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XIII(1): 1-11
1942

THE WILD PIGS OF SOUTH AFRICA:

THEIR DISTRIBUTION AND HABITS, AND THEIR SIGNIFICANCE AS AGRICULTURAL PESTS AND CARRIERS OF DISEASE

A. D. THOMAS and F. F. KOLBE
Onderstepoort

Montgomery (1921) and Walker (1933) in Kenya and Steyn (1928, 1932), and de Kock, Robinson and Keppel (1940) in South Africa have shown quite conclusively that the blood of wild pigs in certain districts contains the virus of swine fever. The fact that such pigs do not succumb to the disease but apparently remain in good health, makes them all the more dangerous as potential carriers. It is well known also that these animals may play an important rôle in the spread of other epidemics such as rinderpest, foot-and-mouth disease and trypanosomiasis. In addition, wild pigs can be very destructive to crops and in some places constitute a major pest to agriculturalists.

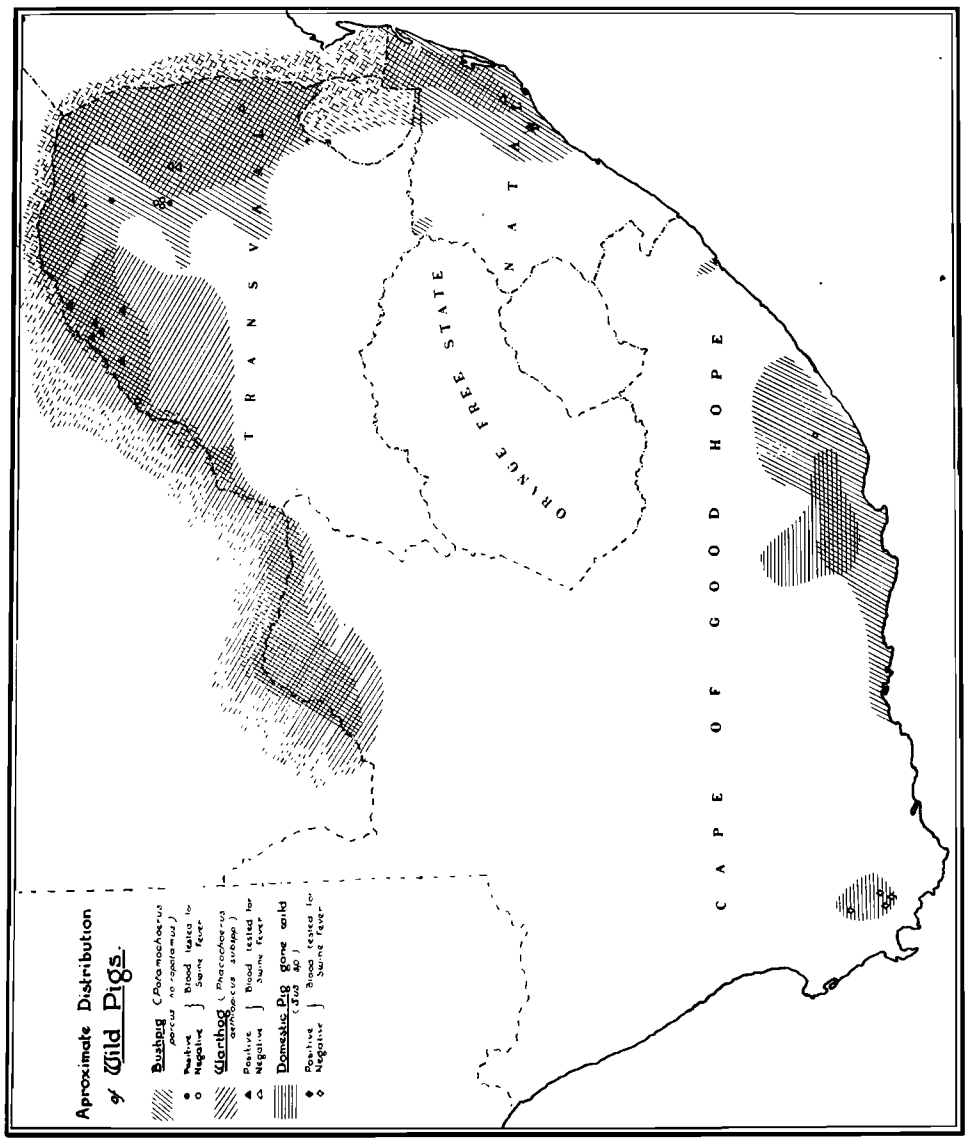
For these and similar reasons we decided to investigate — under the auspices of the Zoological Survey of the Union — the distribution and habits of wild pigs, their influence on our agricultural economy and the most effective methods of control.

The information contained in the present article represents a beginning which we hope may lead to a better understanding of the habits of these animals and will better enable us to control them and the diseases they carry. This information was obtained partly from the answers to a questionnaire distributed as widely as possible throughout the Union, partly from our own observations, and also from the available literature. We were fortunate in having the co-operation of members of the staff of the Veterinary Division and the Forestry Division in all districts to complete the questionnaire as well as to distribute it to likely farmers. In this way about 1,400 forms were distributed and 400 returned. These were carefully analysed and yielded much valuable information.

DISTRIBUTION OF "WILD PIGS" IN SOUTH AFRICA

The term "wild pig" includes only the feral members of the true pig family, namely, the bush pig or bosvark (*Potamochoerus porcus koiropotamus*), the warthog or vlakvark (*Phacochoerus aethiopicus* sub spp.), domestic strains (*Sus* spp.) that have gone wild, and the descendants of some Bavarian wild boars introduced into the Cape. The antbear (erdvark) and the porcupine (ystervark) do not belong to the same order and are not considered in this paper.

The approximate distribution of these types is shown in the accompanying map. The data do not permit us to indicate the relative density of the population in different areas. It will be noticed that the distribution of the types overlaps considerably. This is not surprising in the case of the bush pig and the domestic pig gone wild, for their habits appear to be similar. In the case of the bush pig and the warthog, however, the overlapping is due to the interspersing of the country favourable for the one with country favourable for the



other. For example, thick river bush and mountain forest harbouring bush pig may adjoin typical bushveld suitable for warthog.

The Bush Pig. — Once this pig probably ranged through the natural forests and bush areas of South Africa, from Knysna, through Natal, to the eastern and northern Transvaal. However, more intensive farming, closer settlement, and the 1897 rinderpest outbreak have caused its disappearance from the entire Transkei, East Griqualand and Natal south of the Tugela, except for the Lusikisiki forest reserve and some forested mountain slopes in the Newcastle district. Thus there are today wide gaps in the continuity of the bush pig's distribution, a fact which may yet be of importance in the event of a serious pig-borne epidemic. More recently the bush pig has increased considerably in the well-wooded mountainous parts of Zoutpansberg, Eastern Transvaal, south Eastern Province and Zululand, more especially in the Government forests and reserves where it is protected. It is more rare and exists more precariously in the less favourable environment of the drier and less secluded parts of the country — such as the scrub and bush of the Eastern Province, the riverine thickets of the Transvaal lowveld, the north-western Transvaal, and Bechuanaland.

The Warthog. — This pig ranges from the Umfolozi reserve in Zululand northwards through Swaziland to the lowveld of the Eastern Transvaal and then to the bushveld of the north and north-western Transvaal. It has a more continuous distribution than the bush pig, since the country it favours is not so localized. The warthog breeds relatively fast and may increase enormously in numbers especially in protected areas such as the Umfolozi reserve. On the other hand it is more easily hunted and more susceptible to climatic changes than the bush pig so that it is rare and tends to disappear in farming areas, and its number fluctuates more widely over periods of time.

Domestic pigs run wild. — These are breeding mainly in the dense scrub and prickly pear country in the South-Eastern Cape and are, in parts, believed to be fairly numerous. There have also been reports of domestic pigs gone wild on a few farms in the Transvaal and Natal.

Several years ago the Division of Forestry liberated some Bavarian wild boars in the Kluitjieskraal plantation in order to control destructive insect larvae. These pigs appear to be increasing. Reports have been made that this pig and the bush pig are breeding freely with domestic pigs gone wild. This is not unlikely, but the observations require confirmation.

Habits of Wild Pigs

1. *General.* — Comparatively little is known of the habits of the domestic pig run wild or of the Bavarian wild boar. Generally speaking the bush pig and warthog are both intolerant of strong and cold winds, very dry conditions, and extremes of temperature. Thus

their habitat and activities vary according to the time of the day or the season and they will select that part of their range giving most protection against extreme diurnal or seasonal variations of temperature, sunlight, humidity, wind, wet soil and open water. These factors seem to govern the choice of habitat far more than the presence of suitable feed. As a result, considerable excursions may have to be made in search of food during the suitable periods of the day or night. Under unfavourable conditions — such as drought or persecution — the animals may have to modify their diet considerably.

In the colder months the animals sun themselves in sheltered spots during the early morning and during the greater part of the day when they are not foraging. In summer they forage during the colder time of the day and rest in moist shady spots or in wallows during the heat. In these wallows they become covered with wet soil or mud which dries on the skin to form crusts. Near these wallows one often finds rubbing posts or stones against which the animals rub themselves probably to allay irritation caused by lice, ticks, etc., and by the caked mud.

The breeding season of both bush pigs and warthogs is about the same — November to January — and both make grass nests in which the young are born. The warthog has its nest in a large burrow.

The Bush Pig. — The animals prefer to run in family groups inhabiting fairly well defined areas in the montane forests, wooded kloofs, in riverine thickets, or in reeds. In summer they sun themselves in the early hours, but during the hotter time of the day are

The Bush Pig.

found in the denser and moister parts of the forest or bush, where they have wallows in mud or wet soil and resting places in cool spots. In winter the pigs are usually found in the lighter and drier parts of the range, often at high altitudes, above the mountain morning mists. They sun themselves for long periods in dry grass amongst the short bush and move to deeper bush as the sun gets hotter.

Snouting and wallowing places of bush pig, in old lands covered with "uintjies" and "varkbossie."

During the crop season, when natural foods are scarce, the pigs may migrate temporarily to habitats near crops or they may undertake long nocturnal excursions. They rarely visit the same lands on successive nights. Old lands covered with weeds such as "uintjies" (*Cyperus esculentus*) or "vark bossie" (*Commelina eckloniana*) are also attractive. In rooting these the pigs literally plough over large tracts of land during the night.

In summer the bush pig feeds on fallen fruits and berries, plentiful in the forests, and on roots, corms, tubers, and bulbs of various plants; while in winter it subsists on such corms and roots as it can find. In the forests of the Zoutpansberg the favourite food is the large succulent tuber of the "yam" (*Dioscorea otinefolia*), mushrooms, bracken, ferns and possibly such insect larvae as are found while rooting. These natural feeds are supplemented of course

A grass nest of bush pig, on which young are born.

by crops whenever available or conveniently situated. In the Eastern Province the pigs subsist to a large extent on prickly pear and when food becomes scarce they have been known to feed on carrion and even such live young of game and birds as they can catch. It has been suggested that bark and bracken fern are eaten for their tannic acid content (and possibly anthelminthic properties) rather than for their nutritive value.

During the breeding season — November to January — large grass nests, 8′ x 8′ x 2′, are built in the drier, lighter and most inaccessible parts of the forest or bush. Only during this time does the bush pig restrict itself for any length of time to a portion of its extensive range. The litter is from 2–6 and juvenile mortality is comparatively low. However, leopards and other large carnivores may reduce considerably the number of bush pigs and can act as efficient checks on over-population.

When cornered by dogs, the bush pig backs into a dense bush to protect its rear and puts up a fierce fight. It usually manages to kill one or more dogs before being overcome.

The Warthog. — This pig inhabits light bush or thorn veld where there is sufficient water. During rain and at night it lives in abandoned antbear or porcupine burrows or under overhanging rocks or tree trunks. In drier regions or in winter it is usually near water.

In summer the warthog feeds early in the morning or in the late afternoon. The rest of the day is spent wallowing or sleeping in shade, preferably on slopes receiving the morning breeze. The rubbing posts are usually near their resting places. Early on winter mornings the pig is usually found sunning itself or grazing in the open. It grazes slowly towards the water holes, where it may be found at midday. It grazes for the greater part of the day because of the scarcity of grass, but lies up in the dry grass, protected from cold winds, round about midday. It is often seen grazing with white rhino, wildebees (gnu) or zebra.

The rutting season is from June to August and the young are usually born between November and January, in the burrows mentioned above. The litter usually consists of 3 to 8 young and these do not venture far from home for the first month or so. During this time the family mostly keeps together, but thereafter the boar leaves the sow with the young during the day. The young may now accompany the sow on long excursions. When chased they scatter and hide in the grass or bushes. The boar is again seen with the sow in the rutting season, when the sow may still be accompanied by some of the young. When the family enters the burrow the young go first, followed by the sow and boar, always backwards. The burrows are entered at night or late afternoon, during rain, or when hunted.

The mortality amongst the young seems to be comparatively high, although it is difficult to say whether this is due to disease, nutritional

factors, or to predatory animals. For although it is usual to see four to eight young with a sow shortly after littering, there are usually only two to four a few months later.

The warthog is much hunted by natives, and, when chased into its burrow, is dug or smoked out and speared, or killed by dogs. Sometimes it is surprised in its burrows during rain or at night. It bolts out when alarmed — as when natives stamp on the ground — and is soon accounted for by dogs or spears. Thus this pig has been almost or entirely exterminated in native settlements. In the game reserves large carnivora also take considerable toll. During favourable

The Warthog.

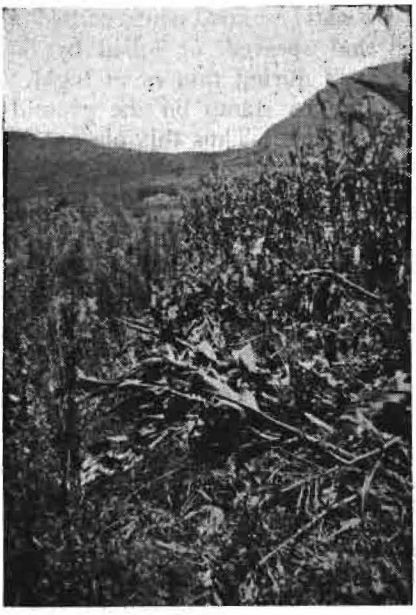
years the number of warthogs may increase alarmingly, where protected from their natural enemies. Drought, however, may reduce the numbers to insignificance.

Economics of the Wild Pig

(1) *Adverse aspects.* — The most destructive to crops is the bush pig. It damages maize, sugar cane and other crops extensively. Maize plants are knocked down right and left with the teeth and only some of the cobs eaten. We have seen a family of bush pigs, consisting of boar, sow and three young, knock down 1,563 maize plants in five visits. In the last visit alone 433 plants were knocked over. In Zululand there have been reports of tremendous damage to sugar cane. The bush pig is also blamed for extensive depredations to pumpkin, potato, sweet potato, pineapple, wheat, and barley crops.

Apart from this, some harm is probably done to the natural

vegetation. Ferns, lilies and other bulbous plants are destroyed, lianes and tree seedlings are rooted up and broken so that the natural succession may be retarded and the natural beauty of the vegetation and cover to the soil disturbed.



Mealies tusked and knocked over by bush pig.

When the numbers of bush pigs in plantations are large the damage to young plants by rooting and to older trees by barking may be considerable and serious.

At times — and particularly in the Eastern Cape — considerable numbers of ground birds' nests and young game are destroyed, as well as an occasional lamb and weak sheep. Still worse is the pig's habit of holing jackal-proof fencing and making an entrance for these destructive pests.

Dissemination of Disease. — Bush pig, warthog and domestic pigs run wild may all be responsible for spreading disease. In the south-eastern Cape the presence of sarcoptic mange in the bush pig and the wild domestic pig makes the eradication of the disease amongst the domestic animals impossible. In Zululand, not only are warthogs and bush pigs carriers of nagana, but they are also important food hosts for the tsetse fly. It has already been mentioned that wild pigs suffered heavily in the 1896 rinderpest epidemic. They are today still involved in the spread of this disease in Central Africa. The same applies in all probability to foot-and-mouth disease.

In regard to swine fever, which is more specifically a pig disease, wild pigs not only assist in spreading the infection, but, what is more important, actually act as reservoirs for this disease by harbouring the virus in their system for indefinite periods.

Although every opportunity to subinoculate blood from wild pigs has been utilized, the data now available are still too meagre to enable us to plot the areas in which carriers occur. It would appear that the percentage of infected wild pigs is high in the northern Transvaal and much lower in the eastern Transvaal, while the infection is absent or very low in wild pigs of the Cape Province and Natal.

(2) *Favourable Aspects.* — Phillips (1926, 1931) has stated the case for the bush pig, probably as favourably as it can be stated, when

he says that germination and dispersal of forest seeds may be aided by the bush pig. One of us has failed to find evidence of this in the droppings of bush pigs in the Zoutpansberg. The rooting of the pigs may improve soil æration and prepare the ground as seed beds. These benefits, if real, are probably counterbalanced by the danger of soil erosion arising from the denudation and rooting done. Wild pigs have also been credited with the destruction of grubs and other insect larvae. The evidence, however, is scanty. We were able to find only four caterpillars in the stomach of a bush pig.

More data are necessary before the economic status — adverse or otherwise — of the bush pig can be determined. At present it seems that it is responsible for more harm than good — even if its possible rôle in disease transmission is ignored.

The Control of Wild Pigs

Natural foes, leopards, lions, wild dogs, and unfavourable conditions such as disease, extremes of climate, drought, and other less understood factors exert a check on over-population. A steady increase of wild pigs in certain parts is probably due in part to a reduction of these natural enemies. Sometimes accidental factors play a minor part, as when poisoned bait was used for jackals, or when arsenic pentoxide was used to eradicate prickly pear.

Many ways of destroying the bush pig or of limiting its destructiveness for crops have been tried. In some parts it is usual for natives to surround their cultivated lands with small open shelters. In these shelters natives and their dogs keep nightly watch. Europeans have also tried this method, but it is a tedious method which, at best, is only partly effective, since the bush pig is cunning and frequently enters the lands without its presence being discovered. Wire fences, unless elaborately built, are useless. Electric fences strung about 18 inches from the ground are said to be effective in Zululand around sugar cane fields. We have not found them of great use and have only operated them successfully when a barbed wire is used and when the soil is moist.

In Central Africa natives dig ditches about 2 feet deep and 15 inches wide, or pits 3 x 2 feet, spaced at equal intervals around the cultivated lands with mounds of loose earth between them. Otherwise a low brushwood pallisade 2–3 feet high is used. These methods are reasonably effective.

Other methods of destruction have been tried by farmers. Shooting in the lands at night by moon or spot-light is a wearisome business, and, since these pigs rarely come twice in succession to the same lands, frequently discouraging. Hydrocyanic acid capsules in meat or prickly pear are dangerous, as is the use of dynamite detonators in sweet potatoes. Spring guns are also unsafe, and the snares, pits and traps set by natives usually collect harmless animals. Thus although some of these methods are supposed to give good results, their efficiency is

a matter of chance and the results inconstant. If one or other of the less dangerous methods could be adapted to the local conditions and habits of the pig, success would be in sight. For this, the life of the bush pig must be studied.

So far, the best results have been obtained from organized hunts with guns, dogs and beaters. However, the organization necessary and the personnel and equipment required, as well as the real danger of losing dogs, makes this method a costly one.

The warthog can be reduced comparatively easily by shooting, hunting with dogs, and gassing and shooting in the burrows to which it runs when pursued by dogs. When a few hunters have to cover a wide area, the simplest way is to hide at the water holes in the morning, midday, or late afternoon.

Summary

1. An attempt has been made to define the distribution of wild pigs in South Africa, to study their habits and to assess the harm and good they do to our agricultural economy.

2. The rôle played by wild pigs in harbouring and spreading disease, especially swine fever, is discussed. Unfortunately the available data concerning the extent of reservoir infection in nature are too meagre to enable the implicated areas to be plotted.

3. Present means of control, for the bush pig at any rate, are difficult, expensive and mostly unsatisfactory. The appreciable increase of this animal in our forest reserves and plantations will no doubt soon necessitate research to discover more effective methods.

Acknowledgments

Our thanks are due to the Director of Forestry and members of his staff all over the Union, to the Senior Veterinary Officers, District Veterinary Officers and Stock Inspectors in the various Provinces, and to the many farmers who so willingly assisted in collecting the information without which this article could not have been prepared.

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CORRECTION

Vol. 12. No. 2. pg. 58. The fourth paragraph should read as follows: 7. 40% nicotine sulphate added to the arsenical dip in the proportion of one part per 1000 by volume to produce a 0·04% mixture solution effectively controls this strain of blue tick. A proportion of the ticks escapes, as only after 6 to 7 dippings at seven-day intervals are cattle entirely freed from ticks.

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XIII(1): 12-14
1942

TWO CASES OF MYOSITIS CAUSED BY SARCOSPORIDIA

R. CLARK and CECIL JACKSON
Onderstepoort

INTRODUCTION

Sarcosporidia are so commonly met with in sections of muscular tissue from animals in this country that their presence is usually ignored. In the vast majority of cases they appear to elicit no reaction from the tissues. The following cases are recorded because they showed focal necrotic centres with a marked inflammatory reaction and these changes could be shown to be due to sarcosporidiosis. Where only the advanced lesions can be found, an aetiological diagnosis can be made only by inference. This description may, therefore, be of aid to those faced with the difficulty of assigning a cause to similar lesions. The occurrence described in the horse is of special interest, as this animal showed clinical symptoms.

Myocarditis in a Pig

The specimen was forwarded by the Director of the City Abattoirs, Durban. Sarcosporidiosis was suspected, but the heart was sent for histological examination as several cases of similar massive infection had been encountered. The skeletal musculature was not visibly affected and the carcass had been passed for human consumption. Macroscopically the myocardium showed a very heavy infection of typical sarcocysts but, in addition, numerous greyish opaque areas of about the same size as the cysts could be seen scattered throughout the tissue.

Histopathology. — The section showed the presence of a fair number of normal sarcocysts, round which there was no reaction. In addition to these, however, there were numerous necrotic foci surrounded by masses of round cells, fairly numerous eosinophiles, and occasional mast cells. Some of the necrotic centres were also surrounded by pallisade tissue. In some of these foci, degenerate sarcocysts could be made out in the centre of the necrotic area. These degenerate cysts were, in almost all cases, much larger than the normal cysts, but remains of the wall could be clearly seen, containing scattered pyknotic cells and, occasionally, undoubted sarcospores. The infiltrating cells nearest to the cyst had undergone necrosis. The interstitium, for a considerable distance around these foci, was markedly infiltrated with round cells and eosinophiles. Even in some of the advanced lesions, where the centre consisted of a solid mass of necrotic material, portions of cyst wall could still be seen. There was, therefore, no doubt that these disseminated necrotic foci were due to sarcosporidia.

Myositis in a Horse

In 1937, specimens from a horse were forwarded by the Government Veterinary Officer, Ladysmith. In the accompanying report he stated that the animal showed a marked bilateral swelling of the masseter muscles, which were soft and doughy but did not pit on pressure. The animal lost condition and later developed violent colic. After treatment with arecolin hydrobromide it passed 'a mass of semi-masticated ingesta.' On post mortem the intestinal tract contained badly masticated food.

From this report it would appear that the condition of the masseter muscles had seriously interfered with mastication and was indirectly the cause of the colic.

Histopathology. — One of us (C.J.) examined the masseter muscle and found multiple small caseous to calcified foci, containing the remains of eosinophiles. These foci were surrounded by infiltrating round cells and eosinophiles, and were weakly encapsulated. Some were also surrounded by pallisade tissue, and occasional giant cells were seen. There was a marked fibrosis with cellular infiltration consisting of round cells and eosinophiles of the interstitium of the whole muscle. A diagnosis of chronic sarcosporidial myositis was made from the nature and distribution of the lesions. A comparison of these lesions with those seen in the myocardium of the pig, described above, leaves no doubt as to the correctness of this diagnosis. From the facts that there was a general fibrosis of the muscle and that all the foci were completely necrotic and encapsulated, it can be assumed that the process was of much longer standing than in the case of the pig.

