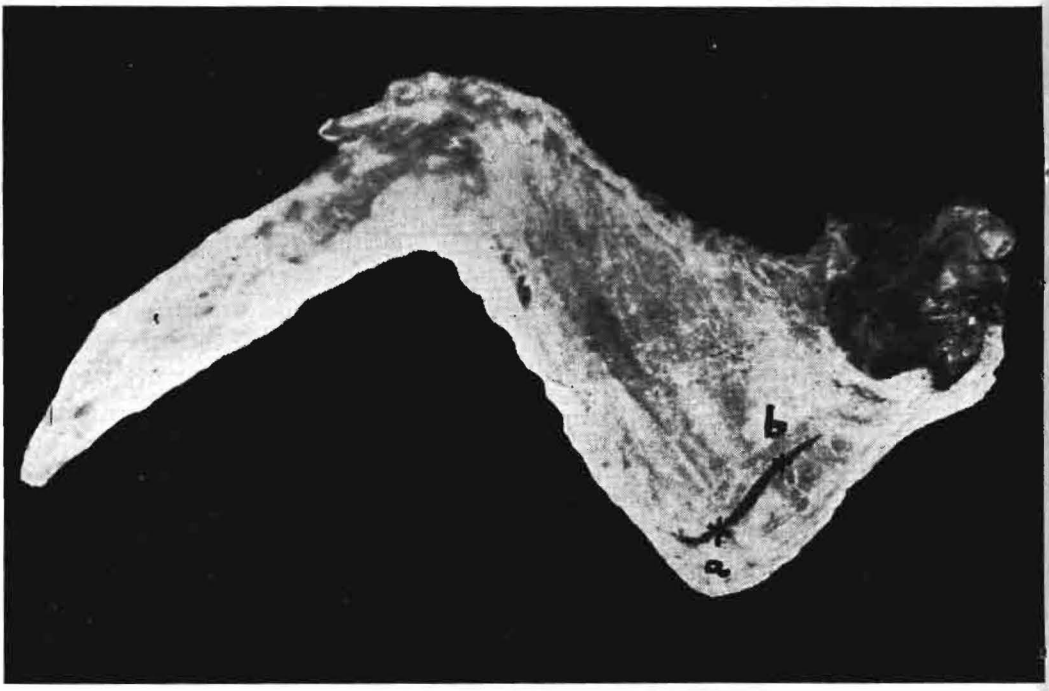
(b) A straight, lance-pointed surgical needle 6·3 cm. long. A fine hypodermic needle was found quite satisfactory for the actual puncture of the vein, but could not be quickly cleaned between each bleeding. It was therefore discarded, as some of the birds were possibly in the incubation period of the disease, and also to save time.

(c) Some swabs of cotton wool steeped in alcohol.

(d) Incubator, running at 37°C.

Control.

The bird is caught by an assistant, with as little disturbance as possible, and held in the dorsal recumbent position. The tail, both legs and left wing are held by the assistant's left hand. With his right hand the



assistant controls the head and body, his thumb being placed over the neck. The operator then extends the right wing and grasps its anterior margin in the hollow of the left hand.

No attempt is made to disinfect the site, but as much skin debris as possible is blown off. Dirt is removed with cotton wool soaked in alcohol and the skin is wiped dry before the bird is bled.

Operation.

The needle is held between the thumb and first two fingers, and is inserted about one cm. into and along the vein. The skin is pierced at *a* (v. photograph). The needle is then raised at an angle of about 25° to the axis of the humerus, in order to enlarge the opening in the skin, and is then withdrawn. Bleeding is usually fairly rapid, but stops when the bird is released. The average rate of bleeding is 40 birds per hour. With fancy birds which are kept continuously confined, bleeding is often slow and clotting of the blood may give trouble. In these cases site *b* (see photograph) is chosen. When this site is used, the flow of blood is frequently rapid. There is a risk of puncturing the accompanying artery if the needle is inserted too far forwards at *b*. When this occurs haemorrhage is usually marked and the owner becomes very alarmed. There is the added possibility of the nerves being damaged. No bad effects have, however, been noticed where the artery has been punctured, and no cases of injury to the nerve have been seen.

When the vein is opened a second assistant hands the operator a tube uncorked at one end, and in this about 1 cc. of blood is collected. The tube is then shaken gently in order to mix the blood and the boracic solution, and finally the tube is filled with blood and is then recorked and returned to the box. It is not necessary to control the bleeding after sufficient blood has been taken, as the flow from the puncture stops as soon as the birds are allowed free movement.

After each lot of 10 birds, the tubes of blood are placed in an incubator running at 37°C. These samples are examined in four hours' time and, in each case where the serum is slow in being expressed, the cork to which the clot is attached is gently twisted, in order to detach the solid blood from the sides of the tube. This invariably has the desired effect. When sufficient serum has been obtained, the tubes are placed in an ice box.

Pigeon blood haemolyses easily and the clot is apparently very fragile. This is especially the case in samples where the flow of blood from the punctured vessel has been slow. If samples have to be sent any distance by post, it is advisable to decant the serum into fresh empty tubes before dispatch.

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Thelazia Rhodesi as a primary cause of Opthalmia in Cattle in Nyasaland.

By S. G. WILSON, B.Sc. (Belfast), B.Sc. (Vet. Sc. Ed.), M.R.C.V.S.,
Provincial Veterinary Officer, Nyasaland.

Ophthalmia, varying from simple conjunctivitis to ulceration of the cornea and suppuration of the eye ball, is relatively common in European herds in Southern Province, and often results in a serious loss of efficiency in the herds. The incidence shows a marked decrease northwards, i.e. proceeding from European to native areas of settlement. Karonga in the extreme north of the Protectorate is a possible exception to this rule, as here the incidence is relatively high for a native area.

The cause (or causes) of this disease is often obscure and treatment is therefore usually empirical. In bush grazing, with tall coarse grasses predominating, eye injuries will be of greater frequency than in more settled countries. There is no doubt that bacteria are implicated in many cases but these infections are most likely of a secondary nature. Coles (personal communication) drew attention to the role of *Rickettsia* in ophthalmia, but conjuctival smears of cases in Nyasaland have so far proved negative.

The first records of the occurrence of the nematode *Thelezia* in Nyasaland were probably those of Gardener (1911). He claimed that these worms were one of the most common causes of ophthalmia then prevalent amongst cattle in the Shire Highlands (S. Province). Griffiths (1922) summarizing the literature on the family *Thelaziidae*, drew attention to the importance from a veterinary point of view of the genera *Thelazia* and *Oxyspirura*. The normal habitat of *Thelazia* is in the lachrymal ducts or under the membrana nictitans. From here, they may wander freely over the eyeball. They are therefore extra-ocular and must be distinguished from the blood filaria larvae often found in the anterior and posterior chambers of the eye. Summarizing the clinical signs shown by a herd infested with *Thelazia*, Griffiths states " One or more of the animals shows signs of photophobia with profuse lachrymation. On examination there may be any degree of corneal opacity from slight cloudiness to complete opacity." Later, suppurative organisms and liability of the eye to injuries due to immobility of the swollen membrana nictitans, play their role in the pathological changes seen. Venmataratnam Chetty (1922) records an outbreak of *T. rhodesi* in Indian draught cattle where over 40 per cent of the affected beasts lost their sight. The pathological changes varied from simple ophthalmia to ulcerative keratitis of one or both eyes. Ramaswamy Iyer (1922) records a similar case of infection in a cow. Both eyes eventually became involved and the irritation

of the *Thelazia* was sufficient to cause severe conjunctivitis with profuse lacrymation leading to opacity of the cornea.

In the Mzimba district of Nyasaland, the Veterinary Department has been very active for over three years in its efforts to improve animal husbandry and establish a native ghee industry. Veterinary supervision has been effective and continuous and our knowledge of indigenous diseases in native cattle has increased considerably. Stock farms have been established and cattle herds kept under close supervision. At no time during these years has acute ophthalmia been observed. Cloudiness and opacity of the cornea is occasionally observed in old animals and even these cases are relatively rare and in a recent census over a wide area, amounted to less than 0·1%. Yet the prevalence of *T. rhodesi* in the eye and lacrymal ducts of cattle is widespread.

It is difficult to examine thoroughly a native herd to establish percentage infection, as the cattle become frantic on restraint. A cursory examination of the eye is not always sufficient, for although *T. rhodesi* are often seen wandering free over the eyeball, their normal habitat is in the deeper parts of the eye. But when opportunity has permitted, such as at slaughter houses or when inoculating intravenously for tyrpanosomiasis, close examination seldom fails to reveal *Thelazia* under the membrana nictitans. In this way their presence has been recorded from almost every part of the district.