Discussion

Inflammatory reactions due to sarcosporidia are mentioned in the literature, and Ziegler (1929) in Joest's *Spezielle pathologische Anatomie der Haustiere* includes a photomicrograph of lesions in muscle very similar to those seen by us in the horse. The general impression in South Africa, however, appears to be that sarcosporidia are always non-pathogenic. This is probably due to the work of Walker (1918) on sheep and horses and that of Viljoen (1918) on cattle, neither of whom encountered a case of sarcosporidial myositis. As these authors have fully reviewed the literature it is unnecessary to refer to the many previous articles dealing with sarcosporidiosis. The ubiquity of the parasite and the fact that there is usually no reaction to its presence shows that, in the vast majority of cases, the presence of sarcosporidia causes no noticeable ill effects. The object of this communication is to show that, in rare instances, severe lesions may arise in at least two domesticated species, viz., the horse and the pig.

The presence of degenerate parasites and a severe tissue reaction may either mean that the body has reacted to the parasite and destroyed it, or that the parasite has become degenerate and subsequently evoked

the inflammatory reaction. A study of the lesions in the myocardium of the pig inclines one to the latter explanation. No reaction could be found round any of the normal cysts, whereas all stages of disintegration of cysts were present and all of these were centres of severe reaction. Furthermore, the invading cells nearest to the degenerate cysts had undergone necrosis, indicating the presence of a toxin in the cysts. It is well known that an extract of sarcosporidia contains a toxin " sarcocystin " which is extremely active when injected into animals. It may well be, therefore, that when a sarcocyst disintegrates in the muscle tissue, sarcocystin is liberated, causing a severe reaction.

Summary

A case of sarcosporidial myocarditis in a pig and one of sarcosporidial myositis affecting the masseter muscles of a horse are recorded, since inflammatory reactions due to sarcosporidia have not previously been encountered in domestic animals in South Africa.

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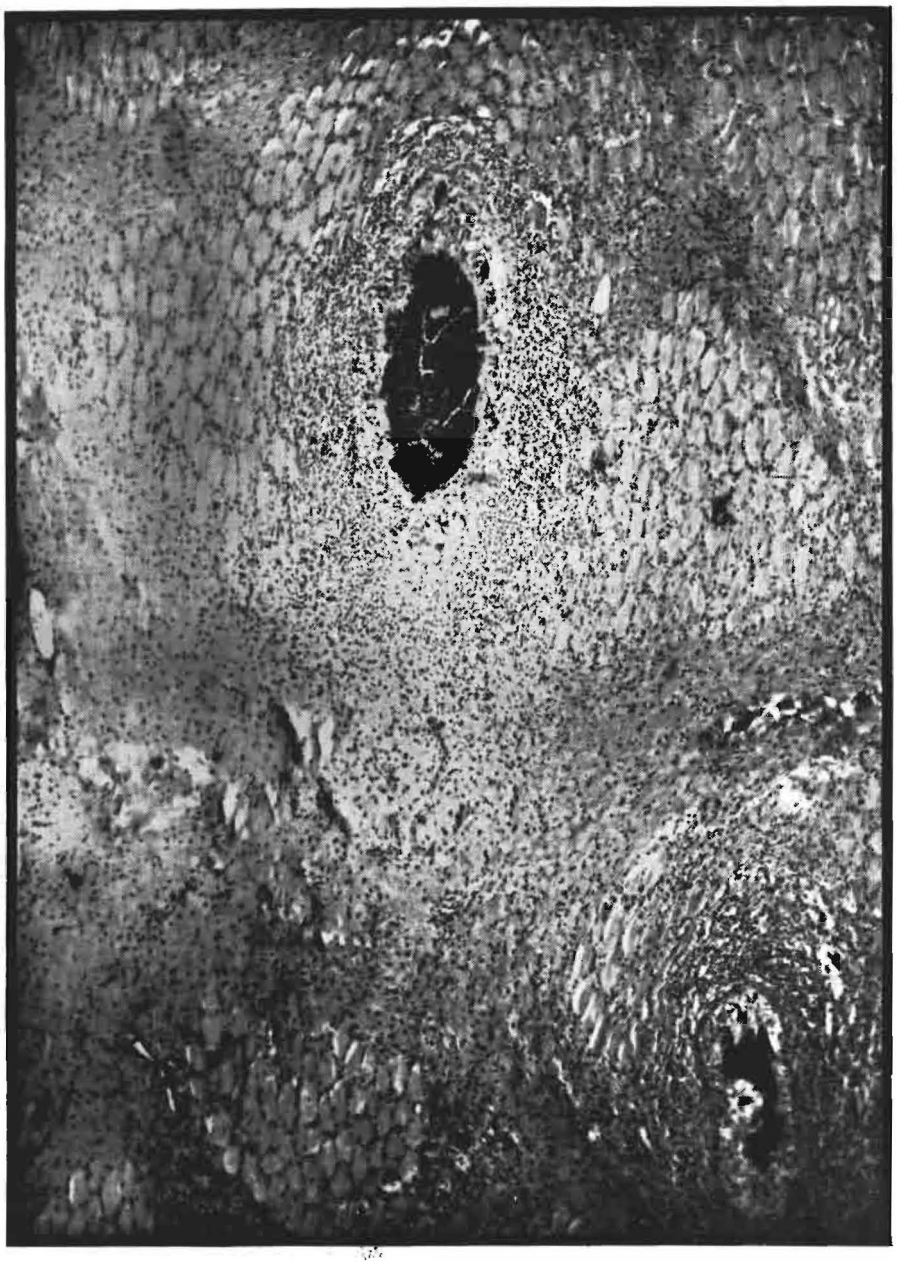
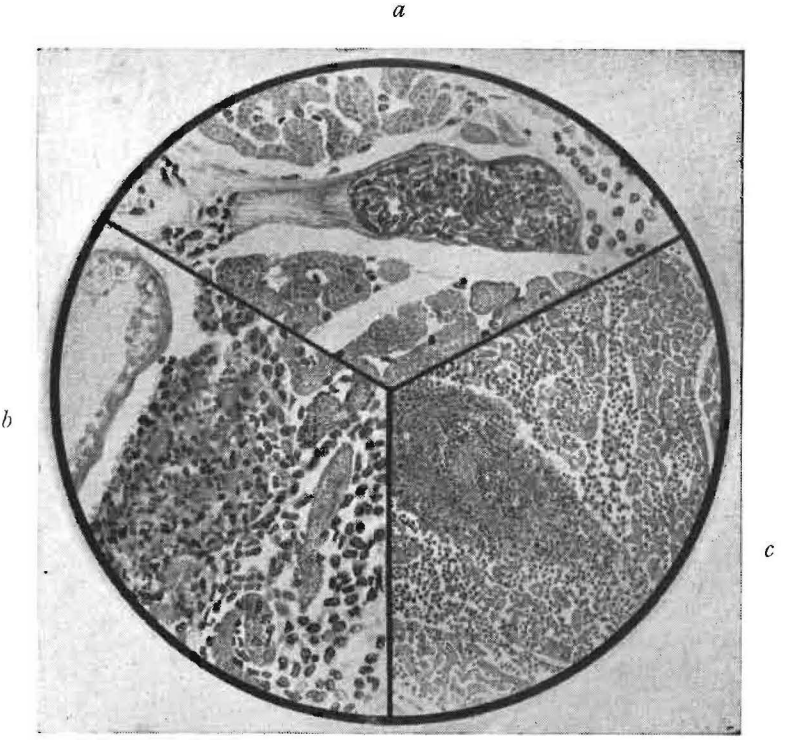


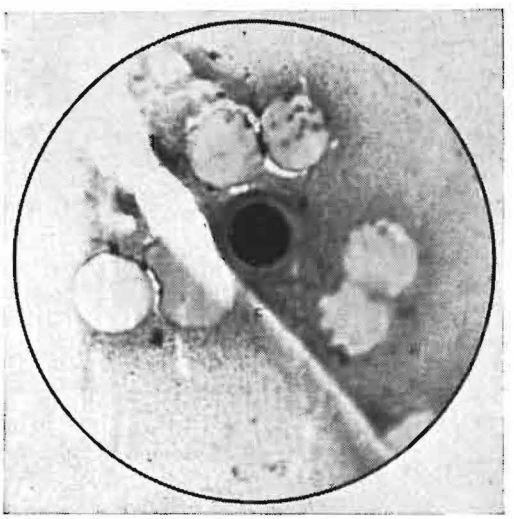
Chronic myositis of the masseter muscles of a horse. Note the marked fibrosis and the remains of two sarcocysts. (Path. No. 20620.)

Two cases of Myositis caused by Sarcosporidia



Sarcosporidial myocarditis in a pig. (Path. No. 26608).
a. A normal sarcocyst with no reaction. (× 250)
b. A degenerating cyst (above) which is the centre of an intense reaction. Note necrosis of the cells nearest the cyst wall. (×250)
c. A necrotic focus with the cyst completely disintegrated. (× 56)

Eperythrozoon felis (sp. nov.) in a Cat



Eperythrozoon felis (× 1600) (Path. No. 27074).

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EPERYTHROZOON FELIS (SP. NOV.) IN A CAT

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During November, 1941, Mr. R. Paine, F.R.C.V.S., of Pietermaritzburg, Natal, forwarded blood and spleen smears from a cat to Onderstepoort. These smears showed marked anæmic changes and the presence on the erythrocytes of supracellular bodies which, morphologically, could not be distinguished from *Eperythrozoon* as described from other animals.

The subject was a male castrated cat about nine years old. Unfortunately the clinical history was very vague as the owner had been away and returned to find the cat ill. When presented for examination it was weak, emaciated and very anæmic with a temperature of 100°F. The animal was destroyed the following day. The post-mortem examination revealed marked fatty changes in the cortex of the kidneys and a few small hæmorrhages under the peritoneum. The spleen was normal in size. The blood smear showed extensive regenerative changes, jolly bodies and normoblasts were frequent and even occasional erythroblasts were present.

A low percentage of erythrocytes (about 10%) showed the presence of typical *Eperythrozoon*, which were present on the immature basophilic cells as well as on the mature cells. Infected normoblasts also occurred. The parasites were practically all ring forms, 0·5 to 1·0 μ in diameter and stained a delicate pale violet with Giemsa. The centre of the parasite did not stain. A few of these bodies were ovoid but comma and rod forms were infrequent. The number of parasites per infected red cell was not high, usually two to four, but some cells contained up to twelve. The organisms were not evenly distributed over the smear, a fact usually noted in preparations of *Eperythrozoon* species and which is considered to be due to mechanical displacement of the supracellular parasites during the process of making the smear. The parasites appeared to be flattened and did not project from the surface of the erythrocyte.

Neitz (1937) has shown that neosalvarsan and the arsenostibio preparation Std. 386B both have a specific action on *Ep. ovis* and these drugs would, therefore, be indicated in the treatment of *Ep. felis*. Neitz (1940) also points out that atoxyl is reported to be specific against *Bartonella muris*. This drug might be tried against *Eperythrozoon* species.

Discussion

Careful examination, especially with the arc lamp when making the microphotographs, convinced the author that the epicellular bodies seen were not artifacts. Morphologically they must fall under the

family *Anaplasmidae,* tentatively proposed by Neitz, Alexander and du Toit (1934). Their epicellular position and the predominance of ring forms precludes their being either *Grahamella* or *Bartonella* and strongly indicates that they should be placed in the genus *Eperythrozoon.*

As *Eperythrozoon* species are known to cause severe anæmia in other animals, e.g. man and sheep, the fact that the subject in this case showed anæmia is significant. As no experimental work has been possible, it cannot be stated whether the blood changes were due to the organisms observed or not, but very thorough examination of the blood smears failed to reveal the presence of any other parasite. As our knowledge of the diseases of cats in South Africa is so meagre the discovery of this new blood parasite may prove of importance to the clinician.

Since this parasite has never been seen in cats or other felines it is presumably a new species for which the name *Eperythrozoon felis* is suggested.

The classification of the *Eperythrozoon* has been reviewed by Neitz *et al* (1934) and a full list of references is given by Kikuth (1932).

Acknowledgments

The author wishes to thank Mr. R. Paine for sending the smears and for supplying the clinical and post-mortem findings. Also Mr. T. Meyer of Onderstepoort, for taking the photomicrograph.

Summary

1. An *Eperythrozoon* has been found in blood smears from a domestic cat. This is presumed to be a new species and the name *Eperythrozoon felis* is proposed.

2. The cat showed marked anæmia which may have been due to this parasite.

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THE VOLE BACILLUS

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From time to time interest in the subject of immunization against tuberculosis is revived by the publication of articles suggesting new lines of research. Since the investigations of von Behring on the immunization of cattle against bovine tuberculosis, with human strains, a number of methods for producing resistance to tuberculosis in man or in cattle have first aroused interest and hope and subsequently caused disappointment. The method which has aroused the greatest interest so far has been the one which makes use of a bovine strain attenuated by subculture on potato soaked in ox bile. The subcultures were made every six weeks for many years and the strain resulting was called Bacillus Calmette-Guerin or B.C.G. — after the investigators who produced it. This vaccine has been the subject of numerous publications and much heated controversy. It has been used on a large scale for human beings, but in France the statistical evidence in support of its value is not very convincing. Opinions also vary as to its value for the immunization of calves against tuberculosis. In many countries eradication campaigns based on the use of the tuberculin test are in progress and, as vaccination would interfere with the test, immunization methods, such as the use of B.C.G. vaccine, are not encouraged.

Recently the possibility of a new method of immunization has been investigated. This is based on the use of a strain of tubercle bacillus isolated from the field vole, *Microtus agrestis*. Wells (1937) described the occurrence of a form of tuberculosis in this species of animal in many different areas in England, Scotland and Wales. In all 143 cases were studied. Griffith (1939), a leading authority on the typing of tubercle bacilli, had made the statement that tuberculosis was unknown in mammals in the wild state. However, following on reports of widespread mortality in voles, a disease resembling tuberculosis was noticed in them. Voles under natural conditions show cycles of increase over a period and then a drop to a low figure for a few months. The possibility of this decrease being due to disease was the starting point of the investigation.

The lesions seen in the voles affected with the disease were as follows:— Caseous areas were present in the lungs and subcutaneous tissues and involved the glands of the neck, axilla, inguinal region, and back. The mediastinal and mesenteric glands were enlarged and caseous and the spleen was enlarged. Acid-fast organisms resembling tubercle bacilli were frequent in the lesions. On Dorset's egg and Petroff's medium a growth similar to that of tuberculosis strains was obtained after six weeks.

Suspensions of the caseous material were inoculated subcutaneously and intraperitoneally into guinea-pigs, voles and rabbits. The voles died of the disease, but the rabbits survived, although showing large local lesions. Three inoculated guinea-pigs showed widespread generalized lesions with numerous acid-fast bacilli. All the animals reacted to tuberculin a month after inoculation. Wells suggested that he was dealing with human or bovine tuberculosis and that the disease might be of importance to human beings. He mentioned that Griffiths was typing the organism, but up to the present no publication on its position amongst the types of *M. tuberculosis* has appeared.

Wells and Brooke (1940) described their experiments on immunization of guinea-pigs against virulent human and bovine strains of the tubercle bacillus by means of the vole bacillus. They did not claim any statistically significant results but thought a preliminary report warranted. It was found that 5 mg. or more of culture subcutaneously might produce progressive disease and death, but smaller doses would only produce a local abscess with caseation and sometimes ulceration which subsequently regressed. In the first experiment three guinea-pigs were inoculated with caseous material from natural cases in voles. This produced a local lesion with ulceration, but no generalization. Nine months later three guinea-pigs, and three controls, were given a very small dose, 0·000001 mg. of a bovine strain of low virulence. Only one of the inoculated guinea-pigs showed signs of slight generalization six months later, whereas the controls showed extensive generalized tuberculosis.

In a second experiment five guinea-pigs were inoculated with 1·0 mg. of vole bacillus culture and five with 0·1 mg. Three months later the guinea-pigs and five controls were given 0·00001 mg. of a recently isolated human strain. The controls died in less than three months of marked generalized tuberculosis. One guinea-pig that had received 0·1 mg. of vole culture died in two months and showed lesions in various organs. The rest of the guinea-pigs were killed at three months. Two of those that had been "immunized" with 0·1 mg. vole culture showed generalization, but those that had received 1·0 mg. showed only small local abscesses. Judging from these experiments 1·0 mg. of vole bacillus culture gave a better immunity than 0·1 mg.

In a third experiment the immunizing value of two different strains of the vole bacillus was compared with that of B.C.G. Group (1) of 26 guinea-pigs received four weekly injections, totalling 2 mg. of a strain of the vole bacillus. Group (2) of 26 guinea-pigs received exactly similar injections of another strain, and Group (3) of 30 guinea-pigs was given four inoculations of 7·5 mg. each of a B.C.G. culture, an amount which was considered optimal for immunization. An epidemic disease killed all but 27 of these animals. Five months after the inoculation, group (1) received 0·000001 mg. of a recently isolated human culture and group (2) 0·000001 mg. of a recently

isolated bovine culture. Group (3) was divided into two and one lot were inoculated like group (1) and the other like group (2). Sixteen controls were put in, half receiving human and half bovine culture. The guinea-pigs were killed after eleven weeks.

In the vole bacillus groups eleven showed nothing or only a local abscess while four showed slight generalization. All the B.C.G. animals showed generalization, but not as marked as in the controls, which all showed extensive generalization. Two-thirds of the guinea-pigs inoculated with vole bacillus showed caseous matter containing the vole bacillus at the site of inoculation. In this last experiment all the guinea-pigs were tuberculin tested either with the precipitated protein derivative or with old tuberculin. The controls became sensitive much sooner than the inoculated guinea-pigs. None of the guinea-pigs inoculated with vole bacillus reacted to a 1/1000 dilution and only three out of the experiment with B.C.G. reacted. The authors concluded that the vole bacillus gave guinea-pigs a degree of protection against tuberculosis apparently far greater than that obtained by other means.

Griffith and Dalling (1940) carried out a number of experiments with the vole bacillus on guinea-pigs and calves. They found that large doses given subcutaneously or small doses given intraperitoneally did not produce progressive disease. Subcutaneous inoculation caused a local abscess with general dissemination, but the lesions were retrogressive and ultimately healed. Large doses intraperitoneally might result in fatal generalized tuberculosis.

The immunizing power of the vole bacillus against a virulent bovine strain was tested. Ten guinea-pigs received 10 mg. of vole bacillus culture over a period of two months in eight doses of 1·0 mg. and one of 2·0 mg. at weekly intervals. Four weeks after the last dose all the guinea-pigs, with five controls, received 0·001 mg. of a virulent bovine strain subcutaneously. The controls died of severe generalized tuberculosis in an average of 87 days. The inoculated guinea-pigs lived on an average about 150 days, dying of severe generalized tuberculosis. The action of the vole bacillus was simply to delay the development of tuberculosis.

In a second experiment each of eight guinea-pigs received, over a period of eight weeks, two doses of 1·0 mg. and five of 2·0 mg. (12 mg. in all) subcutaneously. One day after the last dose the vaccinated guinea-pigs and four controls each received 0·01 mg. of a bovine strain subcutaneously. The controls died in an average of 71 days of severe generalized tuberculosis. The vole bacillus guinea-pigs had not developed marked generalization in 95 days; one showed moderate and the others slight generalization. Some of the guinea-pigs showed lesions at the inoculation site extending to the nearest lymphatic glands. Again, in this experiment, the guinea-pigs inoculated with the vole bacillus were more resistant than the controls, but eventually would probably have died from generalized tuberculosis. These results

were encouraging, so that the pathogenicity and immunizing value of the vole bacillus for calves were tested.

The calves received doses of vole bacillus culture varying from 0·01 mg. to 10 mg. intravenously, but one received 80 mg. intramuscularly. Tuberculous lesions of a chronic type were produced in the calves but tended to disappear without leaving many traces. Slight necrosis, but no proper caseation, was sometimes noticed. The longest period after inoculation that the organism was recovered was 89 days, but that must not be regarded as the limit of survival.

The calves which were not killed, or which did not die of some intercurrent condition, were used in an immunity experiment. Nine calves were available, five of which had received two injections, and their resistance was tested at periods varying from 65 to 175 days later. Two controls were used and the test dose was 7·5 mg. of a culture of a virulent bovine strain per os. The controls were killed, one 208 and the other 223 days after receiving the virulent culture. Both showed wide-spread generalization. Of the vaccinated calves, five showed trivial lesions in the glands of the alimentary tract and the other four had no macroscopic lesions in any organ. In three calves there were abscesses at the inoculation sites, but the pus was sterile. Lymphatic glands from a number of vaccinated calves were inoculated into guinea-pigs and in some cases fatal tuberculosis was set up. The infection of the glands must have been slight as in most cases only one out of two guinea-pigs died.

All the calves were tested with tuberculin and gave definite reactions, some very pronounced, skin measurements of 22 to 36 mm. being obtained. Sensitivity developed as soon as thirteen days after inoculation with the vole bacillus. They were all tested, with the controls, 38 days after feeding with bovine bacilli and reacted well, but on the whole not as strongly as the controls. Good reactions were seen in four calves which did not show any lesions at post mortem later on. Griffith and Dalling in their conclusions considered that the results obtained with the vole strain were unexpectedly good and better than those obtained with the B.C.G. strain. They suggested that further tests were necessary, however, and that in future experiments only the intravenous inoculation method should be used with a dose of 5 mg., the resistance to be tested after six months.

No further articles on the vole bacillus have appeared, but others may be expected as the organism has been distributed to a number of investigators. Two strains have been obtained from Professor Dalling and are being kept and studied at Onderstepoort, from the point of view of pathogenicity and immunizing powers. It is too early to say whether the immunizing properties of the organism will have any practical application, if one bears in mind the history of immunization attempts with tubercle bacilli in the past. The organism will be

subjected to a very critical study in many parts of the world and, until more is known about it, judgment will have to be reserved.

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ACUTE NICOTINE POISONING OF CATTLE

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In our search for a satisfactory tsetse-fly repellent we naturally turned for help to the work of those who have had long experience in protecting plants from insect ravages; and so nicotine sulphate was brought to our notice. We admit now that we paid more attention to the claims of the horticulturist than to the warnings of the veterinarian.

We consider that a satisfactory fly-repellent must be cheap, non-toxic, easily applied, and protective for several days. Of these properties, cheapness is the least important to the experimentalist; the really important thing is to discover something that is protective and non-toxic; then demand will certainly make for cheapness through improvement of formula and by mass production. Accordingly when our first experiments with nicotine sulphate were promising we were not disturbed by the consideration that the drug is expensive.

Our present technique for testing the tsetse-repellent action of any compound is in a series of steps, of which the first three (beyond which we have not yet gone!) are as follows —

(1) The compound is applied to a small clipped area of the skin of a housed sheep. After twenty-four hours, hungry tsetse flies in gauze-mouthed jars are applied for six minutes, and a note is made of their behaviour and particularly of the number that feed. Less than 10% of the test flies feeding indicates repellence, and the second step is taken.

(2) An ox that lives out of doors all day is used. Again only a limited area, but this time of unclipped skin, is treated with the

compound, but now tsetse-feedings are made every day up to the third day after medication, and control batches of flies are applied to an untreated portion of the skin. If not more than 10% feed during the three days' trial, and if the dressing has caused no local irritation, then the third step can be taken.

(3) This is the application of the compound to the whole surface of the animal, usually by spraying, to test its toxicity.

Commercial nicotine sulphate was disappointing in that the simple test of step 1 was not passed by any concentration of less than 3 per cent. However, as this solution appeared to cause no local irritation, even when instilled into the eye, the next step was proceeded with. The results of testing hungry flies on the third day after application of the drug were as follows —

Control bottles							25	out of	50	fed
Against	1%	nicotine	sulphate	....			9	,,	,,	,, ,,
,,	1½%	,,	,,	....			5	,,	,,	,, ,,
,,	2%	,,	,,	....			2	,,	,,	,, ,,
,,	2½%	,,	,,	....			2	,,	,,	,, ,,
,,	3%	,,	,,	....			1	,,	,,	,, ,,

It was considered that the third step, i.e. the testing of the toxicity of a weak solution sprayed over the whole body, might now be taken.