The Zombwe Stock Farm herd may be quoted as an example where close study has been possible. An average daily inspection of the 44 head of cattle usually reveals 4-5 beasts with *Thelazia* or 10 per cent approx. infected. Without artificially inducing lachrymation by dressing, 5-8 worms were recovered in many cases. No eye lesions were noted. A point being investigated is the periodicity of these worms, as during the dry season (i.e. when flies are scarce) infections are much heavier than during the rains in December-April.

Discussion.

From a survey of the literature quoted above the fact that *T. rhodesi* may cause an acute conjunctivitis in cattle has been adequately proved by observers both in Nyasaland and in other parts of Africa and India. The experience of the writer, however, in native herds in Northern Province of Nyasaland, and especially in Mzimba district, definitely shows that a *Thelazia* infection does not invariably give rise to clinical signs nor to pathological eye lesions. Should an epidemic of conjunctivitis break out and the present widespread prevalence of *Thelazia* not be noted, the *Thelazia* might all too easily be diagnosed as the primary cause of disease. The liability would increase when the known pathogenicity of the related *Oxyspirura* in the eyes of fowls was considered, and a natural corolary drawn.

The susceptibility of European cattle is no doubt different, but when dealing with purely indigenous native herds, all other causes for ophthalmia should first be eliminated. Then and only then should *T. rhodesi* be incriminated.

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OBITUARIES.

Charles Tyler.

We regret to announce the death of another link with the pioneers of the veterinary profession in South Africa. Charles Tyler was born in Burslem, Staffordshire, in 1877. He qualified at the Royal Veterinary College, London, in 1900 and came to South Africa soon after.

He was in Vryheid at the time of the Bambata Rebellion, 1906, and saw service with the Northern Districts Mounted Rifles. Subsequently Tyler moved to Mooi River. Here he interested himself in the gymkhana club and was instrumental in organizing several gymkhanas. Later, when stationed at Ladysmith, he became President of the gymkhana club and a foundation member of the golf club. In 1913 he was transferred to Port Shepstone and in 1930 to the Cape. On his retirement in 1932 he went to Bournemouth. In 1937 he returned to Natal with the intention of seeing out his retirement here.

Tyler was a fine rider and keenly interested in horses. In his work he proved an excellent and popular administrative officer. He was a most popular clubman, friendly and versatile, and excelled as an after-dinner speaker in times when that art was appreciated.

Theodor Heinrich Sandrock.

It is with a deep sense of personal sorrow that I have to write these few words of appreciation of a true friend and colleague, so soon after the death of another class-mate.

Theodor Heinrich Sandrock was born on October 9, 1908, at Padang, in Sumatra, Netherlands East Indies. From 1920 to 1924 he went to school in Holland. The following two years he spent in Batavia and then returned to Holland in order to study medicine in Amsterdam. There he took a prominent part in student activities and became a member of the Students' Representative Council.

He relinquished his medical studies in 1933 in order to take the veterinary course at Onderstepoort and it is not surprising that his wide interests and continental outlook, his vivid personality and his apt criticisms had a guiding and even formative influence on junior and senior fellow students.

He graduated in 1937 and was first appointed as Government Veterinary Officer at Dundee. Later he was transferred to Vryburg. In 1938 he married Miss M. S. Forssman. Unfortunately this happy union was not to last long. During the past year his health left much to be desired and last August he was operated on for appendicitis. The long delay, however, had resulted in serious complications and, notwithstanding a courageous struggle, he died on September the 27th, 1940.

Those who knew him cannot recall his memory without a warm feeling of respect and appreciation. One could not know him without being impressed by his personality. His fine intellect and his wide, varied and lively interest in all aspects of living stimulated others to a higher cultural outlook. His practical idealism manifested itself in his daily life. In his studies and in his work that idealism knew no bounds.

His family and friends, the veterinary profession, and this country of his adoption are the poorer by his death.

To his relatives and to his wife we offer our deepest sympathy.

A. de B.

Arthur Edward Lund.

It is with profound regret that we record the death of a colleague, A. E. Lund. Arthur Edward Lund affectionately known to his colleagues and a wide circle of friends as Steve died suddenly at his home at Onderstepoort on the morning of November 5th, 1940. In deference to an expressed wish he was buried on his late father's farm at Winterton, Natal.