Experiment 1

On 10th October, 1941, an old, red, zebu cow (No. 14) in poor condition, was sprayed thoroughly with two litres of 1·2 per cent. aqueous solution of nicotine sulphate. There were some small skin abrasions, and there was a small amount of licking after the drug was applied. Spraying was completed at 10.30 a.m. At 11 a.m. the animal was in distress and staggering. She stood with legs straddled. Respirations, mainly abdominal, were violent and at the rate of 24 p.m. Pulse weak, but frequent (150 p.m.). Muscular tremors in post-scapular and pre-crural regions. Short strings of thick saliva hung from the mouth. By 11.30 a.m. the respiratory efforts were reduced. The pulse was infrequent and weak, and impossible to count owing to tremors, which by now had extended to the head and neck. The rectal temperature was 99·1°. The eyes were sunken. Shortly after 11.30 a.m. she sank to the ground, and after a few attempts to attain the breast position she lay prone and became unconscious. Death occurred at 12 noon.

Post mortem. — No abnormalities other than some engorgement of subcutaneous vessels, and slight hyperæmia of mesentery and intestines, were recorded.

Experiment 2

An old, red, zebu cow (No. 2) in fair condition. She had an intact skin except for brand scars and two small abrasions caused by

struggling in the crush at the time of spraying. Her head was tied up so that she could not lick herself. At 9.10 a.m., on 11th October, 1941, she was sprayed thoroughly with 2 litres of 1·2 per cent. aqueous solution of nicotine sulphate, only the face within reach of the tongue being left dry.

By 9.20 beads of moisture broke out on the muzzle. Temperature 100°F., pulse weak, but throughout the time of observation she either struggled, swayed or trembled so much that the pulse could not be taken properly. This animal continued to show symptoms of acute nicotine poisoning and died at 10.00 a.m.

Post mortem. — Retraction of eye-balls; cyanosis; great engorgement of subcutaneous vessels; poor clotting of blood; flabby, empty heart; contraction of spleen; lack of tone throughout alimentary tract; œsophagus filled with ingesta; abomasum and intestines flabby and with liquid contents; pharynx, larynx, trachea and bronchi contained ingesta; cerebral meninges injected.

Experiment 3

A young, white, zebu bull (No. 34) in good condition. His skin was intact except for brand scars. All but the head was sprayed with two litres made by adding 0·8 per cent. of nicotine sulphate to a 3 per cent. solution of wattle extract. It was hoped that the fine suspension thrown down might be repellent yet not absorbed. This liquid was sprayed without difficulty, and soon dried leaving the hairs matted and pink.

By 9.30 a.m. on 17th October, 1941, spraying was completed, and the bull was released from all restraint. By 9.40 he had passed two small lots of dung. He had licked his sheath and hump. There was salivation, continual licking of nose, and frequent grinding of teeth. By 9.50 he had licked his flank, hoof and, once more, his sheath. Apart from teeth-grinding, he appeared normal, so he was turned into a field, where he commenced grazing. Thereafter observations were not continuous. At 10.30 he was lying down. Respirations as in case 2, apnœa after quick inspiration. Small quantities of dung passed. The sweating stage (if it occurred!) was past, as the muzzle was rather dry. Pulse almost imperceptible. At 11.30 he was about the same, but the eye-balls were now sunken. By 2.30 he had got up and moved to a more shady spot. Although he was still dejected and off-feed, his eyes were now normal and his respirations fast, shallow, but regular. By 4 p.m. he had recovered and was feeding, and thereafter remained normal.

These three experiments showed clearly that nicotine compounds applied extensively were very toxic. It remained uncertain, however, whether the poisoning was due chiefly to absorption through unbroken skin, or to absorption through abrasions, or to licking, or to absorption through abrasions and licking.

Experiment 4

To test the likelihood that abrasions were the sites of absorption. — On 20:10:41 ox No. 6559 was dressed with 1·2 per cent. aqueous solution of nicotine sulphate, but only to the extent that a broken-down brand scar, 3in. x 1½in., was scrubbed with cotton-wool soaked in the solution until the whole wound surface was bleeding slightly. More solution was then applied. No symptoms at all were produced by this treatment.

Evidence that licking was not the cause, and that absorption was through apparently sound skin was furnished by the next experiment.

Experiment 5

A young black-and-white zebu ox (No. 6459) in good condition and with a skin that was sound, except for firm brand-scars, was sprayed with a mixture that contained 0·4 per cent. of nicotine sulphate precipitated by excess of wattle extract. The mixture contained also derris powder (1 per cent.), soft soap (0·25 per cent.), and used engine oil (1 per cent.); none of which is believed to be toxic in the proportions used, though individually or collectively they may enhance the toxic action of the nicotine. He was sprayed thoroughly except on the head. Two litres were used; the mixture in the bucket was well stirred throughout. Spraying was completed at 10.13 a.m., 20th October, 1941, but the animal was kept tied up so that he could not lick his body. By 10.35 he was grinding his teeth, salivating, and passing small quantities of dung. He died at 11.40 after showing symptoms of acute nicotine poisoning.

Post mortem. — Cyanosis; engorgement of subcutaneous vessels; contraction of spleen; poor clotting of blood; flabby, empty heart; flaccid œsophagus; hæmorrhagic periportal and mesenteric glands; large areas of intense inflammation in jejunum, with blood extravasation.

It is this hæmorrhagic enteritis that chiefly distinguishes Case No. 4 from the two other fatal cases, and one wonders if this were not due in part to the sensitizing effect of previous local applications of nicotine sulphate on 21:8:41, 26:8:41, 21:9:41 and 9:10:41 — none of which had set up any symptoms. The other animals had not had nicotine applied previous to the one fatal spraying. The other distinguishing feature is the absence of retraction of the eye-balls; most noticeable in the other cases. Nevertheless a general statement as to symptoms and lesions of acute nicotine poisoning after absorption through the skin may be made.

Symptoms

Nausea; sweating; salivation; greatly disturbed respiration and pulse; retraction of eye-balls; clonic spasms; incoordination; coma; death.

Lesions

Cyanosis; retraction of eyeballs; engorgement of subcutaneous and splanchnic vessels; poor clotting of blood; flabby, empty heart; contraction of spleen; lack of tone throughout alimentary tract.

Discussion

Search through the *Veterinary Bulletins* revealed few relevant abstracts, and these of articles inaccessible to us. De Leur (1934) describes how a nicotine spray for fruit trees was applied to the backs of four cows as a fly repellent with rapidly fatal results. Pieritz (1938) surveys known cases of nicotine poisoning in human beings and animals and contributes original work showing how the application of a weak nicotine solution to a horse for the purpose of killing lice set up typical symptoms. Lander (1926) gives few examples of nicotine poisoning, but makes the important statement that the drug is " readily absorbed through wounds, though not through the whole skin."

As we consider that our experiments, particularly No. 4, show that absorption can be through the whole skin, and as there is a paucity of literature regarding the toxicity of this substance, so widely used by agriculturalists, we feel justified in publishing this fresh evidence of the extreme toxicity of nicotine compounds.

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WATTLE DISEASE: A FORM OF CHRONIC FOWL CHOLERA IN NATAL

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In 1933 Henning and Coles reported the occurrence of fowl cholera in South Africa. Seddon (1914) was one of the first investigators to obtain an organism apparently identical with *Pasteurella aviseptica* from lesions of the wattles of fowls with this disease. Davis (1925) stated that *Pasteurella aviseptica* had been isolated from the wattles of birds with wattle disease in California. He also drew attention to the finding of *B. coli, Ps. pyocyanea* and *Staph. albus* in naturally occurring wattle oedema, but said that the injection of cultures of these organisms into the wattles of healthy fowls failed to

set up the disease. Thomas (1927) reported that wattle disease in Australia could be caused by fowl cholera strains of low virulence.

The object of the present note is to record the results of an investigation of an outbreak of wattle disease in a poultry plant near Pietermaritzburg, Natal. The history given by the poultryman was as follows:—

For approximately seven years the disease had occurred regularly, mainly amongst the cocks, but occasionally an old hen became affected. While the cocks were on free range nothing untoward was noticed, but many became affected within a short time of drafting to the breeding pens. No cases had been seen in cockerels that had been dubbed prior to their arrival in the pens. On the occasion of our first visit, four cocks were found showing typical lesions of wattle disease, while a number were apparently in the initial stages.

The plant was run on the intensive system. The houses were roomy and well ventilated, and the hygiene practised very fair.

Four cocks with wattle disease were brought to Allerton Laboratory for examination. The thick yellow material obtained on removing scabs from the wattle of one cock was mixed with saline and 0·5 cc. of the mixture was injected subcutaneously into each of two rabbits. Both died in less than 24 hours. Bipolar organisms were found in spleen smears. Cultures were then made in broth and on serum agar slants from the heart-blood and spleens of these rabbits. Numerous small dewdrop-like colonies developed on the serum agar. Sub-cultures made on ordinary agar and lemco agar were negative. The organisms were small ovoid Gram-negative bacilli and, when stained with Giemsa, some showed bipolar staining. Acid was produced in glucose and saccharose, but not in lactose, maltose or dulcite. Cultures on serum and blood agar soon died.

Discussion

The fact that this condition has been endemic on the farm for a number of years seems to indicate that there must be a number of carriers of the disease. If cocks that were only slightly affected were kept isolated and well fed they tended to recover with only slight permanent changes to the affected wattles. On the plant, however, such birds were invariably attacked and chased from the food receptacles by their healthy brothers, with the result that their vitality was reduced and the disease spread to other organs and eventually caused death.

That more cocks than hens were affected might be explained by the greater development of the wattles of the cocks. This is also the opinion of Davis and of Thomas. Thomas (1927) states that the organisms cannot pass through the unbroken epidermis of the wattle, but that when injuries due to fighting or any other cause occur, infection is likely to take place. On this plant the sharp edges of the

zinc on the mash hoppers were probably the cause of wounds on the wattles.

No further cases have been observed on the plant this year, as all cocks were dubbed prior to being placed in the breeding pens. This, together with good hygiene, seems to be the most practical method of preventing the appearance of the disease.

Summary and Conclusions

From the investigations it appears that wattle disease is more common in cocks than in hens. From one case of this disease a bipolar organism was isolated by rabbit inoculation, the rabbits dying readily. The bipolar organism appeared to be *Pasteurella aviseptica*. It was not possible to obtain cultures of this organism directly from the lesions. Removal of the comb and wattles was found to be the most practical method of preventing cases from occurring and also the best form of treatment. It was not proved that the bipolar organism was responsible for the wattle disease, but its isolation, coupled with the reports of other investigators, suggests that the cases were endemic fowl cholera.

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THE GESTATION PERIOD OF *PROCAVIA CAPENSIS* (DASSIE).

G. N. MURRAY
Onderstepoort

In his article " On the Size of the litter and the Gestation Period of *Procavia capensis* " van der Horst (1941) mentions that nothing is known about the length of the gestation period of this mammal. Therefore it may be of some interest to record a case that was observed by the writer.

In 1932 there was a practically full-grown female dassie at Onderstepoort; that is, she could not have been younger than one-and-a-half to two years. She became quite tame and roamed about the

place at will. In 1937 a young male was obtained. In November 1939 the old female gave birth to one young (♀). She had therefore become pregnant for the first time when she was about nine years old. The advanced stage of development of the young one at birth was remarkable. A quarter of an hour after birth it was very active and running about with its mother.

On the 3rd April, 1940, it was observed that the male and the old female copulated. When several months had elapsed and no sign of pregnancy was observed, it was thought that the female was not pregnant. It was extremely difficult to palpate the abdomen on account of the well developed abdominal muscles. On the 21st November, 1940, one young (♀) was born. At the time it appeared unlikely that this small animal (live weight 2·5 kg.) could have such a long gestation period. It was thought more likely that the female had again mated at an oestrus subsequent to that on the 3rd April, 1940. However, when this case and van der Horst's observations are taken together, it is clear that the gestation period of *Procavia capensis* is about $7\frac{1}{2}$ months. This female was at least 10 years old when she had her second parturition.

The old female has since died, but shortly before her death she weighed 2·5 kg. The male, which is now about four years old, weighs 4·0 kg., the young female born November, 1939, weighed 3·1 kg. when one year and eight months old, while the one born November, 1940, weighed 1·7 kg. when eight months old.

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VAN DER HORST (1941). The size of the litter and gestation period of *Procavia capensis*. *Science* 93 (2418) : 430-431.

OBITUARIES.

STANLEY ELLEY

Elley qualified at the New Edinburgh Veterinary Collge in 1903 and then joined the C.V.D. of the Cape Colony. However, he soon left the Government service and took up farming in the Oudtshoorn district. He married Miss Maggie Olivier, daughter of Mr. Gert Olivier, a farmer, and former Member of Parliament for the district.

On the outbreak of the Great War, Elley joined the S.A.V.C. and saw service in German South-West and in German East Africa. In 1919 he was released from service and returned to his farm.

Although Elley did not practice his profession, he was very interested in animal husbandry and was well known as a breeder of race horses.

H.H.C.

ARCHIBALD HODDER

Hodder studied at the Dick Veterinary College, where he qualified in 1898. Professor Dewar, in a testimonial, said that Hodder was an intelli-

gent student and that he had artistic ability. Professor Dewar was often assisted by him in preparing diagrams for teaching purposes. An essay on "Cruelty and its prevention" obtained for Hodder the award of a silver medal from the Scottish S.P.C.A.

After a short time in practice, Hodder joined the A.V.D. as a civil veterinary surgeon in 1899. In 1901 he transferred to the S.A.C. as veterinary lieutenant. In 1903 he rejoined the A.V.D. and after serving in several units left the army in 1904 and settled in Bloemfontein to practise.

From 1905 to 1906 he served the Natal Government and, when the Zulu Rebellion broke out in 1906, became a lieutenant in the Natal Veterinary Corps until the end of the campaign. Hodder then settled in Pietermaritzburg — where he sometimes acted as judge for the Royal Agricultural Society — until the outbreak of the Great War. He joined the S.A.V.C. as a captain and served first in South-West Africa and later in Nyasaland. While in East Africa he painted about 50 water colours that were subsequently bought by Mr. Howard Pim of Johannesburg.

After leaving the Union Defence Force, Hodder settled in Johannesburg in private practice. He died at the East Rand Hospital on the 7th of November, 1937. Thanks to Mr. A. M. Diesel, a scrapbook containing much interesting information about our earlier veterinary history has been preserved.

H.H.C.

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GAS GANGRENE IN THE DOG

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On hypothetical grounds one would expect gas gangrene to occur more frequently in the dog than it actually does. Wounds, grossly infected bacteriologically, are common, yet the disease is not often seen in everyday practice as a sequel to such wounds. One is tempted to suggest that the dog has a high natural resistance.

St. Simon (1931) reported that the dog is very resistant to *Clostridium chauvoei* or *Cl. chauvoei* plus staphylococci, but that it is somewhat susceptible to *Cl. septicum*. He isolated *Cl. welchii* (perfringens) from the muscles of three normal dogs. Saxinger (1931) noted six cases of gas gangrene after bites. He did no bacteriological work, but advised the widening of the wound canal, the cleaning up of the edges of the wound, thorough disinfection and the injection of large doses of antitoxin. Andrews, Rewbridge, and Hrdina (1931) caused the death of dogs from a *Cl. welchii* infection when they injected, by the intraperitoneal, intrathoracic, or intramuscular routes, bile salts, bile, or sterile liver extract; they say that *Cl. welchii*, normally present, flares up when tissue damage occurs. Gage (1932) produced a gas bacillus infection in five dogs by introducing wool cloth into the traumatized thigh muscles; he made no mention of deaths. Trusler and Reeves (1934) failed to isolate *Cl. welchii* from the livers of 16 normal dogs or from the muscles of six. However, from the livers, a strict anærobe, and from the muscles a less strict anærobe were isolated; both were large Gram-positive rods. Bottin (1935) produced gas gangrene by introducing the pancreas of normal dogs into the peritoneum or muscle of dogs; *Cl. welchii* was demonstrated. Ascoli (1937) recorded two cases of gas gangrene in the dog and demonstrated Gram-positive bacilli with subterminal spores in the affected muscles.

The experimental work reported in this communication was carried out in 1930 at the Wellcome Physiological Research Laboratories, Beckenham, Kent, in order to gain some idea of the susceptibility of the dog to experimental infection with the commoner gas gangrene clostridia.

Experimental Procedure

Strains used:

Cl. septicum: "K.F.," isolated from the muscle of a bovine that died of blackquarter; produces a potent exotoxin,

Cl. welchii: "S.R. 12," from the National Collection of Type Cultures, London; produces a potent exotoxin.

Cl. histolyticum: "2," origin unknown; produces a potent exotoxin.

Cl. oedematiens: "E," origin unknown; produces a potent exotoxin.

Cl. chauvoei: "Forbes," isolated from the muscle of a bovine that died of blackquarter.

Cultures Injected Intramuscularly

The strains were incubated at 37°C in 15 cc. quantities of Robertson's meat broth (horse flesh broth plus 2% Fairchild's peptone and 0·5% sodium chloride, meat particles half by volume), for three to five hours in the case of *Cl. welchii* and for 24 hours in the case of the others. To stimulate the growth of *Cl. chauvoei* 5 per cent. sterile sheep serum was added to the medium.

The dogs, mongrels weighing between 10 and 20 Kg., received the culture intramuscularly (i.m.) in the thigh. They were observed until they were moribund (when they were killed by chloroform) or dead, or until recovery. Guinea-pigs were inoculated intramuscularly at the same time with the same culture, in order to obtain some idea of the virulence of the inoculum. The results are given in table 1.

The number of dogs used was too small to allow of definite conclusions, but the results show that the dog is susceptible to each of the five anærobes used. The impression was got that it is not highly susceptible, e.g. 1 cc. of *Cl. welchii* failed to kill dog 2, and 1 cc. of *Cl chauvoei* failed with dog 7; experience has shown that 1 cc. of the culture of *Cl. chauvoei* used represents between 10 and 100 fatal doses for a sheep. Dog 4, which received *Cl. histolyticum*, would probably have recovered with nursing and treatment, but was killed because of the ugly-looking wound that resulted when the swelling burst. But for this, the animal appeared normal.

Cultures Injected into the Gums or Fed by Stomach Tube

This work was part of an investigation of an ulcerative disease of the gums, lips, and cheeks of young dogs held in isolation for distemper research. An attempt was made to transmit the disease to healthy pups; spirochætes were looked for; anærobic culture work was carried out; and finally, the effect of injecting cultures of anærobes into the lips, and of feeding cultures ,was investigated. The inoculation was made, as for an intradermic injection, into the buccal mucous membrane of the lip. Table 2 summarizes the results.

Discussion and Summary

The results, whilst showing quite definitely that the dog is susceptible to artificial infection with the commoner gas gangrene organisms, do not indicate a high degree of susceptibility. Large doses

TABLE 1

Cultures of Anærobes injected intramuscularly into Dogs

Inoculum	Result	Smear from local lesion and culture	Post-mortem Examination	Control Guinea-pigs
Cl. septicum, 1 cc. i.m. dog 1.	Moribund o/n, killed.	Sporing Gram+ bacilli, apparently in pure culture. No growth in M.B. from heart-blood or liver.	Site of inoculation very swollen and dark red in colour; s.c. tissues and muscles of leg and abdomen dark red, oedematous and crepitant; no apparent internal lesions.	0.5 cc., 0.25 cc., 0.1 cc., 0.05 cc., all † o/n.
Cl. welchii, 1 cc. i.m. dog 2.	1 day: leg swollen to hock; dog quiet but not unhappy. 2 days: swelling less. 3 days: swelling rapidly disappearing. 5 days: swelling almost disappeared. 6 days: swelling gone.			0.5 cc. † o/n, 0.25 cc. † 48 hrs., 0.1 cc. † 3 days, 0.05 cc. † 3 days.
Cl. welchii, 1 cc. i.m. dog 3.	1 day: leg swollen to hock and very painful; dog refusing food but not water; lying down. 2 days: moribund, killed.	Cl. welchii-like bacilli, apparently in pure culture; Cl. welchii isolated in pure culture from heart-blood and liver.	Fulminating gas gangrene extending over abdomen and chest and down to hock; muscles dark red, oedematous and crepitant.	0.1 cc. † o/n, 0.05 cc. † 48 hrs., 0.025 cc. † 48 hrs.
Cl. histolyticum 1 cc. i.m. dog 4.	1 day: large swelling; dog bright but quiet. 2 days: as 1 day. 3 days: swelling slightly smaller. 5 days: swelling burst; dog killed.		Groin swollen; skin greenish and digested; no apparent digestion of muscles; muscles red but not juicy; lesion limited to 7 cm.—8 cm. around injection site.	0.5 cc. killed o/n; muscles digested to bone but g.p. lively. 0.25 cc. killed 48 hrs. as 0.5 cc. 0.1 cc. † 48 hrs. 0.05 cc. † 48 hrs.

TABLE 1 — (Continued)

Cultures of Anærobes injected intramuscularly into Dogs

Inoculum	Result	Smear from local lesion and culture	Post-mortem Examination	Control Guinea-pigs
Cl. oedematiens 1 cc. i.m. dog 5.	1 day: very painful swelling. 2 days: swelling very large and painful. 3 days: swelling very large and painful, dog killed.		Leg very swollen and on section of muscle a clear limpid fluid exuded. No reddening or gas.	0.1 cc. leg swollen, survived. 0.05 cc. leg swollen, survived. 0.025 cc. no reaction. 0.01 cc. no reaction.
Cl. oedematiens 2 cc. i.m. dog 6.	1 day: very large, very painful swelling of thigh with oedema involving hock and leg; dog very ill, lying down and refusing food. 2 days: dog dead.	No growth in M.B. from heart-blood or liver.	Muscles of thigh very dark red, oedematous, the oedema extending to paw; a gelatinous gassy oedema to breast.	2.0 cc. † o/n, 1.0 cc † o/n, 0.5 cc. † o/n.
Cl. chauvoei, 1 cc. i.m. dog 7.	1 day: swelling, mainly at hock; dog quiet but lively. 2 days: swelling situated mainly around site of injection. 3 days: swelling disappearing rapidly. 5 days: swelling disappearing rapidly. 6 days: swelling small. 8 days: swelling nearly disappeared.			1.0 cc. † o/n, 0.5 cc. † o/n, 0.25 cc. † 3 days, 0.1 cc. † o/n.
Cl. chauvoei, 2 cc. i.m. dog 8.	1 day: painful, soft swelling at point of inoculation; dog refusing food, dull. 2 days: dog very ill, killed.	Cl. chauvoei isolated from heart-blood and liver.	Thigh muscles dark red and oedematous; oedema to paw; no gas in muscles; lesion confined to inoculated leg.	2.0 cc. † 48 hrs., 1.0 cc. † 48 hrs., 0.5 cc. † 3 days.

(o/n = overnight; † = died; hrs. = hours; i.m. = intramuscularly; M.B. = meat broth; s.c. = subcutaneous; g.p. = guinea-pig.)

TABLE 2

Cultures Injected into the Gums or Fed by Stomach Tube

Dog	Culture	1 Day	5 Days
9	Welch, 0.01 cc. into lip.	N.R.	N.R.
10	Welch, 0.1 cc. into lip.	N.R.	N.R.
10a*	Welch, 0.1 cc. into lip.	swelling.	sw. nearly gone.
11	Oed., 0.01 cc. into lip.	swelling.	sw. nearly gone.
12	Oed., 0.1 cc. into lip.	marked sw.	sw. nearly gone.
13	V.S., 0.01 cc. into lip.	N.R.	N.R.
14	V.S., 0.1 cc. into lip.	swelling.	sw. nearly gone.
15	Hist., 0.1 cc. into lip.	swelling.	sw. nearly gone.
16*	Welch, 40 cc. fed.	N.R.	N.R.
17	Hist., 70 cc. fed.	N.R.	N.R.

(* Dogs 10a and 16 received a strain of *Cl. welchii* isolated from a lip ulcer of a naturally-infected dog; Oed. = *Cl. oedematiens*; V.S. = *Cl. septicum*; Hist. = *Cl. histolyticum*· N.R. = No reaction; sw. = Swelling.)

(1 cc. to 2 cc.) of virulent culture caused swelling, or gas gangrene and death when they were injected intramuscularly, whereas smaller amounts (0·01 cc. or 0·1 cc.) inoculated "intradermically" into the gums produced no reaction or transient swellings only. Doubtless the route and site were unfavourable in this case for the development of the microbes. The administration by stomach tube of 40 cc. of *Cl. welchii* culture or of 70 cc. of *Cl. histolyticum* culture produced no apparent ill-effect.