Born on 21st February, 1903, Steve matriculated at Hilton College, Natal, in 1921 and joined the second group of students to be enrolled at the Transvaal University College to study veterinary science. As a student he will always be remembered by his fellows. Possessed of a ready wit, a keen sense of humour, and an argumentative disposition his company could not be other than entertaining. His cheerful personality, coupled with sporting ability well above the average, combined to make him an outstanding student. He was a keen cricketer and tennis player but shone on the rugby field where he will be remembered for his consistent displays in the T.U.C. XV as scrum half from 1923-25.

After qualifying he was appointed a Government Veterinary Officer, being stationed successively in Durban, Cape Town, Porterville Road and Potchefstroom. In 1928 he married Miss "Babs" Thompson of Natal and proceeded on transfer to Pietersburg where he remained for some five years. His popularity followed him to this district and records show that he was a prominent competitor in many gymkhana meetings in the area. His ability and sound judgment received early recognition so that in 1936 he was brought to Pretoria to act

in the capacity of Senior Veterinary Officer, Transvaal. In 1938 he was transferred to Onderstepoort and appointed lecturer in animal management in the Faculty of Veterinary Science. His great love of animals, particularly horses, made him eminently fitted for this post and the students who passed through his hands will always owe him a debt of gratitude for the capable manner in which he taught them to handle a horse, both in and out of the saddle.

His sudden death at so early a stage in a promising career came as a great shock. Many of us can ill afford to lose so staunch and loyal a friend, but in addition there now exists a gap in our profession which it will be exceedingly difficult to fill. We extend to his wife and two sons our heartfelt sympathy, and trust that a knowledge of the esteem in which Steve was held will in some measure recompense them for their irreparable loss. We in turn shall treasure the memory of a stouthearted gentleman. R. A. A.

Dr. J. H. L Cloete.

The untimely death on the 7th November, 1940, of Johan Hendrik Lorrimer Cloete at the early age of 30 has robbed the profession of one of its most brilliant younger members, at the moment when his promising career was entering on a period of outstanding success.

Cloete was a distinguished student from his early days, as the following outstanding academic record shows:—1926 — Placed second in South Africa in the Higher Taalbond Examination. 1927 — First-class Matriculation and Reyersbach Memorial Prize for the best science pass in the Union. 1932 — B.V.Sc., after an excellent all-round study record at Onderstepoort. 1936 — Admitted to Membership of the Royal Sanitary Institute. He qualified for this while leading the busy life of a District Veterinary Officer. 1937 — Selected from among an extensive list of candidates for the post of Lecturer on Veterinary Anatomy in the Faculty of Veterinary Science at Onderstepoort, where he undertook the teaching of both comparative anatomy and embryology. 1939 — D.V.Sc., University of South Africa, for his thesis "Prenatal growth of the Merino Sheep." This extensive and scholarly contribution won high praise from the Board of Examiners, including that great authority Dr. John Hammond of Cambridge. It represented the results of two years of scrupulously accurate observations, as interpreted by an intellect distinguished for its precision and mathematical ability. In the same year, he was awarded the Junior Gold Medal of the British Association for the best short paper on a scientific subject. Here he was in competition with younger workers in all fields of pure and applied sciences, so that his success brought honour to the profession as well as to himself. His essay, "The estimation of the Age of Merino Foetuses," was a summarised version of that portion of his thesis which dealt with the foetus itself and especially with practical and accurate methods of determining foetal age in sheep. The adjudicator on this paper reported, *inter alia*, that the "simple procedure made available by the author's investigation will enable embryological workers to age ovine foetuses virtually at a glance and with a maximum error of three days, a very great advance indeed on our previous inaccurate and comparatively crude knowledge of the ageing of this and related species..... This is the first time that thoroughgoing and modern statistical methods have been applied to the ageing of ovine embryos. It is rare to find statistical methods of such a high order applied to biological problems of this nature, especially when they are concerned with domesticated species...." Members will look forward to reading more of his

scientific papers, which are of equal merit to this one and will be published posthumously.

Cloete commenced his veterinary career in the field, where he had four years experience, chiefly in the Rustenburg and Ermelo areas. After three further years spent at Onderstepoort he resigned from Government Service at the beginning of this year in order to take up a part-time appointment as Veterinary Officer to the Municipalities of Springs and Brakpan and to conduct private practice at Springs. He had scarcely settled in his new life when he was offered the post of Acting Municipal Veterinary Officer, Pretoria, for the duration of the war and to replace Capt. I. P. Marais, who is on active service. Again he was just beginning with conspicuous success to settle down to this work for which he was so well qualified and talented, when after a short illness, from which he was already thought to be convalescent, he died.