No attempt at treatment was made, but there is no good reason why, in practice, that carried out in man should not be adopted — incision, thorough cleansing, removal of diseased muscle, liberal use of sulphonamides both in the infected area and *per os*, and the injection of large doses of a polyvalent (welch, septicum, and oedematiens) antitoxin.

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A NOTE ON BIRCH TAR — A SUBSTITUTE FOR STOCKHOLM TAR

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Onderstepoort

Owing to war conditions a substitute for Stockholm tar has appeared on the market.

Tests, prescribed in the British Pharmaceutical Codex (1934), revealed this substitute to be birch tar obtained by the destructive distillation of the wood and bark of *Betula alba* Linn, whilst Stockholm tar is obtained in a similar manner from the wood of *Pinus sylvestris* Linn. and the other members of the *Pinaceae*.

Repeated applications (twice daily on three successive days) of birch tar to the buccal mucous membrane and skin (inguinal region) of sheep failed to reveal any irritant action. Quantities of 50 cc. and 100 cc. of birch tar, administered by stomach tube, caused only transitory anorexia in sheep. Quantities of 200 cc., however, had a lethal effect, the following being the symptoms and post-mortem lesions observed —

Symptoms. — Apathy, anorexia, diarrhœa, dyspnœa, and an accelerated and weak pulse.

Post-mortem appearance. — General cyanosis; hyperæmia and oedema of the lungs; oesophagitis; stasis of the rumen; hyperæmia of the mucosa of the abomasum and small intestine; slight hyperæmia of the mucosa of the cæcum.

For full-grown sheep 15 cc. of birch tar could be regarded as safe when administered in one dose. When the administration is repeated, doses not exceeding 5 cc. should be given at a time.

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STREPTOCOCCUS MASTITIS IN HEIFERS

S. W. J. VAN RENSBURG
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It is now generally accepted that:—

(a) *Streptococcus agalactiae,* the commonest cause of streptococcus mastitis in cows, is an obligatory parasite which cannot maintain itself outside the animal body for any length of time; (b) infection can only be produced in a herd by the introduction of an infected animal; (c) infection is spread from cow to cow by mechanical methods such as the hand of milkers, teat-cups, flies, direct contact of teats with infected stable floors, etc.; (d) invasion of the udder takes place only through the teat orifice, the organisms being drawn up into the sinus through the teat canal as a result of the suction created by the act of milking or sucking. Attempts to produce infection of the udder by the administration of the causal streptococci *per os* have failed consistently, and infection can be set up artificially via the teat canal only and by no other method.

Consideration of these factors justifies the assumption that all heifers are free from mastitis streptococci prior to their first calving and that infection of their udders can only take place when they are exposed to the recognised methods of infection after the commencement of their first lactation period. It is logical therefore to presume that a mastitis-free herd can be established by the recruitment of heifers which are kept in isolation from the time they calve, and are not allowed at any time to come into contact with older infected cows. This is in fact one of the recognised methods of mastitis control. In many cases it has achieved the desired effect, but within recent years serious breakdowns have occurred in such "clean" herds and the source and method of infection in these cases frequently confound the worker concerned with the herd.

In the past many investigators have found that the incidence of mastitis in young cows is much lower than in older cows, and in heifers in the first lactation it has even been considered to be so low as to be negligible. As recently as 1929, Brigham, McAlpine and Anderson stated that mastitis very rarely, if ever, occurred in first calf heifers. Subsequent investigations have, however, shown that infection is far more prevalent in first calvers than was generally supposed. McLean and Hewitt (1935) considered it a remarkable feature that 25 per cent. of heifers examined by them in Victoria had streptococci in their milk within a month of calving. Stableforth, Edwards and Minett (1935) found that 23 out of 125 cows (18·4%)

in their first lactation were shedding *Streptococcus agalactiae* in their milk. They pointed out that " unless precautions are taken to protect heifers from infection as many as 30 per cent. may become infected in their first lactation." This statement is significant in that it suggests that the authors at that time assumed that infection in first calf heifers is invariably the result of such animals being exposed to infection only after parturition. In an examination of 3,000 cows in 1928 Seeleman found 3·5 per cent. infection in first calf heifers, while two years later the incidence had risen to 13·7 per cent. This is important in that it lends support to the opinion we have formed, namely, that there is a gradual but definite increase in the incidence of mastitis in heifers.

A fairly high percentage of infection in first calvers is conceivable when due regard is paid to the fact that in the herds studied by the various workers quoted above heifers were probably maintained in close contact with older cows and were therefore exposed to infection from the time of calving. It is evident too that the possibility of a pre-breeding infection did not present itself to these workers until 1939 when Minnett remarked that " it now seems that the incidence among heifers in their first lactation may be a good deal higher than was formerly believed, higher in fact in some herds than would be likely or possible if infection took place solely by the teat canal." He drew attention to the possibility of calfhood infection with *Streptococcus agalactiae* which might be retained in a latent manner to be revealed later when lactation began.

There is increasing evidence to support Minett's suggestion of infection being contracted prior to the first lactation. In a study of the presence of mastitis streptococci in bovine mammary tissue Hucker (1937) detected these streptococci in 47 per cent. of the udders of 21 virgin heifers which were included in his investigations. Mattick, Shattock and Moreira (1941) found that 17 first calvers of 377 heifers (4·5%) which were kept in isolation gave positive results within 10 to 20 days of calving. In an attempt to build up a clean herd *de novo* by selecting non-pregnant heifers for experimental purposes, Bull and co-workers (1940) in Australia placed 30 such heifers in isolation on a farm after the pastures, milking shed and surroundings had been rested for a period of ten weeks. Notwithstanding these precautions *Streptococcus agalactiae* was recovered from 16 of 119 functioning quarters during the first week of the first lactation.

Observations made on three herds (A, B and C) under our control offer an interesting comparison. (Fuller details will appear in the Onderstepoort Journal.)

(A) is an old established herd which has been badly infected with mastitis for ten years or more. It was taken over by us for control purposes 18 months ago, and is composed of approximately 100 cows. The first test for mastitis, consisting of both cultural and

microscopic examination of milk samples, revealed an 88·8 per cent. infection. When due allowance is made for udders in the cultural latency stage it will be no exaggeration to regard this as close on 100 per cent. infection. It is obvious that the milk derived from this herd must show a very severe contamination with mastitis streptococci, and since there has been no pasteurization or other precautions taken to provide calves with clean milk it is apparent that for some years now the calves in the herd have been reared on mastitis milk.

The control measures instituted are, briefly: (a) elimination of all cows showing advanced induration of the udder; (b) application of infusion therapy to the remainder; (c) building up of a clean herd from heifers. For the last purpose all virgin heifers are isolated in a paddock and at no time are they allowed to come into contact with other cows. A separate water supply is provided for them and they are not even allowed to travel over a road used by other cattle. A new milking shed and calf pen were constructed in another isolation paddock into which all heifers about to calve are removed. These are milked by separate attendants and are tested for mastitis within the first week of calving. Reactors are immediately removed to the infected herd for treatment. Subsequent to calving and prior to the first test the heifers are milked in another isolation shed in order to prevent them spreading infection to the others in the event of their discharging mastitis streptococci in their milk.

Up to the present 24 of these heifers have calved. Fifteen of the 24 were shedding mastitis streptococci at the first test within a week of calving. Five others that were negative at first have since revealed infection. The remaining four which calved three to four months ago have thus far given negative results, but in view of the short interval one is not justified in concluding at this stage that they are clean. Thus, notwithstanding the elaborate precautions that were taken to protect these heifers against mastitis, 62·5 per cent. showed infection within a week of calving and another 20·8 per cent. within six weeks. In view of the strict isolation and the possibility of a dormant infection in the five that were negative when first tested, it is highly probable that 20 out of 24 (83·3%) were actually infected at the time of calving.

Herd B has also been under our control for 18 months. Compared with A, this can be regarded as a moderately infected herd, the disease being detected in 40 per cent. of the animals in milk at the first test when control measures were instituted. The herd consists of approximately 150 animals and there were 30 heifers in various stages of their first lactation when the first test was applied. Of the 30, 18 (60%) were found to be infected. Twelve months after the introduction of proper hygienic measures, only eight out of 40 (20%) first calvers were infected.

The important difference between A and B herds is that in the

former stricter quarantine was imposed and animals were tested within a week of calving, whereas in B the heifers were grazing and being milked together with the infected cows, and in many of these the first lactation was far advanced before they were subjected to the first test. It can therefore be surmised that a large portion of the 18 infected heifers in B contracted infection after calving. The reduction in the incidence of infection from 60 per cent. to 20 per cent. in B after the institution of proper hygiene not only indicates that control measures were very efficient but also suggests that in the 18 heifers that reacted to the first test infection was probably contracted after calving in the majority, and prior to calving in most of the eight reactors of a year later.

C is an experimental herd which was recruited from heifers heavy in calf. The 20 animals which compose the herd were purchased from various owners three years ago. There is unfortunately no history available with regard to the prevalence of mastitis in the herds from which they were derived, but as they were mainly obtained from breeders in country districts it can reasonably be assumed that the incidence of the disease in the original herds was not high. When they were brought on to this station after having passed the tests for tuberculosis and contagious abortion the heifers were put into an isolation camp specially provided for the purpose. All of them calved within six months of their introduction. During the three years four have died of intercurrent disease while the remainder are now all in their third lactation. The cows are milked in a specially constructed milking shed by attendants who do not handle any other cattle. Milk from the individual quarters is subjected to bacteriological, microscopic and biochemical tests for mastitis within the first week of calving and subsequently at regular four-weekly intervals throughout the lactation period.

During the first two lactations all these cows gave consistently negative results for streptococcus mastitis. Then four of them were transferred to another camp, where they are now kept in contact with infected cows. The remaining twelve have since been augmented by two of their own heifer calves which are now in the first lactation, and the 14 are still consistently giving negative results.

As far as the prevalence of infection in heifers is concerned, the results which we have obtained from herd C are therefore in direct contrast with those achieved by Bull and co-workers who appear to be working under identical conditions and found *Streptococcus agalactiae* appearing frequently in the milk of their animals right from the commencement of the first lactation. Unfortunately the history of the Australian animals prior to their isolation as non-pregnant heifers is not given, and a knowledge of the calf history of both herds might be of great assistance in any attempted explanation of the unexpected appearance of mastitis streptococci in the one herd and the freedom

from infection in the other. One would, however, venture to suggest that if the desired information was available it would probably indicate that the Australian animals were derived from herds with a fairly high incidence of the disease while we were fortunate in obtaining heifers from herds with only a light or perhaps no infection at all.

The conclusion that must be drawn from the observations made on herds under our control and from the findings of Hucker, Mattick, Bull and others is that the presence of streptococcus mastitis in first calf heifers cannot invariably be the sole result of infection contracted after the commencement of lactation. The evidence suggests that in many cases infection in heifers takes place at an early age, and thus lends support to Minett's theory of calfhood infection. Further, it appears that the incidence in heifers varies directly with the severity of the disease in the herds from which the heifers are derived.

This raises the very important question of the method of infection in young heifers in view of the voluminous evidence to the effect that the disease can be set up experimentally only through the teat opening. One objectionable habit, which may be responsible for producing infection in heifers, is the suckling of one another by calves. The responsible streptococci have been recovered from the mouth of calves fed on mastitis milk and it is therefore conceivable that such calves may introduce the organisms into the teat canal of heifers by sucking them.

While this is a possible method of infection it cannot be regarded as the sole factor in such widespread contagion as is present in herd A. One is therefore compelled to advance a theory of an oral infection in calves notwithstanding the inability to produce the disease experimentally by this route. Under natural conditions calves in affected herds are fed mastitis milk continuously for several months. In South Africa it is the custom with many farmers and dairymen to use the milk derived from diseased udders for feeding calves and pigs. Moreover, with many, putting calves on to such infected quarters is part of the treatment for mastitis. It is reasonable therefore to assume that such animals will contract the infection *per os* in spite of the negative results obtained by sporadic experimental administration of the organisms by that route.

Further research is necessary in order to determine definitely the route and time of infection in heifers with a view to testing the above theories. Such work is now being undertaken, but will take several years to complete. This, however, is an academic question. From the practical aspect evidence has been produced to indicate that infection in heifers may assume serious proportions under certain conditions.

The implications of this are wide and entail a revision of our methods of building up clean herds. Instead of starting such herds with heifers they will have to be recruited from calves which are reared on clean milk. The most important factor from the practical and the

economic points of view is the necessity for ensuring that heifer calves are reared on either mastitis-free or pasteurized milk. Unless this is done owners of infected herds must expect the disease to spread with increasing rapidity, and in the course of time to be just as prevalent in newly calved heifers as in older cows. As a result all efforts to establish clean herds from heifers reared in badly infected herds will be nullified.

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" BLOUWILDEBEESOOG "

J. M. FOURIE and P. S. SNYMAN
Government Veterinary Officer, Vryburg, and Senior Veterinary Officer, Bloemfontein

Definition. — " Blouwildebeesoog " is a disease of cattle, sheep, goats and horses, along the Kuruman and Molopo rivers and is contracted when they come in close contact with the blue wildebeest (*Connochaetes taurinus*). The disease is characterized by an exophthalmia, accompanied by blindness, in one or both eyes and in certain breeds of sheep nervous symptoms are also manifested.

History. — According to farmers along the Kuruman and Molopo rivers in the Kuruman district where the disease is locally known as " Blouwildebeesoog," literally, blue wildebeest eye, epizootics of this disease occurred in the years 1927, 1935, 1937, and again in 1941. It is maintained that the occurrence of the disease coincides with those dry seasons, when blue wildebeest migrate in thousands from the Bechuanaland Protectorate, southwards into the Union. At the time of the investigation the disease occurred on the farms along the Kuruman river and the area between the Kuruman and Molopo rivers, from a point more or less at the junction of the Mashowing and the Kuruman and extending for a distance of approximately 100 miles along the rivers.

Subsequently, the disease broke out a fortnight later on farms 80 miles to the east after the wildebeest had trekked in that direction. The disease remained strictly confined to the area into which the wildebeest had trekked and the heaviest infection occurred where the concentration of wildebeest was greatest. On one particular farm 63 karakul sheep were affected out of a flock of 500 grazing on that part of the farm which was overrun by wildebeest, while on the rest of the farm only three sheep out of a flock of 400 became affected.

These wild animals come in very close contact with the stock in the veld, and very often accompany the cattle to the kraals. On the farm Le Roux, one of the farms on which the disease was investigated, a young wildebeest mixed with the sheep and came to within fifty yards of the homestead. The wildebeest were, at the time of the occurrence of the disease, in such poor condition that hundreds were dying from poverty.

On the above mentioned farm, where the disease made its appearance a fortnight previous to the investigation, there were 50 dead and 110 sick out of a flock of 755 pure and cross-bred karakul sheep. Of the sick animals, 12 were *in extremis.* About 25 per cent. of the cattle were infected. One animal was *in extremis* and had both eyes affected; the rest were only affected in one eye.

Subsequent investigations and enquiries revealed that the disease occurred over a much wider area than that described above. It was seen on farms on the Molopo river about 120 miles north-east from the Kuruman river. It was also reported from Tsabon, Bokseputse and Khuis in the Bechuanaland Protectorate. In South West Africa the disease was encountered in the Sandfontein area on the border between the Gobabis district and Bechuanaland. The following very typical description of the disease was given by Mrs. Binding of the farm Mazeppa 198 in the Okahandja district, S.W.A., in a letter to the Government Veterinary Officer at Okahandja:

" The most striking symptom is the enormous bulging of the eye, which stands out. The animals turn round in a circle, have no appetite and mostly die. The sheep which are sick today are lying stiff on the side, but also show the strange swollen eye. From time to time they are struggling with the legs and if one tries to lift them up they fall down again and cannot stand."

From the information at our disposal the disease is associated with the presence of wildebeest, which is most likely the carrier, but does not become affected like domestic animals.

Animals affected. — Karakul, cross-bred karakul, Persian and Afrikander sheep are most susceptible. In one instance in a flock comprising 2,200 karakul and merino sheep in equal numbers, 65 karakul sheep were affcted and none of the merino sheep. Merino sheep and goats are fairly resistant, a few cases only were encountered in these animals and no fatal cases were reported. Cattle are very susceptible but contract the disease in a mild form and no mortality occurs. Horses are also affected, but only two cases were reported in the whole of the area.

Affected sheep showing bulging of the eyes.

Symptomatology

The incubation period is unknown.

Two forms of the disease occur, viz. (a) a form in which only ophthalmic lesions are seen, and (b) a form in which nervous symptoms appear in conjunction with the ophthalmic changes.

(a) *Ophthalmic form.*— In this form one or both eyes may become affected.

The first visible sign is a protrusion of the eyeball, which appears as though it is enlarged and being pushed out from the back. There is at this stage no conjunctivitis or keratitis. Later, the cornea develops a haziness which may turn blue and in some cases white. Sometimes the eyeball protrudes to such an extent that the eyelids cannot be closed. The cornea then becomes dry and necrotic and finally the eye bursts on account of the increased internal pressure. Hæmorrhage may also be noticed in the anterior chamber of the eye. The eyelids also become swollen, and very often the lower eyelids form distinct pouches. The swelling of the eyelids is more prominent in cattle than in sheep. The animal is usually blind in the affected eye and may also be completely blind in one or both eyes before any keratitis has developed. When the animal recovers from the disease, the swelling of the eye and eyelids disappears as quickly as it occurs. The keratitis persists for some time, depending upon the severity of the condition and may eventually disappear without leaving a blemish on the eye, provided no mechanical injury has been caused by the use of caustic eye-lotions. Rhinitis with a copious flow of mucus from both nostrils was seen in a few advanced cases.

Affected cow showing bulging of the eyes.

Ewe turning in circles.

(b) *Nervous and ophthalmic form.*—This form was seen only in the breeds that are very susceptible to the disease, viz. karakul and Afrikander sheep. The nervous symptoms may develop before or after the ophthalmic lesions have occurred. If they

develop before the ophthalmic changes the animal may suddenly drop, get up again, or remain prostrate, or it may start turning round although the eye is not visibly affected. An animal may have one or several of these attacks during the day. In fact, several cases were seen where the animals kept moving in a circle every time they were disturbed. Animals affected in this way move round in the opposite direction to that of the affected eye. An ewe, blind in the right eye without visible lesions, kept moving in a counter-clockwise direction. Some animals show ataxia, walk sideways, pick up one or both legs on the same side exceptionally high, and cannot keep the head still when standing. Affected animals are usually not able to feed or to drink water and are sometimes seen making futile attempts to do so. If the nervous symptoms develop first, then any or all of the ophthalmic lesions described above follow in a day or so. In cattle, horses, merino sheep, and goats, the lesions associated with the eyes are similar to those described above, but nervous symptoms or mortality were not encountered.

Prognosis. — Cattle, merino sheep and goats recover without any after effects, provided no caustic eye lotions have been used. In karakul, Persian and Afrikander sheep the prognosis is less favourable and all animals which develop nervous symptoms die after three or four days. It is often necessary to destroy animals which are rendered completely blind as a result of rupture of the cornea of both eyes. The mortality varies from 30 per cent. to 50 per cent.

Autopsy

No pathological changes were seen in the internal organs. The swelling and bulging of the eyes were due to hydrophthalmos and oedema of the peri-orbital tissues. In cases where the eye was visibly affected, the blood in the veins at the back of the eyeball and also in a portion of the supra-orbital vein was coagulated. The oedema of the eye and the coagulation of the blood was apparently due to venous stasis. The eyeball showed the alterations described above. When keratitis was present the conjunctiva was similarly affected, and when there was an exudate from the nose, the nasal mucous membrane was markedly congested. The brain and meninges were sometimes hyperæmic in advanced cases.

Histological examination of the affected eyes showed oedema and infiltration of cellular elements in the cornea. Iridocyclitis and adhesion in the anterior chamber were also found. The internal organs, liver, kidney, etc., showed no specific histological changes. In this respect the disease did not appear to be associated with snotsiekte or malignant catarrhal fever.

Differential Diagnosis

1. *Ophthalmia.* — Blouwildebeesoog is distinguished from ordinory ophthalmia by the prominent swelling of the eye, and occurrence of nervous symptoms, blindness in the absence of keratitis and conjunctivitis in the initial stages, and the causing of mortality in sheep.

2. *Snotsiekte and/or Malignant Catarrhal Fever.* — Apparently these conditions do not affect sheep clinically. In bovines it usually causes bilateral lesions in the eyes and is characterised by a very profuse nasal discharge, high mortality, and characteristic lesions in the liver, kidneys, central nervous system, and the upper part of the respiratory tract.

3. *Listerella Infection.* — The characteristic cuffing of the blood-vessels of the central nervous system pathognomonic for this disease was absent. For a more detailed consideration see p. 49, this Journal.

4. *Vitamin A Deficiency* does not occur where animals have access to green feed, bushes, etc. Examination of liver specimens from " blouwildebeesoog " cases failed to reveal a vitamin A deficiency.

5. *Starvation.* — " Blouwildebeesoog " must also be differentiated from cases of starvation where the fat in the orbit undergoes serous atrophy and causes bulging of the eyes and subsequently ulceration. This condition was observed by Dr. G. de Kock of Onderstepoort, in the Jansenville area in Angora goats during a severe drought (personal communication).

Summary

(a) A disease which affects sheep, goats, cattle and horses and characterized mainly by ophthalmia and, in certain classes of sheep, by nervous symptoms, is described.

(b) The disease is contracted when susceptible animals come in close contact with blue wildebeest (*Connochaetes taurinus*) which are apparently not affected, but probably act as carriers.

Acknowledgments

The authors wish to express their thanks to Mr. Hobday, Principal Veterinary Officer, Bechuanaland Protectorate; Dr. G. Schmid, Government Veterinary Officer, Okahandja, S.W.A.; and Mr. S. W. de Villiers, Government Veterinary Officer, Omaruru, S.W.A., for their response to enquiries and for reporting on the disease in their respective territories. Special thanks are due to Dr. G. de Kock, Deputy Director of Veterinary Services, for the interest taken in connection with the investigation of this disease, for conducting the histological examination on the material collected, and for valuable suggestions in connection with this publication.

SURVIVAL OF EQUINE WITH SEVERED JUGULAR VEIN

J. R. FREAN and M. DE LANGE
Ermelo

Case. — A few-days-old filly.

History. — One morning last spring a farmer brought in to Ermelo by motor lorry (a distance of about forty miles) the filly which had been discovered that morning staggering about in a weak condition, apparently from loss of blood from a jagged wound in the neck. The owner did not interfere in any way and simply loaded the filly into a lorry and brought her in.

Examination revealed a jagged wound in the lower left part of the neck across the jugular groove and dangling over the wound was from two to three inches of the distal portion of the jugular vein, in which a clot had formed. This length of vein is seen clearly in the accompanying photograph. The vein was completely severed and the proximal end had apparently withdrawn into the jugular groove. It was not visible and we, naturally, did not search for it.

Treatment consisted of ligation through the clot and suturing the wound. A week after the occurrence the owner informed us that a diffuse oedematous swelling had developed above the wound, extending to the parotid and submaxillary regions. This disappeared subsequently and an uneventful recovery occurred.

ABSTRACT

LISTERELLA ENCEPHALITIS

The disease Listerella encephalitis (circling disease) of sheep, cattle and goats is fully described by the author.* It is an acute specific infectious encephalitis of sheep, cattle and goats caused by a short, gram-positive bacterium of the genus *Listerella*. The disease occurs from December to June in the United States. The following extract from Olafson's paper should be of interest to members in the field.

Etiologic Agent and Pathogenesis of the Disease

The disease is due to a small gram-positive rod that resembles a diphtheroid but differs in being motile. *Listerella* has been proposed as the name for this genus.

Practically nothing is known about the natural habitat or distribution of the organism when not in the nervous tissue. On farms where outbreaks occur year after year, one wonders whether some of the animals may be carriers and harbour the organism. The disease may appear suddenly on a farm where it has not occurred before even though no new animals have been added.

The disease starts as a rhinitis. In affected flocks the organisms probably get into the nasal passages of most animals, but only exceptionally is the invasion deep and extensive with progression to the brain.