Cloete's was a distinctive personality. His penetrating judgment and his dislike of the slipshod was as much in evidence in his social life as in his scientific activities. He saw through people as well as problems. He detested humbug and usually his direct honesty compelled him to expose it. In all that he planned or accomplished he was satisfied with nothing that was not first-rate. He was often irked by the restrictions which the scientific worker experiences as a public servant; not insubmissive to intelligent discipline, but resentful of inefficient "red-tape" and unintelligent conservatism. His courageous character and orderly mentality made him — not merely among the younger men — about the finest public speaker in the profession. This statement may come as a surprise to those who had not heard him address an audience, since he was of a modest nature and by inclination somewhat retiring.

He served the Association ably and willingly for the last couple of years of his life as a member of the Editorial Committee of this Journal and had assumed the editorship and been elected to the Council just before his death.

Honesty, good humour, dependability, competence — that was Cloete. It may well be a long time before a young man of talents comparable with his arises to fill his place in the profession.

To Mrs. Cloete and her two young children we extend our deepest sympathy.

C. J.

THE ASSOCIATION.

Council Meeting held at Polley's Hotel, Pretoria, 12th November, 1940.

Present. — H. H. Curson (Chairman), D. G. Steyn, A. S. Canham, J. L. Dickson, P. J. du Toit, R. Alexander, A. C. Kirkpatrick, A. D. Thomas and S. W. J. van Rensburg (Hon. Sec.-Treas.).

Apology for Absence. — Mr. S. T. Amos.

(1) *Minutes of Meeting* held on 11th April 1940, having been circulated were taken as read and were confirmed.

(2) *Arising from these minutes.* —

(a) *International Veterinary Conference.* — The Secretary reported that before Finance Committee could complete the necessary arrangements for trans-

mitting the £50 voted by the last meeting to Prof. de Blieck, Holland was invaded and the money was therefore not sent. Dr. du Toit stated that a letter sent by him to Prof. de Blieck about this matter had been returned to him by the post office. It was decided to leave this question in abeyance during the war.

(b) *Expert Witness Fees.* — The opinion of members was that it would be futile to make further representations during the war period and it was agreed not to take further action at this stage.

(c) *Veterinary Faculty.* — The meeting was informed of the representations made, and as the matter was still the subject of negotiations between the Department of Agriculture and the Council of the University it was decided to let it stand over pending receipt of further information from the Secretary for Agriculture.

(3) *New Members.* — The following new members were proposed and it was unanimously decided to recommend their admission to full membership of the Association to the General Meeting: A. A. L. Albertyn, B.V.Sc.; J. Y. Bogue, M.R.C.V.S.; P. H. Brown, B.V.Sc.; P. P. Hugo, B.V.Sc.; W. H. G. Schatz, B.V.Sc.; S. G. Turner, B.V.Sc.; S. B. Woollatt, F.R.C.V.S.

(4) *Arrears.* — In the case of three members it was decided to advise them by registered post that failing payment of arrear subscriptions before the next General Meeting that meeting will be asked to sanction removal of their names from the list of members.

(5) *Benevolent Fund.* — The Secretary stated that Dr. de Kock had recently brought to his notice the plight of the widow of a late member and had urged that assistance be granted to her. The President had also investigated this case and had telegraphed that afternoon recommending immediate and substantial aid. It was decided to grant her £2 p.m. for the next 12 months and to review the position at the end of that period.

Dr. Thomas suggested that a small standing Committee be formed whose duty it will be to try and keep in touch with the dependants of deceased members, and after discussion it was resolved that this be regarded as one of the functions of the Finance Committee.

(6) *Application for Loan.* — The Secretary submitted an application from a student, who through unforseen circumstances was deprived of the financial means for continuing his studies, asking for a loan of £150 to be paid to him in three annual instalments and to be repaid over a period of three years after he had qualified. The application was supported by recommendations from the Dean and five members of the Veterinary Faculty, three of whom (Drs. Graf, Jackson and Thomas) were prepared to guarantee the loan.

After discussion it was agreed to grant the application under conditions to be drawn up by the Finance Committee.

(7) *Sale of Poisons.* — Dr. Steyn read the report of the Committee consisting of himself, Drs. Monnig, Parkin, Thomas, and Mr. Clark. It was decided to recommend to the General Meeting that this report be forwarded to the Heads of the Departments of Agriculture, Public Health, Native Affairs, and Justice and to the Commissioner of Police.

(8) *Abattoir Control.* — A resolution by the Natal Branch was discussed and referred to the General Meeting for further consideration.

The meeting adjourned at 10.15 p.m.

S. W. J. van Rensburg,
HON. SEC.-TREAS., S.A.V.M.A.

35th General Meeting held at Onderstepoort, 13th November, 1940.

Present. — H. H. Curson (Chairman), P. S. Snyman, J. I. Quin, H. O. Monnig, D. G. Steyn, J. R. Scheuber, C. J. van Heerden, R. Alexander, P. J. J. Fourie, H. Graf, R. du Toit, H. P. Steyn, J. Quinlan, J. H. R. Bisschop, H. Theiler, A. S. Canham, E. M. Robinson, M. Sterne, J. Nicol, A. C. Kirkpatrick, D. Coles, J. L. Dickson, A. D. Thomas, P. J. du Toit, G. de Kock, J. J. G. Keppel, M. M. Neser, A. M. Diesel, B. S. Parkin and S. W. J. van Rensburg (Hon. Sec.-Treas.).

Apologies for Absence. — Messrs. S. T. Amos and L. W. Rossiter.

Obituary. — The Chairman referred to the great loss the Association had sustained through the death during the past year of four young members namely J. L. Mainprize, T. H. Sandrock, A. E. Lund and J. H. L. Cloete. The name of another colleague Charles Tyler was added to these, and the meeting stood in silence for a few moments.

(1) *Minutes* of the General Meeting held on 15th November 1939, and of the Special General Meeting held on 14th December 1939, having been published were taken as read and were confirmed.

(2) *New Members.* — The following new members recommended by Council were unanimously elected: A. A. L. Albertyn, B.V.Sc.; J. Y. Bogue, M.R.C.V.S.; P. H. Brown, B.V.Sc.; P. P. Hugo, B.V.Sc.; W. H. G. Schatz, B.V.Sc.; S. G. Turner, B.V.Sc.; and S. B. Woollatt, F.R.C.V.S.

In reply to a question the Secretary stated that the Association now had very nearly 100 per cent membership.

(3) *Election of Council.* — The ballot for the four vacancies on Council resulted in the election of Drs. J. H. L. Cloete, D. G. Steyn, A. D. Thomas and N. F. Viljoen. The vacancy created by the death of Dr. Cloete would be filled by Council at its next meeting. Council for 1940-41 was therefore announced as follows:—

President. — Mr. S. T. Amos.
Vice President. — Col. C. J. van Heerden.
Hon. Sec.-Treas. — Mr. S. W. J. van Rensburg.
Members. — Dr. R. Alexander, Capt. J. L. Dickson, Dr. P. J. du Toit, Mr. A. C. Kirkpatrick, Dr. D. G. Steyn, Dr. A. D. Thomas, Capt. N. F. Viljoen.

(4) *Presidential Address.* — The Chairman called upon the Secretary to deliver a message from the President. The Secretary stated that Mr. Amos had made all the necessary arrangements to come up from Durban in order to attend the meeting, but unforseen circumstances had prevented this at the last moment, and he had sent a phone message that morning. He wished to convey to the meeting his thanks for their loyal support and for their confidence, which was shown by again electing him without opposition, and he expressed his deep appreciation for the many sentiments of goodwill and support that had been conveyed to him by members at various times. The President appealed to all members not to let the present world crisis diminish their loyalty to the Association but to work along a single channel for the good of the profession.

A question which he considered ought to receive the serious attention of the meeting was the appointment of non-veterinarians as abattoir superinten-

dents. It was the duty of the Association to bring to the notice of the authorities concerned the desirability of appointing to such posts only those people who have had proper scientific training in both the ante- and post-mortem examination of animals and in the diagnosis of the various diseases and the lesions produced by them.

The meeting passed a unanimous resolution instructing the Secretary to convey its greetings to the President and to wish him continued good health.

Capt. Curson then vacated the chair and called upon Col. van Heerden, the new Vice-President, to take over.

(5) *Arrears.* — The meeting approved of the action taken by Council in connection with three members who were over three years in arrear.

(6) *Resignation.* — The resignation of Col. J. Wakefield Rainey, who had left the country, was accepted.