Symptoms

The affected animal is depressed, stands by itself, and is indifferent to its surroundings. A cow may show difficulty in finding its stanchion or in getting through a door. Later, the animal pushes against the wall or may lean against a post for hours. On the second or third day, the tendency to circle appears. Circling may be either to the right or left, but an individual circles in only one direction. Drooping of one ear and some degree of facial paralysis appear. Testing the tone of the lips by lifting them away from the gums often enables one to detect unilateral paralysis. Lack of tone or partial paralysis of an eyelid may be present. The muscles of the neck tend to pull the head to one side. If the neck is forcibly straightened the head tends to fly back to the original position when released. An animal may carry a wad of hay in the cheek for two or three days. An occasional animal shows excitement. About the third or fourth day the animal has difficulty in keeping on its feet, falls down, and cannot get up. Later it lies flat on its side and may show turning movements. The disease lasts for three to seven days. In affected flocks or herds the disease attacks from one to twenty per cent. It is unusual to see more than one case in a herd of cattle. The mortality is practically 100 per cent.

Lesions

There are no gross lesions in the central nervous system. The microscopic inflammatory lesions are marked and characteristic. Perivascular cuffs or infiltrations are many cells deep and show four types of cells in varying proportions. These are lymphocytes, monocytes, neutrophiles, and eosinophiles. The lymphocytes are most common and abundant. The most characteristic lesion is the presence of microscopic cellular foci in the parenchyma. These appear to be independent of the vessels and the cells forming them are chiefly neutrophiles. Bacterial stains show the organisms in these lesions but not in the perivascular infiltrations. In peracute cases,

diffuse neutrophilic infiltration and oedema occur in the parenchyma. Nerve cells adjacent to the inflammatory areas appear well preserved. Varying amounts of lymphocytic or neutrophilic meningitis are present. The extent and diffuseness of the lesions vary. In general, the brain stem from the anterior cervical cord to the thalamus is most severely affected. Lesions occur in the white matter of the cerebellum and the cerebellar meninges. Lesions have not been found in either the spinal cord or the spinal meninges. This disease is thus a bacterial encephalitis showing selective localization in the brain.

Differential Diagnosis

Few diseases have as typical and constant symptoms or are as readily recognized as circling disease. Only the peracute form with marked depression and short course offers any difficulty.

The *toxæmia of pregnancy* is the only common disease that needs to be differentiated from bacterial encephalitis. The seasonal incidence is the same and the two are of about equal duration. Facial paralysis and ear paralysis occur in encephalitis but not in pregnancy disease. Toxic pregnant ewes may turn the head to one side but after straightening the neck there is not the tendency for the head to fly back to the original position as seen in circling disease. Acetone is present in the urine of the toxic pregnant ewe from the beginning of the illness.

Feed-lot toxæmia or "overeating disease" causes nervous symptoms that might be confused with encephalitis. The history of heavy feeding and the short course of the illness (6—12 hours) aid in establishing the diagnosis.

G. DE K.

* Peter Olafson (1940) *Cornell Veterinarian* 30:141.

"BAYER" GOLF TROPHY COMPETITION, 1942

This year, thirty-two competitors entered the field to compete for the trophy presented by Messrs. Bayer Pharma. Play was keen and the winners are to be congratulated on their excellent performances. The players were the guests of the donors for luncheon and throughout the day. The club secretary is to be commended on his efficient organisation which ensured that the event was an unqualified success.

The prizes were presented by Dr. P. J. du Toit, Director of Veterinary Services, who congratulated the winners, and, in thanking Messrs. Bayer Pharma (Pty.) Ltd. for a most enjoyable day, paid tribute to the services rendered by 'Bayer' to each of the allied professions represented at the gathering.

The list of prize-winners is as follows:

Winners. — Dr. A. J. Broughton and Sgt. C. Timlin.
Runners-up. — Dr. J. G. A. Davel and Sgt/Maj. Stead.
Third Prize. — Major J. C. H. Stead and Capt. A. E. Davis.
Special Prize (best over 18). — Dr. J. F. Dippenaar and Sgt/Maj. Richardson.
Best Morning Round. — Dr. J. W. H. Casewell and Mr. R. du Toit.

THE ASSOCIATION

Council Meeting held at Polley's Hotel, Pretoria, on 19th May, 1942, at 3.30 p.m.

Present. — S. T. Amos (President), A. C. Kirkpatrick, A. D. Thomas, D. G. Steyn, R. Alexander, M. Sterne, G. de Kock and S. W. J. van Rensburg (Hon. Sec.-Treas.).

Apologies. — Letters of apology for non-attendance were submitted from J. L. Dickson, P. Snyman and N. F. Viljoen.

(1) *Minutes* of meeting held on 4.5.41 were confirmed.

(2) *Arising from these minutes*:

(a) *Finance*: Dr. Alexander (Chairman of Finance Committee) reported on the results of the Committee's investigations with regard to investments, auditing and bookkeeping.

Trustees: It was resolved that Finance Committee with the Hon. Secretary-Treasurer be trustees of the funds of the Association.

Students' Loans: It was reported that after consideration of all aspects the Committee had decided against establishing a Students' Loan Fund and was of opinion that advances to students should only be considered in exceptional circumstances. This was agreed to, the Committee to consider the terms and limitations to be imposed in such cases. The Committee recommended that an extra £50 p.a. be advanced to student A for the last two years of the course. Approved.

Subscriptions. — In view of the very satisfactory position of the funds of the Association the Committee suggested that Council consider the advisability of granting some concession to members either by allowing a substantial discount for the payment of subscriptions within a stated period or by a reduction in subscription. After discussion, however, Council decided that the reserve fund was not yet sufficiently strong to warrant such action. The need for strengthening the Benevolent Fund was particularly stressed, and it was decided to recommend to the next General Meeting that the allocation to this fund be raised from 5/- to 10/- per member.

(b) *Deputation to Secretary for Agriculture*: The President stated that he had received communications from the Dean of the Faculty and from Dr. de Kock. The latter elucidated certain points in the memorandum submitted by him. After full discussion on this and other aspects of the case Dr. Steyn proposed and Dr. Thomas seconded that the matter be dropped. This was carried by 3 votes to 1.

Dr. de Kock expressed his appreciation for the manner in which the President had handled this matter.

(3) *New Members.* — It was decided that acceptance of the following be recommended to the next General Meeting: F. E. Cavanagh, Miss R. Datnow, P. J. Goosen, J. P. Moll, V. R. Kaschula, N. R. Reid, J. D. Smit, S. L. Snyders, J. I. Taylor and F. J. Veldman.

(4) *Hormone Treatment of Racehorses.* — The President reported on the indiscriminate and unprofessional manner in which hormones were being administered to racehorses in certain areas. Dr. Steyn submitted a memorandum giving the most recent information on the physiological and pharmacological actions of desoxycorticosterone. After discussion Dr. Alexander proposed and Dr. Steyn seconded that " after full consideration of the report received on the indiscriminate and improper use of hormones

on racehorses in certain areas in South Africa, and after full consideration of the reports on the results of research work on the effect of hormones as revealed in the literature, this Council strongly condemns such indiscriminate and improper use of hormones." This resolution was passed unanimously.

Dr. de Kock and Mr. Kirkpatrick left the meeting at this stage.

(5) *Veterinary Practice in Pretoria.* — The Secretary submitted copies of correspondence which had passed between himself and the Dean of Faculty *re* private practice and the supply of material for tuitional purposes at Onderstepoort. This was approved of.

(6) *Standing Committees.* — Letters of resignation from the Editorial Committee were received from Drs. Sterne and Thomas. These were accepted with regret, and it was decided to place on record Council's appreciation of the services rendered by both these members during their long period of service on the Committee.

The meeting was advised that Dr. Robinson had consented to take the Editorship of the Journal, and he was duly elected to the Committee.

A letter was also received from Dr. Jackson resigning from the position of Hon. Librarian, and a report from the Chairman of the Library Committee was submitted. It was decided that letters be sent to Dr. Jackson conveying Council's appreciation of his services as Hon. Librarian and to the Director of Veterinary Services expressing thanks for providing a room for the library.

(7) *Witwatersrand Branch of S.A.V.M.A.* — A letter from Mr. M. H. V. Brown was submitted. This was to the effect that at a meeting of 17 Rand veterinarians held on 23rd April, 1942, it was decided to form a branch of the Association, and asking Council's official sanction for this. The formation of such a branch on the same lines as the Natal branch was unanimously agreed to.

(8) *Annual General Meeting.* — It was decided that the date and nature of the next General Meeting be left to the General Purposes Committee in consultation with the Director of Veterinary Services.

The meeting concluded at 6 p.m.

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

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THE EFFECT OF LARGE SCALE ACTIVE IMMUNIZATION AGAINST ANTHRAX

MAX STERNE, J. NICOL and M. C. LAMBRECHTS.

Introduction

There is no doubt that anthrax vaccines are effective under laboratory conditions, but the evidence of their value in the field is less satisfactory. Therefore the publication of statistics on large scale anti-anthrax inoculation is of more than academic interest. The highly developed nature of the field veterinary service in South Africa has enabled us to collect accurate data on the results of inoculating about five-and-a-half million cattle yearly. These data are presented in this paper.

Anti-Anthrax Inoculation in South Africa

Viljoen, Curson and Fourie (1928) reported on anthrax in South Africa up to 1925. They showed that in 1923 about 7,500 deaths were diagnosed as anthrax, but pointed out that this number was probably a vast understatement, as veterinary supervision was in general sketchy. The real incidence might well have been five to ten times that reported, for officials stationed in anthrax areas at that time say that the disease was often epidemic and that large numbers of animals and many human beings died. The years from 1921 to 1925 saw the beginning of an intensive campaign of inoculation against anthrax, and by 1925 a million doses of vaccine were being given to farmers yearly. The number of deaths reported from anthrax in that year was about 3,000.

Since 1925 the use of vaccine has spread widely and by 1936 about six million doses (5½ million for cattle) were being used annually. This means that from 1936 half the cattle in South Africa have been inoculated every year and, since the bulk of the inoculation is in the more heavily infected areas, the concentration of inoculated cattle has in parts become very high.

At present the amount of vaccine used remains at just under six million doses per year. The number of deaths from anthrax in the whole of South Africa reported in 1941 was under 700. The reduction is actually much greater than the figures show, because checking and control are more efficient than fifteen years ago.

The discussion that follows is mainly of anthrax in the Transkei —a part of the Cape Province—because the precise data available for this area give the inoculation the character of a very extensive experiment.

Anthrax in the Transkei

The Transkei has a cattle population of just under one-and-three quarter millions. It is divided into 27 magisterial districts, 26 of which are inhabited almost exclusively by Africans. The remaining district, Mt. Currie, is an area of prosperous and progressive European settlement. The veterinary policy, as regards anthrax, differs in Mt. Currie from that in the rest of the Transkei, so that this district will be considered separately.

The African inhabitants of the Transkei are backward in the Western sense, and live rather poorly on the products of their husbandry. Hygiene is primitive and the inoculation of the cattle was the only measure that could be taken against anthrax. It was impossible to apply other methods of control, such as the destruction of carcases or the disinfection of kraals. Thus any reduced incidence of anthrax can only be due to the inoculation or to secular changes in the anthrax bacillus or to changes in telluric conditions. However, there has been no change in climate and little in the conditions of living during these years nor has any change been detected in the characteristics of the anthrax organisms isolated. The type of cattle and their number have remained about the same from 1928 to 1941.

Before 1928 inoculation was carried out on infected locations only. The success of such a procedure depends on deaths from anthrax being reported expeditiously and on preventing movements of cattle from infected to non-infected locations and vice versa. Nicol (1933) suggested that better results would be obtained, with little extra trouble, if all the cattle in a large area were inoculated at the same time. Such a protected block, he suggested, would offer a powerful resistance to the spread or introduction of anthrax. After a trial of this 'block' system in one district in 1928, Nicol introduced it in eleven of the twenty-seven Transkei districts (cattle population 700,000) in 1929. It was gradually extended to the rest of the Transkei, until by 1934 all the cattle in the whole area (with the exception of Mt. Currie district) were being inoculated in May-June every year. The data relevant to the anti-anthrax campaign in the Transkei 1925-1941 are set out in table 1. Figures for the period before 1925 were given by Viljoen *et al* (1928).

Column five gives the number of outbreaks (see table for definition of outbreaks) recorded annually. In 1926 the veterinary staff was small and veterinary supervision not as thorough as it is now, so that the actual number of outbreaks is estimated to have been five to ten times that shown and thousands of deaths are known to have occurred. The recorded number of outbreaks remained roughly constant up to 1929. Actually this signifies a considerable decrease, because veterinary control improved greatly during those years, and a far

TABLE 1.

Anthrax in the Transkei (26 Districts) 1925 - 1941.

Year	* Total no. of cattle in the Transkei	No. of cattle inoculated	No. of smears examined	*** No. of outbreaks of anthrax	**** No. of deaths from anthrax
1925-6	1,292,000	261,000	32,000	354	
1926-7	1,376,000	235,000	41,000	372	
1927-8	1,441,000	475,000	56,000	433	
1928-9	1,506,000	1,062,000	83,000	351	
1929-0	1,560,000	945,000	88,000	100	
1930-1	1,614,000	1,034,000	114,000	84	
1931-2	1,591,000	1,258,000	119,000	64	
1932-3	1,564,000	1,290,000	120,000	62	69
1933-4	1,484,000	810,000**	138,000	79	100
1934-5	1,505,000	1,502,000	123,000	96	116
1935-6	1,554,000	1,554,000	143,000	143	227
1936-7	1,569,000	1,569,000	187,000	111	185
1937-8	1,544,000	1,549,000	236,000	39	57
1938-9	1,590,000	1,590,000	164,000	27	33
1939-0	1 619,000	1,606,000	210,000	24	28
1940-1	1,648,000	1,677,000	171,000	17	17

* Occasionally it appears as though more cattle were inoculated than existed. The explanation is that the cattle returns were not made up at the same time of the year that the inoculation was done and the discrepancy merely reflects a seasonal fluctuation in the total number of cattle.

** Only partial 'block' inoculation carried out this year.

*** An occurrence of anthrax is considered a 'new outbreak' if there has been no case of anthrax on the location or farm during the previous twelve months.

**** The figures for deaths prior to 1932 are too unreliable to be worth mentioning. The figures given here are accurate.

larger number of deaths were being notified. Column 4, which gives the number of smears examined annually, can be used to estimate the improvement in notifications. (In practice the submission of a blood, spleen, or gland-smear from a dead animal is accepted as a report of death and an elaborate system has been built up to ensure the submission of smears). In well-controlled native areas the number of smears submitted should be *at least* ten per cent. of the cattle population. In times of drought the number may reach fifteen per cent. In 1928, for example, 56,000 smears were examined from a population of 1,400,000. The expected number was from 140,000 to 210,000. Thus only one death in three or four was reported. From 1928 to 1933 there was a steady decrease in the number of outbreaks discovered and an increase in the number of smears received. Therefore the drop in the incidence of anthrax is probably more notable than the figures indicate. In 1934 a field checking-system was instituted that still further improved control. From 1935, therefore, the data are very reliable indeed. The control system is primarily organized for the eradication of East Coast fever.

From 1934 there was a second rise in the recorded number of outbreaks to a peak in 1935-1936. A similar rise occurred all over South Africa. The refinements introduced in the checking system may have been partly responsible for unearthing more occurrences of anthrax. On the other hand there had been a considerable diminution in the immunizing power of the vaccine—as judged by laboratory tests—in the three years before 1936. The reason was that the catastrophic outbreaks frequent in earlier years no longer occurred and so the public became more critical of reactions caused by the vaccine. The attempts made to avoid the production of these reactions resulted in a loss of potency. This was probably the main cause of the increase in anthrax. During this period many farmers complained that the vaccine no longer cut short outbreaks as it used to do. In 1936 a modified and improved vaccine—as judged by laboratory tests—was used in the Transkei and the downward trend commenced again. In the rest of South Africa the old vaccine was not replaced until later and the incidence of anthrax continued to increase until the improved vaccine was introduced.

Finally, in the Transkei in 1941, the number of outbreaks was 17 and the number of deaths 17. What evidence is there that the great reduction since 1928 was due to inoculation? Table 2 is a repetition of table 1, except that the eleven districts where 'block' inoculation was started in 1928 are compared with the remaining fifteen Transkeian districts. In the latter districts inoculation was done on infected locations only (the number of cattle inoculated varied between 40 and 60 per cent.) until 1934, when the 'block' system was extended to include the whole of the Transkei. Thus two areas are compared: in one all, and in the other about half of the cattle were inoculated. In table 3 the figures for the 11 districts—where 'block' inoculation has been carried out since 1928—are compared with those from Mt. Currie, where the farmers do most of their own inoculating and the 'block' system has never been introduced.

One is not justified in drawing inferences from the comparison of figures for outbreaks prior to 1930; for the data up to that time are not trustworthy, although they improved year by year. From 1930-1934 considerable reliance can be placed on the statistics. Since 1934 the control has been so comprehensive and effective that the figures given can confidently be accepted as reflecting the actual state of affairs. It is, however, permissible to assume that the degree of infection was originally about the same in both areas, for the separation of the Transkei into the two areas was for administrative purposes only, and there was no difference in any significant respect. Moreover, this separation ceased in 1934, when 'block' inoculation was extended to the whole 26 districts. It appears from these figures that the area where intensive inoculation was first started showed the greater decline

TABLE 2

Anthrax in the Transkei, 1925-1941

Comparison of Results obtained in Eleven Districts where 'Block' Inoculation was Started in 1928 *with Fifteen Districts where 'Block' Inoculation was not Completely in Force until* 1934

Year	Total no. of cattle in		No. of cattle inoculated in		No. of outbreaks of anthrax in		* No. of deaths from anthrax in	
	11 dists.	15 dists.	11 dists.	15 dists.	11 dists.	15 dists.	11 dists.	15 dists.
1925-6	593,000	699,000			184	170		
1926-7	638,000	738,000			170	202		
1927-8	657,000	785,000			248	185		
1928-9	679,000	827,000	697,000	461,000	236	115		
1929-0	693,000	867,000	647,000	297,000	60	40		
1930-1	717,000	897,000	658,000	376,000	36	48		
1931-2	710,000	880,000	703,000	555,000	34	30		
1932-3	686,000	877,000	715,000	515,000	12	50		
1933-4	659,000	827,000	358,000	452,000	3	76		
1934-5	670,000	835,000	670,000	832,000	19	77	21	95
1935-6	687,000	867,000	688,000	866,000	44	93	64	163
1936-7	685,000	883,000	685,000	883,000	26	85	39	146
1937-8	677,000	867,000	677,000	872,000	10	29	23	34
1938-9	701,000	890,000	701,000	890,000	10	17	11	22
1939-0	711,000	908,000	730,000	877,000	9	15	11	17
1940-1	719,000	929,000	728,000	999,000	4	13	4	13

in the incidence of anthrax. Both areas showed the second peak in 1935-6 which was probably due to a deterioration of the vaccine.

TABLE 3

Anthrax in the Transkei, 1925-1941

Comparison of Results obtained in Eleven Districts where "Block" Inoculation was started in 1928 *with Results in Mt. Currie District where no 'Block' Inoculation has been carried out.*

Year	No. of cattle in		No. of outbreaks of anthrax in		No. of deaths from anthrax in	
	11 districts	Mt. Currie	11 districts	Mt. Currie	11 districts	Mt. Currie
1925-6	approximately 700,000	approximately 70,000	184	5		
1926-7			170	6		
1927-8			248	2		
1928-9			236	1		
1929-0			60	1		
1930-1			36	1		
1931-2			34	3		
1932-3			12	1		
1933-4			3	2		
1934-5			19	3	21	3
1935-6			44	16	64	17
1936-7			26	19	39	29
1937-8			10	17	23	20
1938-9			10	4	11	6
1939-0			9	4	11	6
1940-1			4	2	4	2

When interpreting the figures one must realise that, since 1934 at least, smears have been examined from practically every animal that died in the 11 districts. From Mt. Currie only about 600 smears are received annually—about a tenth of the probable number of deaths. Thus, since about 1934, and probably since 1932, a far higher proportion of the actual number of deaths from anthrax has been diagnosed in the 11 districts than in Mt. Currie. Previous to this, when control was poor in both areas, a relatively greater proportion of diagnoses would have been made in Mt. Currie, as this is a civilized district compared with the rest of the Transkei. Thus the relative increase in anthrax in Mt. Currie is probably far greater than we can show. It should also be remembered, when discussing these tables, that a marked diminution in anthrax in a large area would affect the neighbouring areas. Mt. Currie, although possessing the advantages of prosperity and civilization, is more heavily infected with anthrax than the backward districts in the rest of the Transkei.

Anthrax in Natal

This Province has a cattle population of between two-and-a-half and two-and-three-quarter millions and is another example of a well-controlled area. It has extensive and advanced European-inhabited areas—some of the most improved farming areas in the country—as well as very large native reserves. The number of cattle on European and on Native-owned farms is about equal. Anthrax was never as alarming in Natal as in the Transkei, but it threatened to become serious.

The campaign has been conducted differently from that in the Transkei. In parts of this province, where detailed supervision is difficult, 'block' inoculation is carried out. In other parts, where a careful check is possible, inoculation is done when a death from anthrax occurs or when there have been deaths from anthrax on the farm in previous years. This plan is possible because, owing to the presence of East Coast fever, there is a rigid control of cattle movements and because smears are submitted from every animal that dies. The veterinary staff is large and the control very efficient, so that it is impossible for anthrax outbreaks to remain undetected or to get out of hand. About half the inoculation is on the 'block' system.

Table 4 shows the progress of this campaign.

There was a rise in the number of outbreaks up to 1932. This was partly due to an increase in the number detected as a result of improved control. A considerable fall ensued, but there was a second peak in 1937. Thereafter there was again a rapid fall and in 1941 there were only 57 outbreaks with 119 deaths in the whole province. It is unlikely that the second rise was due to better control—for the

TABLE 4

Anthrax in Natal, 1925-1941

Year	* Total no. of cattle in Natal	No. of cattle inoculated	No. of smears examined	No. of outbreaks of anthrax	No. of deaths from anthrax
1925-6	2,142,000	30,000	30,000	55	198
1926-7	—	63,000	37,000	93	320
1927-8	2,369,000	177,000	45,000	58	109
1928-9	2,445,000	353,000	68,000	76	181
1929-0	2,485,000	196,000	72,000	112	203
1930-1	—	311,000	137,000	133	263
1931-2	—	277,000	293,000	208	534
1932-3	—	378,000	247,000	157	357
1933-4	2,321,000	324,000	423,000	152	303 **
1934-5	2,407,000	577,000	269,000	106	172
1935-6	2,454,000	639,000	304,000	172	284
1936-7	2,459,000	492,000	309,000	202	485
1937-8	—	606,000	380,000	135	292
1938-9	2,653,000	642,000	242,000	85	173 **
1939-0	—	623,000	374,000	53	74
1940-1	—	593,000	330,000	57	119

* From the *Report of the Agricultural and Pastoral Production of the Union of South Africa,* and only approximate.

** In 1933 the date for the submission of annual reports was changed so that this return of smears, deaths, and outbreaks is for 14 months. In 1938 the older arrangement was reverted to so that the returns for 1938-9 are for ten months only. It has been possible to reconstruct the Transkei figures from monthly reports so that tables 1 and 2 and 3 do not suffer from this discrepancy.

control was excellent before this; but it corresponded, like the similar exacerbation in the Transkei, to a known weakening of the vaccine. The fall recommenced a year later than in the Transkei, where the improved vaccine was used the year before its introduction into Natal. Between three- and four-hundred-thousand smears have been examined annually for a number of years. This represents practically every animal that dies, so there is only a remote chance that an outbreak of anthrax could be missed.

The final result, 57 outbreaks in about two-and-three-quarter million cattle, compares fairly well with the Transkei result, 17 outbreaks in one-and-three-quarter million. The number of doses used in Natal averages about 600,000, a third of that used in the Transkei. If it is assumed that the reduction in anthrax is due to inoculation, it becomes clear that the rigid control of individual farms, such as is possible in many of the Natal European areas, much reduces the need for widespread inoculation. However, although the Natal results are good, they are not so good as those from the far more backward and originally far worse infected Transkei.

Anthrax in the Rest of South Africa

It is interesting to compare the incidence of anthrax in the excellently controlled Transkei and Natal with that in the rest of South Africa. There are parts of the rest of South Africa where 'block' inoculation is done, and considerable areas where a semi-supervised scheme is in force—on infected and in-contact farms. Generally the inoculation is left to the individual farmer. From table 5 it will be seen that two-and-a-half to three million doses are used annually in the rest of South Africa, which has a cattle population of about six to seven millions. Thus between one third and one half of the cattle are immunized every year. How do the results here, where there is a limited amount of systematic inoculation, where most of the inoculation is done by the farmers themselves, and where in large areas veterinary control is very limited, compare with those in the Transkei and Natal? In table 5 are set out the outbreaks in the Transkei and Natal compared with those in the rest of South Africa. The number of smears submitted from each area is also given, as this gives a reliable measure of the state of control.

The number of cattle in the Transkei and Natal is about four-and-a-half millions compared with six-and-a-half millions in the rest of the country. The reported outbreaks in the Transkei and Natal were at first proportionately greater than in the rest of South Africa. Since 1937 the ratio has fallen to less than half that expected. However, it must be remembered that the figures from the Transkei and Natal have been very accurate since 1934 at least, while those from the rest of the country are greatly understated. In 1941, for example, 501,000 smears were examined from the Transkei and Natal, with their cattle population of four-and-a-half millions, and only 105,000 from the rest of South Africa, with a population of six-and-a-half millions. Thus only about one-seventh of the probable number of deaths was reported from the rest of South Africa. The anthrax outbreaks in this area would have been far higher had anything like the real number been reported. It is quite usual for considerable outbreaks of anthrax to be discovered in supposedly clean districts when for some reason or other proper control is instituted. For example, one of us (J. N.) says, in a report on the Ciskei in 1941: "For years we have had no anthrax in Peddie due to non-production of smears. Since March the district has been put under East Coast fever restrictions and you will note that we have had 12 outbreaks with 23 deaths up to the end of June."

In fact field officers are continually saying that the real incidence of anthrax in areas where few smears are taken is much higher than reported.

If it were possible to compare deaths instead of outbreaks, the

TABLE 5

Anthrax in the Transkei and Natal compared with Anthrax in the Rest of South Africa

Year	No. of cattle inoculated in			No. of smears examined in			No. of outbreaks of anthrax in		
	Transkei	Natal	Rest of South Africa *	Transkei	Natal	Rest of South Africa	Transkei	Natal	Rest of South Africa
1925-6	261,000	30,000		32,000			354	55	588
1926-7	235,000	63,000		41,000			372	93	512
1927-8	475,000	178,000		56,000			433	58	—
1928-9	1 062,000	353,000		83 000			351	76	413
1929-0	945,000	196,000	2,250,000	83,000			100	112	305
1930-1	1,034,000	311,000	2,250,000	114,000	137,000	—	84	133	309
1931-2	1,258,000	277 000	2,500,000	119,000	293,000	—	64	208	374
1932-3	1,290 000	378,000	2,750,000	120.000	247,000	—	62	157	356
1933-4	810,000	324,000	3,000 000	138.000	423,000	—	79	152	** 418
1934-5	1,502,000	577,000	3,250,000	123.000	269.000	—	96	106	288
1935-6	1.554,000	639,000	3,750,000	143.000	304.000	—	143	172	444
1936-7	1.569,000	492.000	3.000,000	187.000	309,000	72,000	111	202	573
1937-8	1,549,000	606,000	2,750,000	236,000	380.000	82,000	39	135	589
1938-9	1,590,000	642,000	2,750,000	164.000	243,000	80,000	27	85	** 409
1939-0	1,606,000	623,000	3,000,000	210,000	374,000	108,000	24	53	252
1940-1	1,677,000	593,000	3,000,000	171.000	330.000	105,000	17	57	262

* The figures for the number of cattle inoculated in the rest of South Africa are not accurate. They are obtained by subtracting the number of cattle inoculated in the Transkei and Natal from the amount of vaccine issued. The figures are given to the nearest quarter million.

** As in table 3 the year 1933-4 in the case of Natal and the rest of South Africa includes 14 months and 1939-9 includes 10 months. This is not the case with the Transkei.

differences would be far greater—but these figures are altogether too unreliable. It is well known, however, that in the well-controlled areas the number of deaths per outbreak is very low, whereas heavy mortality sometimes occurs in poorly controlled parts of the country.

A very interesting point is that the Transkei showed a peak in 1935-6, Natal in 1936-7, and the rest of South Africa in 1938-9. In each case the renewed decline coincided with the use in that area of an improved vaccine.

Discussion

The data presented show that there is much less anthrax in well-controlled and well-inoculated areas than in those where inoculation —although on a considerable scale—is more haphazard. In the Transkei, once a heavily infected part, annual inoculation of all cattle is associated with a very low incidence of anthrax. There is evidence that anthrax decreased more rapidly in the districts where 'block' inoculation was first carried out. No hygiene measures were possible in the Transkei, yet anthrax, which was once a deadly menace is now rarely seen. Many districts have been clean for several years. It is noteworthy that in none of the 17 outbreaks reported in 1941 was there more than one death.

In Natal, 'block' inoculation was practised where close supervision was difficult. For the rest, cattle on infected or on in-contact farms only were immunized. Close supervision ensured the speedy diagnosis of any cases of anthrax that occurred. The decrease of anthrax in Natal, although notable, is not so marked as in the Transkei.

The figures for the rest of South Africa are very unreliable. This unreliability can be guaged by the fact that only about one-seventh of the probable number of deaths are reported, whereas in the Transkei and Natal it can be said that practically all deaths are reported, or, what is more to the point, smears are received from almost every animal that dies. Nevertheless, the data even as they are, show that anthrax has decreased more rapidly in the Transkei and Natal than in the rest of South Africa. Were all deaths reported, the difference would be far more striking. This difference can hardly be due to anything but variation in the extent to which inoculation is practised. It is noteworthy that the temporary check in the decrease in anthrax that occurred all over South Africa between 1934 and 1936 could be correlated with a decrease in the immunizing power of the vaccine [Clark (1938)]. In each area the incidence of anthrax again began to fall at a time corresponding to the introduction of an improved vaccine. The preparation of the improved vaccines has been described in previous papers. [Sterne and Robinson (1939), Sterne, Robinson and Nicol (1939), Sterne (1939)].

If it is assumed that the steady reduction of anthrax in the Trans-

kei is due to immunization—and the evidence favours this view—then the continuation of 'block' inoculation should result in a further decrease or, at least, in a maintenance of the present satisfactory position. In Natal there is a probability of some fluctuation; for an accidental introduction of anthrax into any of the large number of non-immunized herds could result in a local flare-up of the disease. For example, there was more anthrax in Natal in 1941 than in 1940. The strict supervision in force will limit such accidents, but they are bound to occur as long as anthrax exists in South Africa. The statistics from the rest of South Africa are not complete, as has been repeatedly stressed. The considerable fluctuations that will probably occur will depend more on the extent to which notification of deaths is enforced than on the actual anthrax situation. Here the reported incidence is only a rough guide, whereas in the Transkei and in Natal the reports reflect the actual state of affairs.

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AKUUT TIMPANITIS (OPBLAAS) BY HERKOUERS

J. I. QUIN
Onderstepoort

As kliniese teken, is opblaas by herkouers nou verbonde met die gisting van voedsel wat as 'n normale proses voortdurend in die voormae van hierdie diere plaasvind. Daar bestaan egter geen duidelikheid aangaande die patogenese van hierdie verskynsel nie, aangesien die herkouer sonder moeite daartoe in staat is om van die groot hoeveelhede gas in die rumen gereeld ontslae te raak. Die feit dat alleen sekere voedselstowwe soos b.v. groen lusern, dit kan veroorsaak lewer bewys daarvoor dat een of ander faktor in die plant self 'n bepaalde rol in die etiologie van opblaas moet speel. Verder bestaan daar die aanduiding dat dit verbonde is met 'n spesifieke steurnis in die groei en metabolisme van die plant as gevolg van die invloed van klimaatsomstandighede en moontlik ook die van grond, bemesting, besproeiing, of selfs meganiese kwetsing van die plante, b.v. deur vertrapping. Aan die ander kant weer is die individuele dier self tot 'n sekere mate verantwoordelik vir die ontstaan van opblaas, aangesien dit dikwels gepaard gaan met honger en gulsigheid.

Sover grond- en klimaatstoestande steurnis in die plant mag veroorsaak bestaan daar die mees uiteenlopende sienswyses. Volgens die bevindings van McIntosh in Noord Amerika is daar egter aanduidings dat die instandhouding van lusernlande deur toereikende bemesting en besproeiing die gevare van opblaas verminder. Die weiding op verwaarloosde en uitgeputte lande, inteendeel gaan dikwels gepaard met die verskyning van opblaas. In hierdie verband moet die noodsaaklikheid van 'n deeglike studie op lusern onder suid-afrikaanse omstandighede beklemtoon word.

Wat die moontlike giftigheid van lusern betref kon van der Walt en Steyn geringe spore van blousuur (tot 2 mg. per 100 gram) in vars materiaal vasstel. Daar bestaan egter geen bewys dat dit verantwoordelik is vir die opblaas nie.

Skaap proewe op Onderstepoort uitgevoer

Gedurende die afgelope paar jaar is verskeie proewe veral op merino skape op Onderstepoort uitgevoer met die doel om kunsmatig opblaas te veroorsaak om sodoende die patogenese van die toestand te bestudeer. Vir die doel was volwasse merino skape gebruik wat afsluitbare permanente fistels in die rumen gedra het. Deur hierdie fistel tegniek was dit moontlik om verskillende stowwe in die rumen

in te laat en ook om gas en rumen materiaal vir verdere ondersoek direk uit die rumen te versamel.

Lusernvoeding

Herhaalde proewe was uitgevoer waarby gesnyde groen lusern, in verskillende stadia van groei en verleptheid aan skape gevoer was, sonder dat enige duidelike kliniese beeld van opblaas teweeg kon gebring word, of die diere nou uitgehonger was of nie. Wel was dit die bevinding in een geval dat 'n besending gedroogde gebaalde lusern uit die Cradockse distrik in staat was om opblaas te veroorsaak in skape op Onderstepoort. Die eienskap van die lusern het egter spoedig verdwyn na aankoms hier, sodat geen verdere proewe daarmee uitgevoer kon word nie. Hierdie ondervinding, in ooreenstemming met die van andere, lewer bewys daarvoor dat dit geensins maklik is om op kunsmatige wyse opblaas deur lusern voeding te veroorsaak nie daar die faktore wat dit bepaal erg labiel is en dus baie skielik kan verander. Nietemin was daar duidelike bewys, volgens gasmetings asook uit die ondersoek van rumen materiaal, dat lusernvoeding die gistingsproses in die rumen versterk en dat dit gepaard gaan met 'n snel verlopende opbruising en die vorming van groot hoeveelhede gas. Die oopmaak van die fistel kort na die vreet van lusern veroorsaak vinnige uitspuiting van 'n dun skuimende masa. Alhoewel opbruising baie skielik te voorskyn kom na 'n lusern maaltyd, verdwyn dit ewe eens, gewoonlik binne 'n uur of twee daarna.

Oorsaak van Gasvorming en Opbruising in die Rumen

Om die opblaas probleem enigsins te verstaan is dit noodsaaklik om na te gaan waardeur die gas in die rumen veroorsaak word en waarom dit nie op normale wyse en spoed sou kon ontsnap nie. Met die oog hierop het ons 'n lang serie proewe op Onderstepoort onderneem, die resultate waarvan korteliks hier aangehaal mag word.

1. *Inpomp van Lug in die Rumen.* — Om na te gaan of die spoed van gasvorming as sulks die oorsaak van opblaas kan wees was die fistel buis in verskeie skape gekoppel aan 'n groot gas silinder wat met lug onder hoë druk volgepomp was. Op die wyse kon die lug onder variërende spoed in die rumen ingelaat word. Die bevinding was dat die diere sonder veel moeite of ontsteltenis in staat was om groot hoeveelhede gas, soveel selfs as 25 litre lug per minuut, deur die slukderm op te breek. Die vulling van die rumen met kos en water, asook die posisie van die dier, b.v. met die voorkwarte óf hoog óf laag geplaas, dit alles het geen invloed gehad op die gemak waarmee die dier van die oormate lug in die rumen ontslae kon raak nie. Aangesien die hoeveelhede lug wat ingepomp was baie groter was dan die volume gas wat gedurende dieselfde tydperk onder die hewigste gisting in die rumen self sou kon gevorm word, is dit duidelik dat

die spoed van gasontwikkeling en die hoeveelhede gas gevorm op sigself geensins die oorsaak van opblaas kon gewees het nie.

2. *Suiker Fermentasie in vitro Uitgevoer.* — Deur die toevoeging van vars lusernsap of waterige ekstrakte van droë lusern tot 'n fles wat klein hoeveelhede (50 cc.) van die waterige inhoud van die rumen bevat het, kon bewys gelewer word dat daar 'n besonder snel verlopende gisting plaasvind wat reeds binne die eerste 10 tot 20 minute sy hoogtepunt bereik en daarna spoedig tot normale bedaring kom. Deur die gas in manometers op te vang en die volume oor vasgestelde tydperke af te meet, kon die gistingsproses noukeurig gevolg word. Hieruit is afgelei dat dit kenmerkend dieselfde is as wat in die rumen self plaasvind soos aangedui deur die skielike opblaas van 'n dier na die vreet van lusern. Verder kon bewys word dat oplossings van die drie suikersoorte sukrose, glukose en fruktose dieselfde skielike fermentasie ondergaan in teenwoordigheid van rumen inhoud as die lusernsap self, terwyl geen een van die ander suikers, asook stysel, sellulose of eiwit hierdie eienaardigheid vertoon nie. Dit was dus 'n bewys dat een of ander suiker in die lusern verantwoordelik gehou moet word vir die gasvorming en gevolglik dat 'n verandering in die suikergehalte van die plant materiaal verbonde was met sy opblaasvermoë.

'n Ander bevinding was dat die rumen inhoud van diere wat langdurig op 'n rantsoen bestaande uit swakgehalte droë grashooi of kaf gehou was, of selfs oor 'n tydperk van 2 tot 3 dae uitgehonger was, sy vermoë verloor het om suikeroplossings vinnig te laat gis en dus byna geen gas kon lewer nie, terwyl diere wat reeds 'n tyd lank aan lusernvoeding gewoon was by uitstek 'n skielike fermentasie van suikers kon bewerkstellig. Temeer kon dit bewys word dat die toevoeging van geringe spoortjies (1 :40,000) kopersulfaat oplossing, asook 'n hele reeks ander gifstowwe hierdie gistings reaksie verswak of selfs geheel-en-al rem. Die gevolgtrekking uit hierdie proewe was dus duidelik nl. dat ons hier te doen het met 'n spesifieke ensiem stelsel verder dat hierdie stelsel onderhewig is aan groot verandering en in in die rumen wat verantwoordelik is vir die skielike gisting van suiker; noue samehang verkeer met die geaardheid van die voedsel en die toestande in die rumen.

3. *Die geaardheid van die ensiem stelsel.* — Dit kon bewys word dat die ensiem maklik neergeslaan word deur ligte sentrifugering en dat dit dus gebonde is en nie in vrye oplossing verkeer nie. Verder is dit vasgestel (in samewerking met Myburgh) dat dit gevoelig vir veranderings in die pH is, veral buite die perke pH 6.5 tot 7.5 en ook vir hitte, daar dit by 55°C na 'n halfuur nie meer aktief is nie. Deur mikroskopiese ondersoek is dit gevind dat die neerslag uit die rumen vloeistof behalwe infusoria ook groot getalle van 'n onbeskrewe gissoort bevat. Na die toevoeging van suiker by die rumen vog is dit by uitstek hierdie gisselle wat die skielike gas ontwikkeling binne

enkele minute veroorsaak. Gepaard met die gasvorming vind daar 'n ewe vinnige omsetting van suiker na glykogeen plaas, soos aangetoon deur die intensiewe diep bruin kleuring van hierdie organismes met jodium oplossing. Dit lewer dus bewys daarvoor dat die suiker nie meteens volledig uitgefermenteer word nie maar dat die organismes 'n sekere gedeelte daarvan assimileer in die vorm van glykogeen wat volgens huidige beskouing 'n aaneenskakeling van sowat 12 tot 14 molekules glukose veronderstel.

4. *Die Gas in die Rumen.* — As die dier normaal in staat is om groot hoeveelhede rumen gas deur die slukderm op te breek, waardeur dan word hom hierdie vermoë so skielik onder sekere omstandighede ontneem? Uit proewe met skape op lusernlande wat op Losperfontein Proefplaas (in samewerking met Starke) 'n tyd gelede uitgevoer is asook uit die werk op Onderstepoort gedaan, kon die volgende gevolgtrekkings gemaak word:—

(a) Opblaas kan met sekerheid deur vars onverlepte, onbeskadigde lusern veroorsaak word.

(b) Die verskynsel hang saam met die gulsige inname veral van groen lusern blare, en nie so seer met die stengels nie. Gevolglik kom dit veral voor onder hongerige, produserende diere soos lammerooie, en melkkoeie en veral wanneer hulle op 'n vars stuk lusern met hoë blaargehalte wei.

(c) Alle ander faktore inaggeneem, styg die gevaar van opblaas met die suikergehalte in die blare wat, volgens analieses deur Louw uitgevoer op Losperfontein lusern, van 2.5% in die vroë oggend beloop tot oor die 6% suiker in die laat namiddag (bereken op 100 gram droë materiaal). Die analieses is gemaak op lusern monsters tydens die werklike voorkome van dodelike opblaas onder lammerooie.

(d) Die dier sterf as gevolg van asemnood veroorsaak deur die skielike en enorme spanning in die rumen waardeur die asemhalingbewegings van die midderif onmoontlik gemaak word. Terselfdertyd verbied hierdie druk die terugvloeiing van veneuse bloed na die hart met die gevolg dat verdere belemmering van respirasie en sirkulasie veroorsaak word.

(e) Daar is hoegenaamd geen aanduiding van verstopping in die slukderm nie, inteendeel staan die kardia gedeelte van die slukderm tregtervormig wyd ope by die dood van die dier.

(f) Op hierdie tydstip is die voormaë styf gevul met 'n opbruisende, gistende massa, min of meer van die geaardheid van 'n digte seepskuim.

(g) Deurdat die gasborrels nie vinnig genoeg breek nie word die vrystelling van die werklike gas vertraag. Aangesien

die dier wel in staat is om gas op te breek, maar nie skuim nie, styg die druk in die rumen geweldig en skielik.

(h) Volgens alle aanduidings word die breek van die skuim vertraag deur die hoë kalloied gehalte van die groen blare en veral deur saponiene teenwoordig in lusern. Die werking van saponiene is by uitstek gekenmerk deur verhoogde oppervlakte spanning en die vorming van skuim.

(i) As hierby inaggeneem word die wisselende suikergehalte van lusern en die geweldige vermeerdering in die aantal gisselle by diere op lusern dieët dan word die patogenese van opblaas iets meer verstaanbaar.

(j) Sover dit die bestryding van opblaas betref is dit duidelik dat die gebruik van die maagbuis, die *trokar* of middels so as terpentyn en lynolie wel 'n verligting van die toestand mag veroorsaak dog geensins die probleem vir die vee-eienaar oplos nie.

(k) Die oplossing van hierdie lastige vraagstuk, as daar van 'n oplossing in 't geheel gepraat kan word, lê opgesluit in 'n meer doeltreffende metode van voeding van die herkouer, so toegepas dat alvorens die diere toegang kry tot groen lusern, hulle eers op een of ander hooi of gras rantsoen geplaas moet word. Daardeur sal die aptyt, gedeeltelik altans, bevredig word, en die diere dus minder gulsig wees as hulle op die lusern kom. Verder sal 'n gedeeltelike vulling van die rumen met hooi of gras die opbreek van die gasborrels met groter gemak bewerkstellig. In ieder geval kan groen lusern alleen, uit die aard van die saak, nie aan die behoeftes van die produserende herkouer voldoen nie, want vereers bestaan dit hoofsaaklik uit 'n waterige konsentraat veral in die voorblom stadium. Verder dwing dit die dier deur uitermate gisting en uitswelling van die rumen tot herhaalde onderbreking in sy voedsel inname. Gevolglik word die rumen gevul, *nie* met vaste voedsel nie maar met 'n waterige skuim waarvan 'n groot gedeelte eers kwyt geraak moet word alvorens daar ruimte in die rumen bestaan vir 'n verdere inname. Die onbevredigende resultate wat dikwels verkry word met die vetmaak van lammers op lusernweiding staan moontlik ook verbonde aan dieselfde moeilikhede.

Al hierdie waarnemings dui aan hoeveel meer ingewikkeld die spysvertering van die herkouer is dan die van ander soogdiere, en gevolglik ook hoe gevaarlik enige steurnis van die gistingsprosesse in die vermoë vir die gesondheid van die dier mag wees.

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ACUTE TYMPANITIS (HOVEN) IN RUMINANTS (SUMMARY).

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Clinically, acute hoven is closely associated with normal fermentation and gas production in the forestomachs of ruminants. Moreover it is conditioned by both plant and animal factors. From investigations conducted on sheep with rumen fistulae and fed on different diets, it has been shown that lucerne stimulates fermentation and excessive gas production accompanied by a foaming up of the ingesta. Although extremely sudden in its onset, the process likewise subsides very rapidly, usually within 30 minutes. Fermentation of glucose, sucrose, and fructose by rumen ingesta in vitro results in a similar, fulminating type of gas production not seen with any of the other sugars tested, neither with starch, cellulose or protein. This characteristic fermentation is caused primarily by a species of false yeast cell present in large numbers, especially in the forestomachs of lucerne fed sheep. Through a process of fermentative assimilation as already described for various other organisms, part of the sugar is synthesised into glycogen and stored as reserve carbohydrate by the yeast cells while the remainder is rapidly broken down to carbon dioxide, comprising the main gas evolved, and water. Copper sulphate in high dilution inhibits this process. Foam formation is aggravated by saponin present in lucerne. Due to the difficulties in regard to eructation, an excessive accumulation of intraruminal foam leads to overdistension of this organ, and subsequently to immobilization of the diaphragm. Respiratory distress accompanied by decreased return of venous blood from the abdominal region may result in sudden death.

The incidence of acute hoven may be limited by the feeding of a mixed diet in which green lucerne is made to follow the other components e.g. grass hay and concentrates.

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A NOTE ON ACUTE VERMINOSIS IN CATTLE IN A SEMI-ARID AREA

J. M. FOURIE
Vryburg

Although pathogenic nematodes are frequently encountered in cattle in this country, great significance is usually not attached to their presence and little attention has been paid to them in the veterinary literature for farmers in South Africa. It is known that calves may be severely affected by heavy infestations acquired on moist pastures, especially in coastal regions. Cases of mass and unusually acute infestation seen on farms in the Hoopstad, Kuruman and Vryburg Districts, which are in a semi-arid area, seem worthy of record.

During October 1940, one of the most progressive and careful farmers in the Hoopstad District complained about disease and mortality amongst his cattle. Young animals about 18 months old were affected. At the time of investigation two were already dead and six were found to be very ill.

Symptoms

The animals affected were in very poor condition and the most pronounced symptoms were large oedematous swellings in the throat region up to the size of an ostrich egg, weakness, signs of severe anaemia and very persistent watery diarrhoea.

Post-mortem Appearances

The carcase of one animal killed *in extremis* showed pale mucous membranes, watery blood, extreme cachexia, hydrothorax, hydropericard, ascites, and gelatinous infiltration and replacement of all fatty tissues. The abomasum contained reddish-brown fluid-like ingesta and very many *Haemonchus contortus*. A fair number of *Bunostomum phlebotomum* and a severe infestation with *Cooperia pectinata* were seen in the small intestine, which also showed catarrhal enteritis. The colon contained a few *Oesophagostomum radiatum*.

Treatment

Dosing with 40 cc. diluted tetram led to marked improvement of the less severely affected cases. Animals which were already very weak either died or recovered very slowly. In view of the condition of the animals it was not considered advisable to give a dose of tetram. In other instances the copper sulphate and nicotine mixture gave good

results. It is not known which particular parasites were affected by the drugs, but removal of the wireworm alone should lead to considerable improvement. In cases where the anaemia is marked it is advisable to give a little iron sulphate in the bone meal or in a lick.