(7) *Reports.* — The reports of the Secretary and Standing Committees were approved.

(8) *Sale of Poisons.* — Dr. Steyn read the report by the Sub-Committee and informed the meeting of the recommendation of the Council meeting held the previous evening. After discussion it was resolved that, as recommended by Council, this report be forwarded to the Heads of the Departments of Agriculture, Public Health, Native Affairs, and Justice and to the Commissioner of Police.

On the proposal of Dr. de Kock it was decided that in the case of the Department of Agriculture the report be submitted to the Secretary for Agriculture by Drs. du Toit and Steyn.

Dr. du Toit pointed out that it was advisable to amend the remarks on laudanum.

(9) *General.* —

(a) *Abattoir Control.* — The following motion passed by Natal Branch on 26th June was discussed: "That this meeting of the Natal Branch of the S.A.V.M.A. views with utmost concern the tendency that has arisen in the Union to appoint officers without the necessary veterinary qualifications for supervision, inspection and control of abattoirs."

Dr. Fourie stated that for the past 18 months he and Dr. Clark of the Department of Public Health had been serving on an Inter Departmental Committee to enquire into abattoirs on the Reef. Their report was now in the hands of the Secretaries for Public Health and Agriculture.

Dr. du Toit informed the meeting that for handling export meat the registration of abattoirs had now to be effected through the Veterinary Division and that it had been laid down that veterinarians must undertake ante-mortem and post-mortem inspection and that animal husbandry officials would only be responsible for the grading and transporting of the meat.

Further discussion centred on financial considerations such as the inability of small municipalities to pay sufficiently high salaries for veterinarians; the present scarcity of veterinarians; the watch continually kept by the Association for any openings which may provide opportunities for the appointment of veterinarians; and the excellent propaganda value of those abattoirs which are under veterinary supervision.

On the suggestion of Dr. Thomas it was decided that the information provided by Drs. Fourie and du Toit be conveyed to the President of Natal Branch

and that this meeting is in full sympathy with the motion passed by that Branch.

(b) *Act No.* 16. — Capt. Curson asked whether anything had been done with regard to the proposed amendments. Dr. du Toit replied that the Board was dealing with these, but that there was no hope of getting legislation passed while the war was on.

(c) *Death of Members.* — Referring to the death recently of several members Mr. Nicol stated that as the Journal is only published quarterly, news of a member's demise sometimes reached his colleagues only after two or three months. Dr. Scheuber proposed and it was agreed that the Secretary notify all members, by circular, of any deaths among members.

The meeting concluded at 10.45 a.m.

S. W. J. van Rensburg,
HON. SEC.-TREAS., S.A.V.M.A.

NATAL BRANCH.

Minutes of the Annual General Meeting of the Natal Branch of the South African Veterinary Medical Association held at Allerton Veterinary Research Laboratory, Pietermaritzburg, on the 26th June, 1940.

Present.

In the Chair: Mr. W. A. Dykins.

Dr. de Kock, Mr. A. S. Canham, Mr. N. C. Starke, Mr. S. T. Jackson, Mr. D. A. Haig, Mr. P. H. Brown, Mr. C. M. Sharpe, Mr. A. M. Diesel, Dr. P. R. B. Smith, Mr. J. Zwarenstein, Mr. F. B. Wright, Mr. W. Schatz, Mr. R. Paine, Mr. S. B. Woollatt.

Secretary: Mr. L. C. Blomefield.

Apologies for Absence.

Mr. S. T. Amos, Durban, and Mr. F. J. Carless, Mooi River.

The Secretary read the notice convening the meeting.

President's Address.

The President referred to the death during the year of three well-known and respected colleagues, Mr. Hamilton, Major Barnes and Mr. Tyler, and also to the death of Mrs. Tyler and Lieut. de Kock. The meeting signified their sympathy in the usual way. The President then proceeded to address the meeting. (See p. 168).

New Members.

The following were proposed, seconded and welcomed:—

P. H. Brown, S. G. Turner, A. A. L. Albertyn, W. Schatz, P. P. Hugo, S. B. Woollatt.

Mr. Diesel suggested that the Secretary should write to Major Bogue inviting him to become a member of the Association.

Office Bearers.

The following were elected:—

President: W. A. Dykins.

Vice Presidents: A. M. Diesel, A. S. Canham, R. Paine.

Secretary and Treasurer: N. C. Starke.