Environment

On this particular farm in the Hoopstad District good rains were recorded during the preceding summer months until April. During both May and June half an inch of rain fell, but during July and August there was no precipitation. In September there was a light shower which had little visible effect on the veld. There was still a fair amount of old grass and most of the cattle were in fair condition. The soil is very sandy throughout and the pasture is grass-veld, with some old lands. The farm is well divided into paddocks and the drinking water is supplied from cement troughs around which little or no moisture was noticed. The cattle were spoon-fed supplementary phosphates three times per week. The owner informed me that during the later part of winter he had noticed about twenty young head of cattle with swellings in the throat region.

Discussion

According to information gathered from farmers and from personal observations made in the Hoopstad and Vryburg Districts, it would appear that young cattle may suffer, more often than is usually supposed, from the effects of round-worm infestation, principally during the critical period of the year when the grazing is inadequate. It is considered that the problem will only arise under conditions inducing poverty and that such aggravated symptoms as are described above will only be seen in young stock.

Mass infestation with wireworm and hookworm, especially in young cattle was seen in the autumn of 1941 on many farms on the Molopo river in the Vryburg and Mafeking Districts. As a result of heavy rains in January and February the river was flooded and became a marshy vlei with deep pools which retained water well into winter. These extensive outbreaks along the river show that, as is to be expected, moisture is an important factor in verminosis of cattle in this area. According to Mönnig (1931), the conditions found in the dung heaps of cattle are ideal for the development of worm larvae, which crawl up on the grass which grows around the dung heaps and infect cattle grazing on such grass. It is known that cattle do not readily graze the grass which grows on or around their dung heaps, but do so only when the grazing becomes scarce. During periods of scarcity this habit would tend to favour infestation, in the manner suggested by Mönnig.

The sandy nature of the soil in these parts, phosphorus deficiency in the grazing, and the short herbage often existing after winter may

be factors contributing to the severity of the infestation. It would appear that in these mass infestations *Haemonchus contortus, Bunostomum phlebotomum,* a *Cooperia* species and *Oesophagostomum radiatum* usually occur in combination.

The question arises whether cattle, especially young growing animals, fairly heavily infested with blood-sucking internal parasites will not be more liable to develop pica. If this is the case, worm infestation may contribute in part to the many cases of lamsiekte seen in young cattle in the North-Western Free State and Bechuanaland areas of the Union. Under certain conditions it may therefore become necessary to dose susceptible cattle during winter, before the most critical time of the year. In his annual report for 1939-40 the District Veterinary officer at Vryburg stated that many cattle in Bechuanaland are suffering from a high degree of worm infestation and that this is probably one of the reasons why cattle are not thriving on many farms.

Acknowledgement

The author wishes to thank Dr. H. O. Mönnig of Onderstepoort for the identification of the parasites from specimens forwarded and for suggestions in connection with this publication.

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PRELIMINARY NOTE ON THE TREATMENT OF CANINE COCCIDIOSIS

B. S. PARKIN
Onderstepoort

Recently considerable success has been obtained in the treatment of natural cases of coccidiosis of dogs. The causal coccidia were Isospora bigemina, I. rivolta and I. felis. Negative faecal examinations for the oöcysts were obtained in 7 days and improvement was marked within 24 hours after the commencement of treatment.

The treatment was the administration at intervals of twenty-four hours of one or more enemata of 10 c.c. of 1% solution of sodium sulfanilyl sulfanilate (Lederle, New York) per kg. live body weight.

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THE TOXICITY OF USED MOTOR OILS

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A case was reported in which porridge, which had been stored in an iron drum containing a small quantity of used motor oil, was fed to cows. A number of these cows became ill and died. It was suggested that the used motor oil was responsible for death. However, since the quantity of motor oil was very small and because used motor oil was not considered to be highly toxic, this was doubted. It was, however, thought advisable to determine the toxicity of used motor oil. For this purpose we used Shell motor oil, which had been drained from a motor car engine that had done at least a thousand miles without the oil having been changed. Rabbits were drenched with the oil by means of a stomach-tube and the following observations made:—

Quantities of 50 and 100 cc. of the oil had nothing but laxative effects. Two rabbits, which had received 480 cc. of the oil in four doses in the course of thirty hours died on the fifth day after commencement of dosing. The most prominent feature in these cases was that the liver was markedly enlarged, ochre in colour and very much reduced in consistence.

Microscopical examination by Mr. R. Clark of the Section of Pathology, Onderstepoort revealed a marked diffuse accumulation of fat in the hepatic cells with distinct necrobiotic changes. Similar changes were observed in the kidney.

Discussion

It appears that very large quantities of used Shell motor oil are capable of causing death in animals, but that these quantities are in great excess of those likely to be consumed by animals, or administered to them in the treatment of disease. But a possibility, which must not be lost sight of, is that porridge stored in galvanised iron drums may contain toxic quantities of zinc and/or lead. It must also be borne in mind that used motor oil may contain various impurities more toxic than the oil itself.

In this connection the following information was kindly supplied by Dr. F. Tromp, Director, Fuel Research Institute of South Africa, Pretoria:—

(a) It is very unlikely that oil drained from motor car engines will contain any impurities other than small quantities of petrol. The

combustion products of petrol and oil present in the drained oil are not being considered here as the above tests have shown that they are toxicologically not of great importance. However, it should be mentioned that the cylinders of some makes of cars contain lead-copper alloys with the result that in certain circumstances (burning out or excessive wearing of the cylinders) the engine oil may contain fair amounts of lead and copper.

(b) Tractor engine oil usually contains fair amounts of paraffin.

(c) In aeroplanes tetraethyl lead is commonly used as an "anti-knock," consequently the engine oil contains lead. The engine oil sludge has been found to contain up to 20 per cent of lead.

Paraffin is a fairly toxic substance, the various brands differing in degree of toxicity (Steyn, 1931) while lead is a dangerous poison, especially for cattle.

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A SCHEME OF THE POTENTIALITIES OF THE MESENCHYME.

CECIL JACKSON and GILLES DE KOCK
Onderstepoort

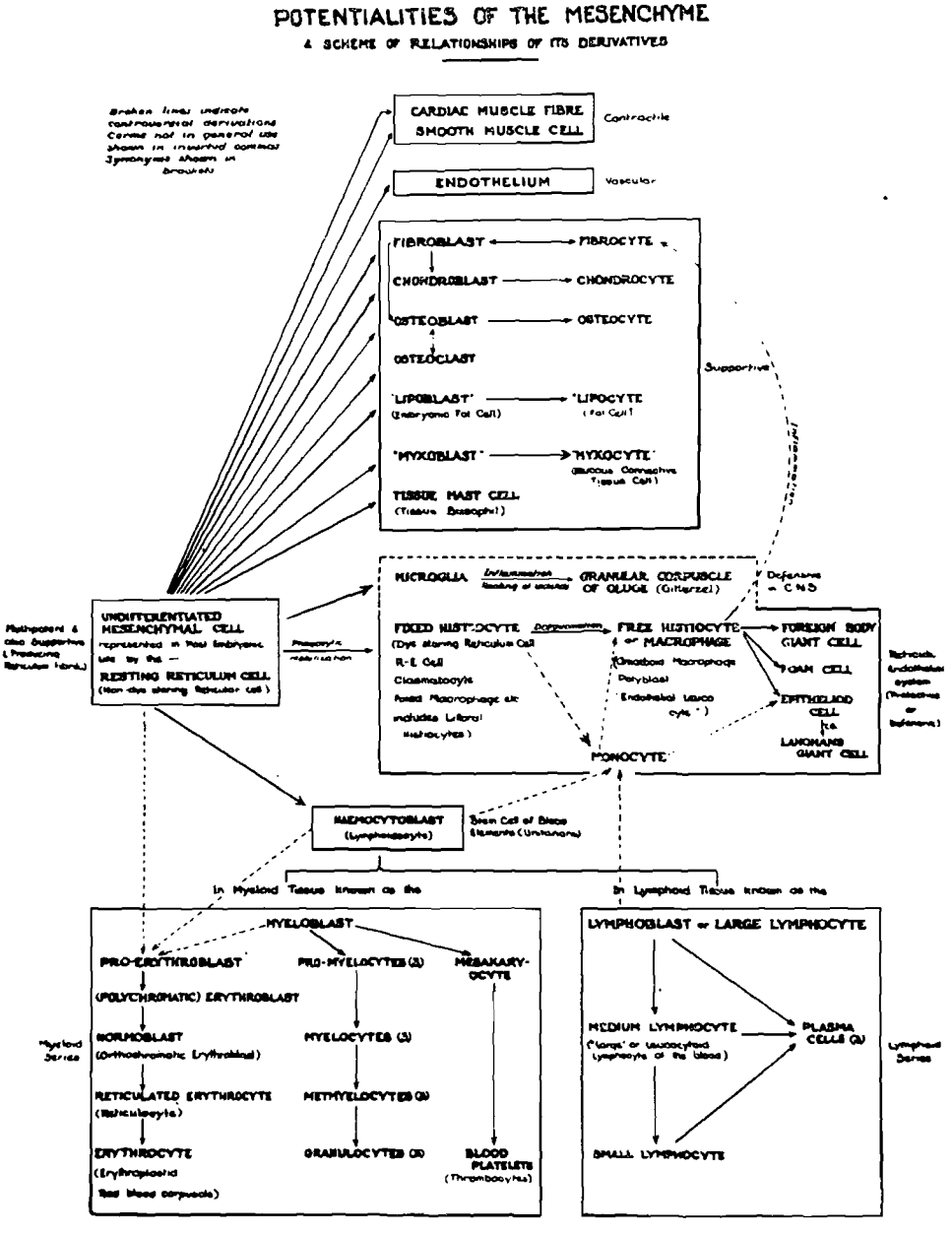
The accompanying diagram of the potentialities of the mesenchyme and the relationships of its derivatives has been introduced for teaching purposes at Onderstepoort, in an attempt to achieve some uniformity of conception and nomenclature in the different departments and after consultation between those members of the staff engaged on pathology, histology, and embryology.

It is offered here for what it may be worth, and no particular claim is made to originality.

The scheme could have been made more complicated by the inclusion of still more of the controversial derivations (shewn by dotted lines), but only at the expense of making it more cumbersome for students to grasp. Except in broad outline, we have sought to avoid expressing our personal views, but rather to reflect what appears to be the consensus of the opinions expressed in the very voluminous literature surrounding this subject.

Regarding the origin of the blood cells, the scheme is essentially unitarian in conception, but a reconciliation with at least the terminology of the dualists is attempted, on lines previously suggested by one of us (Jackson, 1934), who furthermore teaches and demonstrates

microscopically the derivation of the erythrocytes of normal adult domestic mammals from the large lymphoid cells (myeloblasts) of the bone-marrow and would have preferred to indicate this more definitely on the chart. However, in deference to the lack of uniformity in the literature, this dogmatic teaching has not been reflected.



The reason for not associating the microglia with the R E system more definitely than by broken lines is that these cells do not appear to store the vital dyes, as tested by the usual technique. In all other respects they would seem to belong to this system, and their mesenchymal derivation, though still denied by a few authors, is not open to very serious doubt.

The more important synonyms are given in brackets, while heavy type indicates the terms we prefer to use. Where not in general use, the names are placed between inverted commas.

An enlarged version of the chart, which includes a painting of each cell type mentioned, has been prepared for the Onderstepoort Museum.

Assistance has been obtained from the diagrams published by Bloom (1938) and Maximow and Bloom (1938). A complete bibliography would be out of place here, but mention should be made of the excellent review of Maximow (1927).

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A CASE OF TUBERCULOSIS IN A DOG

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Onderstepoort

Tuberculosis in the dog is not of frequent occurrence and the recorded incidence varies greatly. Figures from European sources give the incidence from clinical examination as .04% to 1% and from post-mortem records as .83% to 5.6%. Lovell and White (1940) found that 4.6% of dogs coming to post-mortem from the clinic of the Royal Veterinary College in London showed tuberculosis. The symptoms in these cases were mainly of two types. When the primary lesions involved the mesenteric lymph nodes, vomiting and diarrhoea of a few weeks' duration were noted. When the lungs were primarily affected, loss of condition and coughing were noted over periods of up to a year. The serous membranes were often involved and in some cases tubercular pleurisy or peritonitis was found. Healed-out

lesions of tuberculosis were not found. In only 4 cases out of 22 was the bovine type of the tubercle bacillus isolated. In these cases the mesenteric nodes were affected.

Approximately 75% of tuberculosis cases in the dog are caused by the human type, mainly owing to the opportunities for infection. One case of avian infection has been recorded. Sometimes there is a definite record of tuberculosis in the family with which the dog is associated. But there is no record of transmission from dogs to human beings, although the danger exists. In France Calmette (1923) points out the frequency of the disease in dogs which frequent cafes and restaurants with their owners and mentions that in 25% of cases tuberculosis existed in the owner's family.

The case under consideration occurred in a dog belonging to the owner of a travelling circus. The patient was a Great Dane dog sent to Onderstepoort in August 1940, for treatment on account of loss of condition and sarcoptic mange infection. It had a history of an attack of pneumonia some time previously. Treatment for mange was successful, but in spite of a good appetite the dog did not improve and showed intermittent temperature reactions and diarrhoea. The possibility of tuberculosis was suspected and on 9th October, 1940, an intradermal tuberculin test was carried out by Dr. Fourie. A well-marked positive reaction occurred.

The dog was killed on 18th October 1940, and at post-mortem lesions of a tubercular type were found in the lungs, liver, spleen, and serous membranes. In the right lung a large yellowish consolidated area was found, and in the left a number of firm yellowish nodules. The bronchial and mediastinal glands did not show macroscopic changes. Nodules were found on the peritoneum and pleura. The liver showed numerous firm yellowish areas.

Pathological Examination

A report on the microscopic changes, made by Dr. Thomas, showed them to be those of generalized tuberculosis. The liver showed extensive lesions with necrotic centres (but no giant cells) and there were no lesions in the periportal glands. The lungs showed granulation centres with proliferative cells, endothelial, neutrophile and lymphocytic. Numerous tubercles were seen in the pericardium. Giant cells and epithelioid cells were seen in the trabeculae of the spleen.

Bacteriological Examination

The bacteriological examination showed the presence of acid-fast bacteria in the lesions and cultures were made direct from them on Dorset's egg medium. Pure cultures of an acid-fast organism of the mycobacterium type were obtained, the growth being dry and scaly.

Two guineapigs were inoculated subcutaneously with an emulsion of material from the lesions. One died on the 13th day, too early

for lesions to have developed. The second one was killed on the 42nd day and enlargement and pus formation were seen in the precrural gland on the side on which the inoculation had been done in the leg. Lesions of tuberculosis were present in the subsacral glands and in the spleen in the form of scattered nodules.

Two more guineapigs and in addition two rabbits were inoculated subcutaneously with a small amount of culture material. No attempt was made to measure the amount accurately. Owing to an error these animals were all killed a month after inoculation. No lesions were found in the rabbits, but both guineapigs showed local lesions with pus centres in the precrural glands and a few lesions in the liver and spleen.

Four further guineapigs were then inoculated, two subcutaneously and two intraperitoneally with culture material, fairly big doses being given, but without actual measurement. One inoculated intraperitoneally died at six weeks with well-marked generalized tuberculosis. The other one was killed at two months and showed extensive generalized tuberculosis. The two guineapigs inoculated subcutaneously were left until three months had elapsed. At post-mortem both showed well-marked generalized tuberculosis.

In the meantime the organism had been well established by several subcultures on Dorset's egg medium without glycerine and it was decided to attempt to type it accurately.

Cultures were made on the litmus glycerine potato medium recommended by Griffith for type differentiation. On this medium a heavy, yellowish, dry, crumbly growth occurred in about a month. On repetition similar growths were obtained. Several human and bovine strains were used as controls, the bovine ones giving scanty, or no growth, while the human ones gave growths similar to that with the dog strain.

Inoculations were then made into rabbits and guineapigs with measured amounts of culture material. Two rabbits each received 10 mg. and two guineapigs 1 mg. subcutaneously. One guineapig died at three weeks from an inter-current infection. Only enlargement of the precrural gland on the side of the inoculated leg was seen.

One of the rabbits was killed seven weeks after inoculation. The only lesion found was extensive abscessation in the inoculated thigh, with acid-fast organisms frequent in the pus. The surviving guineapig was killed at the same time. It was in good condition and active, but at post-mortem showed fairly well-marked generalization with nodules in the liver and spleen, and a few in the lungs.

The other rabbit was killed at the same time but showed no visible lesions at post-mortem.

Conclusions

From the experiments carried out it would appear that the strain of *M. tuberculosis* from a dog had a moderate virulence for guineapigs and very little for rabbits, using the standard dosage for typing tuberculosis strains. The virulence, associated with the cultural characteristics, justify one in regarding the strain as being of the human type.

References.

Calmette, A. (1923). *Tubercle bacillus infection and tuberculosis in man and animals.* Williams & Wilken Co.

Lovell, R. and White, E. G. (1940). Tuberculosis in Dogs. *Br. Jl. Tuberculosis* 34:17.

AN UNUSUAL SITE OF ATTACHMENT FOR TICKS

R. DU TOIT and H. O. MÖNNIG

On the 6th February, 1942, a farmer, living about 15 miles north of Onderstepoort in the Pretoria district, reported having found ticks in the mouths of a number of his cattle on several occasions. The report was made by telephone and it appeared that he had that morning discovered an animal with a tick attached in its mouth.

This farm is situated in an area where heartwater is very prevalent and we considered an examination of the animal desirable, with the object of investigating the possibility of this disease being transmitted by ticks situated in a site where they would not be exposed to the usual methods employed for tick eradication.

On arrival, the owner informed us that from time to time over a period of a few months, he had discovered ticks in the mouths of a number of cows to which he had been administering bone meal daily by means of a spoon. At first he had attached no significance to the phenomenon, but after about a dozen such ticks had been found, he began speculating about the possibility of the presence of ticks at this site among the remainder of the animals on the farm, which were not dosed with bone meal.

We examined the animal in question and found an adult male bont-legged tick—*Hyalomma aegyptium* var. *impressum* Koch—firmly attached by means of the hypostome to the mucous membrane of the hard palate. The actual site of attachment was about the centre of a transverse furrow between two of the palatine ridges, halfway along the length of the hard palate. Upon removal, which required appreciable force, the tick was found to be living and apparently normally active and, to all appearances, entirely unaffected by the saliva in

which it had been constantly bathed. None of five other animals which we examined showed the presence of ticks in their mouths and there was no opportunity of examining any of the other cattle on the farm.

The farmer, who appeared to be well acquainted with the commoner species of ticks, assured us that among the ticks which he had found previously were several specimens of bont tick, *Amblyomma hebraeum* Koch, the transmitter of heartwater. However, none had been engorged, but all were firmly attached and living when removed.

The occurrence appears to be worth recording; and it is hoped that, where opportunities exist in heavily tick infested areas, field officers will follow up this lead and report any similar observations.

It is considered unlikely that female ticks would be able to complete engorgement in such a site as, when distended with blood, they would probably be crushed during the acts of mastication and rumination. The possibility exists, however, that diseases such as heartwater may be transmitted if any feeding takes place by infected ticks.

BOOK REVIEW

Plague on us: Geddes Smith

In recent years a number of books have appeared in which attempts have been made to explain in popular form, some aspect or other of medical research work. Some of these such as the books of Paul de Kruif have been very successful but it is rarely that one meets with a book written in such masterly and lucid style as "Plague on us." It deals with the very vital subject of epidemiology and refers to some of the most recent discoveries in medical science. Throughout the book there is an absence of exaggeration and hero-worship which makes many similar ones so irritating.

The author has divided his book into seven chapters with a prologue and an epilogue. These chapters form a natural sequence and follow the study of infectious disease from early times up to the present. The first one deals with some of the great plagues in the past and contemporary thought on them. Mention is made of the disease known as the English sweat, which appeared mysteriously after the Wars of the Roses and as mysteriously disappeared a few years later, never to reappear.

The second chapter is called "Past Thinking" and deals with speculation on the causes of various diseases before the era which commenced with Pasteur's researches. In the third chapter, called "The Sick Man," the problems of infection of the body by the organisms producing disease and its resistance to such infection are discussed in the light of the most recent research work. In the next chapter the author departs from the individual and discusses the various aspects of infectious disease as it affects man in the mass. The next problem dealt with is the defences of the human

body to disease and the methods by which they can be reinforced. The most modern vaccination methods are dealt with and the most recent chemotherapeutic agents are discussed. A fascinating chapter is devoted to what is called "Detective Work." In it the investigation of a number of typhoid outbreaks is described as well as the epidemic of amoebic dysentery which commenced in two Chicago hotels at the time of the World's Fair and was associated with a defective and obsolete plumbing system.

Perhaps the most interesting portion of the book to the critical reader is the one called "Unfinished Business." This deals with the problem of the infectious diseases over which there is no adequate control at present and may not be until more knowledge about them is available. In the case of two diseases the position has become complicated by factors not previously realised. In plague for instance it is known that the disease is widespread in wild rodents in the United States of America and that yellow fever exists over large areas of Brazil in an endemic form, some animal, probably a monkey species, acting as a reservoir.

The epilogue to the book summarizes some of the main points previously discussed. It makes the big point however that the research work which has been done in the Pasteurian tradition has rather led investigators away from the study of the sick man himself and that we must now return to him. Insufficient is known of the physiology of the diseased cell and to go a step further, the molecules of the cell. The author concludes with the following sentence which very adequately sums up his book:

"When pestilence falls on the people there is a story to tell. The story of the people who do not fall sick has never been told. Perhaps it is the most important part of epidemiology."

Plague on Us. Geddes Smith. Commonwealth Fund Division of Publications. Oxford University Press. Price 16/-.

SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION

BALANCE SHEET as at 31st MARCH, 1942.

LIABILITIES.						ASSETS.					
Subscriptions Paid in Advance				£7	1 0	Cash at Bank				£103	8 11
Book Fund Suspense Account				26	5 6	Subscriptions in Arrear				250	17 0
Natal Branch—						Deposits Standard Building Society				521	15 3
Balance 1/4/41	£9	12	0			Deposits United Building Society	£325	0	0		
Add Subscriptions Collected		10	0			Add Accrued Interest	3	15	0		
				10	2 0					328	15 0
Prize Fund—						Union Loan Certificates	787	10	0		
Balance 1/4/41	145	0	0			Add Accrued Interest	41	0	0		
Add Profits Book Fund	22	7	2			Government 3½% Local Regd. Stock	398	0	0		
Interest	5	9	11			Add Accrued Interest	7	0	0		
	172	17	1							405	0 0
Less Award	10	0	0			Government 3½% Savings Bonds	200	0	0		
				162	17 1	Add Accrued Interest	2	0	1		
Benevolent Fund—										202	0 1
Balance 1/4/41	520	5	4			Student Advances				106	18 4
Add Interest on Investments	21	9	11								
Interest Contribution	2	5	5								
Profit Group Endowment Fund, 1940-41-42	49	1	1								
Contribution of 5/- per Membership subscription collected	38	10	0								
Donation	5	5	6								
	636	17	3								
Less Payments	48	0	0								
				588	17 3						
General Fund—											
Balance 1/4/41	1,647	9	1								
Add Interest Accrued prior to 31/3/41	53	0	0								
Surplus for year as per Income and Expenditure Account	251	12	8								
				1,952	1 9						
				£2,747	4 7					£2,747	4 7

SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION

INCOME and EXPENDITURE ACCOUNT for the YEAR ENDING 31st MARCH, 1942.

Dr. | Cr.

EXPENDITURE.

To Interest at 3½% on balances of Prize Fund			£5	9	11		
Benevolent Fund			2	5	5		
						£7	15 4
To Journal—							
„ Printing and Reprints		£135	3	0			
„ Stationery and Postage		11	15	8			
					146	18	8
Less Reprints Sold		3	5	0			
Subscriptions		22	13	6			
Advertisements		64	6	2			
					90	4	8
						56	14 0
To Subscriptions written off						8	8 0
„ Stationery						12	10 3
„ Clerical Assistance and Typing						47	6 0
„ Expenses General Meeting						2	8 3
„ Wreath						1	1 0
„ Bank Charges						5	7 9
„ Honorarium						10	0 0
„ Audit Fee						5	5 0
„ Miscellaneous Expenses						10	12 8
„ Balance (Surplus of Income over Expenditure for the year)						251	12 8
						£419	0 11

INCOME.