Committee: C. M. Sharpe, S. T. Jackson, D. A. Haig, H. G. J. Franz, and F. B. Wright.

General Business.

In view of the remarks made by the President (see p. 168) the following resolution was put to the meeting and carried:—

> THAT this meeting of the Natal Branch of the South African Veterinary Medical Association views with utmost concern the tendency that has arisen in the Union to appoint officers without the necessary veterinary qualifications for supervision, inspection and control of abattoirs.

It is recommended that this resolution be submitted to the Hon. Secretary of the South African Veterinary Medical Association for further consideration and transmission to the authorities concerned, if deemed necessary.

The President then called upon Dr. de Kock to address the meeting on the subject of Rinderpest in Tanganyika.

The President thanked Dr. de Kock on behalf of the members for his comprehensive and interesting talk on rinderpest, saying that it had made them appreciate the magnitude of the task which is being tackled in Tanganyika.

By the courtesy of Messrs. Bayer Pharma (Pty.), Ltd., two films of veterinary interest were shown to members.

The meeting commenced at 9.30 a.m. and concluded at 1 p.m.

N. C. Starke,
SEC.-TREAS., NATAL BRANCH, S.A.V.M.A.

Presidential Address.

Annual General Meeting of the Natal Branch of the South African Veterinary Medical Association at Pietermaritzburg, 26th June 1940.

I do not intend delaying the progress of this meeting with a long presidential address. I propose handling it on unorthodox lines in order to expedite the proceedings, and 'if I cannot strike oil in ten minutes, I will stop boring.'

In the first place, I want to thank those gentlemen who elected me as the titular head of the profession in Natal during my absence last year, and I should also like to thank the immediate past President (Mr. Paine), and the Secretary (Mr. Blomefield), for the valuable services they have rendered. I also want to extend a very hearty welcome to all members here to-day, especially those who have come from 'over the Berg' to throw some light on the problems met with in the field. I want to express the thanks of this meeting to Dr. De Kock and Mr. Van Heerden for the

sustained interest they have taken in our branch of the S.A.V.M.A. It was with a certain amount of hesitancy that your Committee decided to hold this meeting, as the international position has deteriorated to an alarming extent. It is safe to say that civilisation, as we know it, has never been in a more perilous state, and it is extremely difficult to form any idea of what the ultimate end is going to be; we can only hope for a speedy ending of the catastrophe and that right, justice and truth will prevail..... I should like to refer very briefly to events of veterinary importance. engaged by the Government..... The findings of the Commission which recently investigated veterinary education in Great Britain foreshadowed radical changes in, and State grants for, veterinary education. Finally, I should like to refer to the unveiling of the monument to the memory of that great scientist, Sir Arnold Theiler. The work of Theiler and Dr. Hutcheon of the Cape have contributed immensely towards placing veterinary science on the map, one working in the laboratory, and the other in the field. Their lives should be an inspiration to many of us who are too prone to bask in the sunshine of the achievements of others.

I am addressing you to-day as a municipal veterinary officer, and I wish to place emphasis on this aspect of our work. I have been one who has always maintained that the veterinarian should control such municipal activities as (i) abattoir administration, (ii) animal transport, and (iii) milk hygiene.

The veterinarian, by virtue of his calling, training and scientific attainments, is the proper person to control these activities, and any local authority ignoring these cardinal points will head for disaster in the long run. It is pleasing for me to record that a few progressive local authorities such as Johannesburg and Durban are quite alive to the essential rôle played by the veterinarians in the gamut of municipal administration, and have made them heads of departments directly responsible to the councils concerned. It was a matter of pleasure to me that the City of Port Elizabeth adopted a scheme of abattoir control which I had formulated at the request of the Town Clerk some four years ago, and placed a veterinary surgeon in charge, and I rather hoped that a similar arrangement would have been made at Kimberley, especially as in the latter centre the abattoir will cater for export in addition to local requirements. It is also a matter of disappointment to me that a veterinarian was not appointed on the official delegation which visited other countries, such as the Argentine and Australia, to collect information on the meat trade and abattoir control generally. In the interests of the public some recognition should have been given to our profession in this regard.

I am simply offering these remarks under a deep sense of conviction, and not from any personal motives. I would end my address by advocating increased vigilance on the part of our Association to ensure that positions requiring veterinary skill are filled by veterinarians, and I make the suggestion as much in the interest of the Union of South Africa as in that of the profession itself.

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