By Subscription Accrued	£390	16	6			
Less Contribution to Benevolent Fund	38	10	0			
				£352	6	6
„ Interest Accrued on Investments				66	14	5
				£419	0	11

HISTORY OF THE SOUTH AFRICAN VETERINARY CORPS.

Then bind our realm in brotherhood,
Firm laws and equal rights,
Let each uphold the Empire's good
In freedom that unites.

F. G. SCOTT.

PREFACE

Although South Africa is rich in military history it is astonishing how few regiments have published records, records which if known to the general public would make special recruiting efforts unnecessary.

With the appearance of this résumé the S.A.V.C. joins the select group of units which possess a published history.*

This happy position has been made possible by the generosity of the S.A.V.M.A., whose Editorial Committee has my profound thanks for its encouragement and financial support.

Leadership and discipline are the essentials of any organisation. By having a published record future generations will possess traditions which will enable them to live up to the professional motto *Pristinae Virtutis Memores.*

Thanks are due to several individuals for assistance, but in particular must be mentioned the D.V.S., Col. P. J. du Toit, who allowed photographs to be prepared at Onderstepoort, and the D.D.V.S., Lt.-Col. C. J. van Heerden, who made arrangements about the typing.

Material of historical interest e.g. badges, horse-shoes, pamphlets, etc., is to be preserved in a museum.

H. H. CURSON, Captain,
S.A. Veterinary Corps.

Depot Company S.A.V.C.,
P.O. Roberts Heights.
31.12.41.

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* Other units of the Active Citizen Force which have published records are:
Royal Naval Volunteer Reserve (S.A. Division) up to 1919.
Royal Natal Carbineers (1855 – 1911).
Prince Alfred's Guard (1856 – 1906).
Imperial Light Horse (1899 – 1902).
Transvaal Horse Artillery (1904 – 1926).
Transvaal Scottish (1902 – 1932).

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* Page numbers will be inserted in the reprints of the completed work.

LIST OF ILLUSTRATIONS *

* Page numbers will be inserted in the reprints of the completed work.

ABBREVIATIONS

A.N.V.C.	African Native Veterinary Corps.
A.D.V.S.	Assistant Director of Veterinary Services.
A.V.C.	Army Veterinary Corps (now R.A.V.C.).
A.C.F.	Active Citizen Forces.
C.G.S.	Chief of the General Staff.
C.V.D.	Civil Veterinary Department.
D.V.S.	Director of Veterinary Services.
D.D.V.S.	Deputy Director of Veterinary Services.
D.M.S.	Director of Medical Services.
D.H.Q.	Defence Headquarters.
E.A.V.C.	East African Veterinary Corps.
G.H.Q.	General Headquarters.
Jl. S.A.V.M.A.	Journal of the South African Veterinary Medical Association.
K.A.R.	King's African Rifles.
L.H.	Light Horse.
M.L.A.	Member of the Legislative Assembly (now MP.).
M.R.	Mounted Rifles.
N.C.O.	Non-Commissioned Officer.
O.C.	Officer Commanding.
P.F.	Permanent Force
P.V.O.	Principal Veterinary Officer.
R.A.V.C.	Royal Army Veterinary Corps.
S.A.V.C.	South African Veterinary Corps.
S.A.M.R.	South African Mounted Rifles.
S.A.P.	South African Police.
S.A.H.	South African Horse.
T.V.C.	Transvaal Veterinary Corps.
U.G.G.	Union Government Gazette.
U.D.F.	Union Defence Forces
W.O.	Warrant Officer.

HISTORY OF THE S.A.V.C. 1910 – 1939.

INTRODUCTION

(i) Preliminary

The veterinary history of the pre-Union permanent (i.e. police) and volunteer forces has been published (Curson 1932, 1933, 1934), so in considering the story of the S.A.V.C., it is necessary to ascertain what fate befell (a) the colonial permanent forces and (b) the colonial militia or volunteer organisations.(1)

(a) As a result of the Defence Act, No. 13 of 1912, a Permanent Force of five regiments of S.A.M.R. was created as from 1.4.1923.(2) Each regiment was assigned to an area for which it was responsible for constabulary duties, and areas not so provided were policed by the S.A.P., which had arisen from the amalgamation of eight separate pre-Union police bodies (3), following the promulgation of the Police Act, No. 14 of 1912.

At the time of Union, the V.O. to the Natal Police, Inspector J. B. Collyer, was the only one remaining in any of the S.A. colonial permanent forces. He was transferred to Pretoria, attached to the 4th S.A.M.R., placed in charge of the animals at the combined training depot for the S.A.M.R. and S.A.P., and also acted in an advisory capacity to the Inspector-General, Permanent Force (Brigadier-General H. T. Lukin) on all veterinary matters.

In June, 1913, Capt. Collyer (see *U.G.G.* 15.8.1913) was transferred to the P.F. Staff and graded as a 3rd grade Staff officer. He then underwent a course of instruction at Woolwich with the A.V.C. In March, 1914, the Veterinary Hospital at Tempe was purchased from the Imperial Government and Capt. Collyer was transferred to the Military Training School, Tempe, given the title of O.C. Veterinary Training School, placed in veterinary charge of all animals at Tempe, and instructed to form a central veterinary store at the Veterinary Hospital for the supply of drugs and instruments to the S.A.P. and to the P.F. and A.C.F. Ordnance officers throughout the Union were instructed to forward all veterinary drugs, instruments and equipment to the central store and these on receipt were found to be generally obsolete and unserviceable and were reported on as such.

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(1) The Orange River Colony, in spite of Ordinance No. 35 of 1905, had no volunteer units.
(2) Four regiments were disbanded in 1922 and the 5th lost its identity on 31.3.1926.
(3) Cape Mounted Police, Cape Urban Police, Cape Rural Police, District Mounted Police, Native Territories Police, Natal Police, Transvaal Police and Orange River Colony Police.

A requisition for instruments and equipment was submitted, but at the outbreak of the First Great War none of the articles requisitioned had been supplied. At the outbreak of hostilities, the entire veterinary establishment consisted of one V.O., one corporal clerk, and one civilian storeman.(4)

It is interesting to observe in the annual report of the Staff Officer for Administrative Duties (Lt.-Col. M. C. Rowland) (year ending 30.6.1913) that the organisation of a Veterinary Service was contemplated in 1914. He adds, "In the meantime such Veterinary Services as are necessary are arranged by me."

In regard to the S.A.P., the first veterinary appointment was made on 14.11.1914, Capt. J. M. Tate having transferred from the Civil Veterinary Department.(5)

(b) In consequence of Act No. 13, 1912, referred to above, certain colonial militia and volunteer units, not veterinary, were embodied in the A.C.F. as from 1.7.1913. As has been explained previously (Curson 1934), it would appear that the Defence Force authorities at first desired veterinary services to be organised on a regimental basis and not as a corps.

The position can be best summarised by quoting a passage from a minute dated 25.7.1913 from Major W. Power, O.C. Natal Veterinary and Remount Corps, to the Staff Officer, No. 5 District, Durban, as follows:— "I feel certain that had the N.V. & R.C. been taken over in a body to the Active Citizen Force all the members would have been willing to have transferred, but under the conditions offered them, do not see their way clear to be drafted into separate regiments."

Thus the unsatisfactory position continued until the commencement of the Great War when Col. J. Irvine Smith, as will be described later, obtained approval for the gazetting of veterinary regulations which had been drafted by Collyer.(6)

A record of the S.A.V.C. falls naturally into the following periods:—

(a) Chapter 1. German S.W. African Campaign (1914 – 1915).
(b) „ 2. German East African Campaign (1914 – 1918).
(c) „ 3. Inter-Great Wars Period (1919 – 1939).
(d) „ 4. Second Great War (1939 onwards).

Before dealing with these periods, it is proposed to give other particulars of an introductory nature.

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(4) The Inspector-General in his first annual report (U.G.61.'13) for the year ending 30.6.1913 refers to a V.O. for the whole force of five regiments and the intention to organise "a Regimental and Squadron Farriery Establishment." Incidentally the V.O. reported on a large number of remounts "as being on the average a poor lot."

(5) This officer retired on pension with rank of Major on 31.10.1925. He was succeeded by Capt., now Major, D. Morton, M.R.C.V.S.

(6) These included (i) Introduction, describing the constitution of the S.A.V.C., (ii) details concerning the P.F. Section, (iii) particulars of the A.C.F. Section, (iv) Appendix A on establishments, (v) Appendix B on promotion of officers and (vi) Appendix C on promotion of other ranks.

Early Records

Data bearing on the genesis on the S.A.V.C., such as occurring in P.F. regulations(7), are difficult to obtain and the information available at D.H.Q. is scanty. Lack of space does not permit a discussion on the early legislation referring to the veterinary side of the U.D.F., but it is intended subsequently to refer briefly to the

Fig. 1.

Col. J. Irvine-Smith, c.b.e., v.d., j.p.,
F.R.S.I., Director of Veterinary Services U.D.F. 1914 - 16 and Honorary Colonel S.A.V.C. 1.2.1916.

existing position as laid down by regulations introduced after the First Great War.

Apparently no special provision was made for a P.F. Veterinary Reserve, the availability of the A.C.F. Reserve of Officers, constituted

(7) **Vide** volume of regulations brought up to date August, 1940, and printed by Cape Times Ltd., Cape Town (Chapter XI, para 86).

under section *twenty-four* of the original Act, on the conditions set down in the regulations for the Reserve of Officers (*vide* Government Notice No. 434 of 27.2.1913) being sufficient.

Regarding the A.C.F. section of the Corps, the same difficulty concerning early records exists and in spite of regulations(8), no adequate historical account is available. However, the unit as such has had only a brief existence and with the assistance of colleagues and the literature cited later, the task of compiling a record is comparatively simple.

Military Veterinary Education

As indicated above, military education, including veterinary, was to be centralised at Tempe, Bloemfontein. During the First Great War the Military School was closed and in January, 1920, was reopened at Roberts Heights, where it now exists as the S.A. Military College. The Veterinary School, originally planned to give both officers and other ranks a military background, has never materialised.

Early Scheme regarding S.A.V.C.

The P.F. section was intended for duty with the S.A.M.R. including the artillery, which was to be composed of five permanent batteries. The subordinate staff was to be drawn from the S.A.M.R. This arrangement, however, was upset by the outbreak of the First Great War.

The A.C.F. section was to have been organised so as to provide trained personnel for each military district or command, as each army area is now designated. While the S.A.V.C. was highly organised during the First Great War, the ideal of organised veterinary personnel for each military area was never attained.

Registration of Horses

Horses for the Permanent Force were provided by the Government (*vide U.G.G.* 24.12.1912). The average cost of feeding horses in S.A.M.R. districts was 1/3¼d. *per diem* and in S.A.P. areas it was 1/1½d. The Director of Supplies was responsible for the service. The Director of Transport acted as Remount Officer.

In connection with the A.C.F., a citizen's horse was to be three years old or over, 13½ h.h. or over, and fit for field service. It was to be inspected by a board containing at least an officer of the P.F. Regulations for registration, allowance, insurance, and compensation were published under Government Notice No. 1533 of 1912 (*U.G.G.* 8.11.1912), later amended by Government Notice No. 884 of 1913 (*U.G.G.* 6.6.1913).

(8) **Vide** volume of regulations brought up to date in January, 1941, and printed by Cape Times Ltd., Cape Town (Chapter XIV, paragraph 41).

Fig. 2.

LT.-COL. J. G. BUSH, D.S.O., V.D., D.V.S., 1916 - 19 and 1921 - 31.

The above photograph was taken in 1930 and shows Col. Bush in the uniform of the South African Staff Corps.

Helmet. White with white puggaree, with scarlet edging. Plume of white swan feathers 8 inches long with red feathers underneath. Plume is fixed to the helmet by means of a gilt socket which fits into a gilt base of acanthus leaf pattern. On the helmet is a gilt chain looped up from left to right.

Tunic. Full dress tunic of dark blue cloth piped down front edges, collar, cuff flaps and skirt flaps with $\frac{1}{8}$" white cloth and centre bar on centre flap behind with $\frac{3}{16}$" white piping. Scarlet collar and cuffs. The collar is laced round the top and bottom with $\frac{5}{8}$" gold lace. The collar, cuffs, cuff flaps, skirt flaps and centre bar are laced with $\frac{3}{8}$" gold lace. There are eight 1" buttons down the front, three 1" buttons on each cuff flap and three 1" buttons on each skirt flap. The shoulder cords are in gold and badges of rank in silver embroidery.

Aiguilette. Blue and red basket cord with metal tags.

Sash. Blue silk with two red stripes and blue tassels.

Gloves. White kid.

Sword. Mounted officer's sword with steel scabbard.

Overalls. Dark blue cloth with $1\frac{3}{4}$" scarlet stripes down side seams.

Boots. Half Wellington, black.

Spurs. Box.

Senior Officers of S.A.V.C.

The subjoined tabulated statement shows the position at a glance:—

Head.	*Appointed.*	*Resigned.*	*Remarks.*
Col. J. I. Smith	1.9.1914	29.2.1916	D.V.S. during G.S.W.A. campaign(9).
Lt.-Col. J. G. Bush ...	1.3.1916	28.3.1919	D.V.S. in Union during G.E.A. campaign.
Maj. J. B. Collyer ...	29.3.1919	30.6.1921	Was Staff Officer, Veterinary Services.
Lt.-Col. J. G. Bush ...	1.7.1921	1.5.1931	Decreasing demand for veterinary services.
Capt. W. S. Clapham(10)	16.2.1933	16.9.1939	
Col. P. J. du Toit ...	5.8.1939	——	Assisted by Maj. A. M. Howie from 5.11.39 to 5.5.40 when Lt.-Col. C. J. van Heerden became D.D.V.S.

Headquarters of S.A.V.C.

From September, 1914, to September, 1916, headquarters (including the Records office) was at the Drill Hall, Johannesburg. Thereafter it was at D.H.Q., Artillery Barracks, Pretoria.

The Training Depot for the two years 1914 – 1916 was at Booysens, and thereafter, in times of crisis, at the Veterinary Hospital, Roberts Heights.

Status of Unit

While provision was made for the needs of the P.F., the S.A.V.C. during both the First and Second Great Wars was essentially an A.C.F. organisation. Notwithstanding this, during the Second Great War, not only were P.F. regulations largely the guide in procedure, but the P.F. flash was worn by all ranks.

Distinguishing Marks

By day a veterinary hospital is indicated by a rectangular red flag in the centre of which is a small white equilateral triangle. By night two lighted lanterns, one above the other, at the entrance serve the same purpose. These symbols are identical with those used by the R.A.V.C.

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(9) On the creation of A.C.F. as from 1.7.1913, Smith was transferred to the Veterinary Section, Reserve of Officers (**U.G.G.** 12.6.1914).

(10) Although Capt. Clapham's designation was V.O. Permanent Force, he undertook other duties.

(ii) Uniform

GERMAN SOUTH-WEST AFRICAN CAMPAIGN

Officers: Khaki (drab veld) was worn([11]).

Headgear: A helmet, Imperial Army pattern, or dress cap. The badge was the Union coat-of-arms. Around the peak cap was a maroon band.

Fig. 3.

Major J. B. Collyer, *Staff Officer, Veterinary Services, U.D.F. 1919 - 21.*

Tunic: Officer pattern, i.e. open at neck. The unit distinction was a maroon gorget patch and no collar badge was worn. The badge of rank was indicated on the shoulder strap, e.g. three stars for a captain. The four large and ten small buttons of gilded metal contained the

([11]) "Veld" colour was adopted in 1922, i.e. grey-green.

Union coat-of-arms. A Sam Browne belt was worn, but not a lanyard with whistle. Collar, shirt, and tie were khaki.

Breeches: Riding breeches were worn with brown leather leggings, brown boots, spurs and leather shields.

When off duty khaki trousers replaced the breeches and a cloth belt the Sam Browne belt.

Rank and file — Headgear: Felt hat. Earlier in the war the brim was not turned up and the crown was thrice dented at the top. Later, the crown was indented longitudinally and the brim was turned up on the left side.

Tunic: At first the A.C.F. pattern of khaki serge, closed at the neck and with an upright green collar, was worn. Later, khaki drill was employed and the collar was either double or turned down with an open neck, exposing a khaki shirt with which was worn a khaki collar and tie. The shoulder title was of metal and consisted of the letters S.A.V.C. A leather bandolier was worn over the left shoulder. Farriers wore a horse shoe on the right arm.

Breeches, leggings, boots and spurs were of the same type, but of an inferior quality to those described above. The first A.C.F. breeches had a green stripe.

No collar badges were worn, and often a black tie was seen instead of khaki.

GERMAN EAST AFRICAN CAMPAIGN

Officers — Headgear: Same as above, only that a helmet was invariably worn. The helmet badge bore the springbok head, facing to the right. The flash was diamond-shaped, maroon in colour and worn on the left side of the puggaree. The dress cap did not have a maroon band.

Tunic: As described above. A lanyard with whistle was worn over the shoulder. Rank was occasionally denoted on home service on the cuff of the sleeve. Neither collar badges* nor maroon gorget patches were worn.

Breeches, leggings, boots and spurs: As above.

Rank and file — headgear: Same as for officers.

Tunic: Made of khaki drill and closed at the neck. The collar was double, i.e. turned down. The shoulder title was S.A.V.C. and a bandolier was worn.

Riding breeches, etc.: As for rank and file in the G.S.W. African campaign.

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* Springbok collar badges surmounted by a crown are now obtainable; but naturally are not worn by the S.A.V.C.

Fig. 4.

Col. P. J. du Toit, D.V.S., 1939.

SECOND GREAT WAR

Officers — headgear: Either a helmet U.D.F. polo type, or dress cap. The former is generally worn with riding breeches and the latter with trousers. The badge is the distinctive badge of the unit, i.e. the prancing stallion. Only the P.F. flash is worn and then on each side of the puggaree.

Tunic: As described above, the material generally being barathea. Collar badges of the unit pattern are worn. The stallion facing the badge of the opposite side. Rank is indicated on the shoulder strap, at the base of which is a red (so-called orange) tab one inch in width signifying willingness to serve outside the Union of South Africa. The one P.F. officer (Major Howie) wears the distinctive P.F. button (springbok) whereas other officers wear the Union coat-of-arms.

In hot weather the tunic may be removed whereupon rank is indicated on the shoulder strap of the khaki shirt. In such cases a Sam Browne belt is also to be worn.

Riding boots are laid down for wear with breeches, but brown leggings are frequently worn with riding kit.

Rank and file — headgear: A helmet or dress cap (drill material *not* barathea) is worn, the former with riding kit. The unit badge is issued to the rank and file. All ranks wear the P.F. flash.

Tunic: Pattern as described above. In addition to a collar badge the shoulder title $\frac{\text{S.A.V.C.}}{\text{S.A.V.D.}}$ or $\frac{\text{S.A.V.C.}}{\text{Z.A.V.D.}}$ is worn. As in the case of officers, volunteers for the duration of the war wear buttons showing the Union coat-of-arms and orange tab. Web equipment with rifles is laid down.

Riding breeches, etc.: As above.

The D.V.S. as a staff officer wears the uniform, not of the unit, but of the Staff Corps. During the First Great War the cap band was blue, but for several years the colour has been red as in the Imperial Army.

The uniform worn by A.C.F. units, e.g. No. 1 Veterinary Hospital personnel was of green veld (adopted in 1922) and the pattern is as shown in Fig. 31.

Badges

(a) *Union coat-of-arms*: This was brought into use in 1913 and was worn first by members of the U.D.F. Staff. It was adopted during the S.W.A. campaign by units which did not possess a distinctive badge, e.g. S.A.V.C., Transport and Remounts, etc. On reorganisation of the U.D.F. in 1922, the badge *without* the bottom pointer was adopted by the S.A. Instructional Corps and is still worn by members of that unit.

The coat-of-arms is surrounded by a circle bearing above the words "South Africa" and below "Zuid Afrika." Surrounding the arms is a crown and below frequently a "pointer." Since 1923 the Afrikaans version "Suid Afrika" has replaced the Dutch form. See Fig. 9.

(b) *Springbok*: This badge was first worn in 1915 by South African troops which proceeded to East Africa, Egypt and Europe. Surrounding the head of a springbok is a circle on which is inscribed "Union is strength" above and "Eendracht maakt macht" below. From 1923 the Afrikaans equivalent "Eendrag maak mag" has replaced the Dutch form.

Fig. 5.

LT.-COL. C. J. VAN HEERDEN, D.D.V.S., 1940.

The badge was worn by the S.A.V.C. in the G.E.A. campaign. See Fig. 10.

(c) *S.A.V.C.*: A prancing stallion on a "scroll" containing the letters S.A.V.C. – Z.A.V.D., the corps initials in English and Dutch

respectively. In 1923 the letters S.A.V.D. replaced Z.A.V.D. Above the stallion is a crown. The badge was first adopted in 1921, having clearly been derived from the coat-of-arms of the Royal Veterinary College, London, the right supporter being a rearing stallion.

Fig. 6.

MAJOR A. M. HOWIE, O.B.E., A.D.V.S., 1940.
Photo: J. Dodgson, Pretoria.

The badge of gilded metal is frequently blackened and is worn by members of the P.F. and A.C.F.

The stallion faces right in respect of the "cap" badge, but when worn in the collar, the stallion is turned towards the badge of the

opposite side. From 1923 the collar badge has been smaller than the "cap" badge([12]). See Fig. 11.

During the Second Great War, it has been issued to all ranks.

Buttons

(a) *Union coat-of-arms*: This pattern was used until 1923 for both the P.F. and A.C.F. Since then, however, it has been worn only by the A.C.F. See Fig. 12.

(b) *Springbok*: This button introduced in 1923 for the P.F. represents a springbok's head surrounded by a circle in which is inscribed, left side "South Africa," and right side, the Dutch or Afrikaans equivalent. The pattern so described forms the centre of a quadrilateral figure arranged cross-wise, the upper angle being concealed by a crown which surmounts the circle. See Fig. 13.

Shoulder Titles

(a) *S.A.V.C.* These letters fashioned of gilt metal were worn until 1921.

(b) *S.A.V.C.*

Z.A.V.D. From 1921 the English and Dutch initial letters of the unit name were worn.

(c) *S.A.V.C.*

Z.A.V.D. It is understood that from 1923 the Afrikaans equivalent S.A.V.D. has been used.

Officers do not wear shoulder titles. See Figs. 7 and 8.

Arm Badges

A farrier is distinguished from a dresser by a horseshoe, worn on the right arm of the tunic or shirt, the toe being upwards. If an

([12]) Col. Bush, formerly D.V.S., has informed the writer that originally it was intended that the motto **Vis unita fortior** should appear on the "scroll" as in the first Natal Veterinary Corps.

(Captions to illustrations on opposite page.)

Fig. 7. — *Shoulder title, S.A.V.C., worn until 1921.*
Fig. 8. — *Shoulder title, S.A.V.C., worn from 1921.*
Fig. 9. — *Helmet badge (across = 1⅜"), worn by S.A.V.C. during German South-West African campaign.*
Fig. 10. — *Helmet badge (actual diameter 1½"), worn by S.A.V.C. from 1916 to 1921.*
Fig. 11. — *Helmet badge, S.A.V.C., worn from 1921.*
Fig. 12. — *Tunic button, worn by all ranks up to 1923 and since then only by A.C.F.*
Fig. 13. — *Tunic button, worn by members of Permanent Force from 1923.*
Fig. 14. — *Arm badge, worn by shoeing smiths and distinguishes them from dressers.*

FIG. 15.

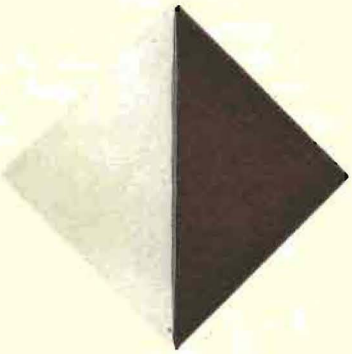

FIG. 15A.

FIG. 16.

FIG. 17.

FIG. 15. — *Flash worn by S.A.V.C., Permanent Force. Actual size* 2½x2".

FIG. 15A. — *Flash worn by S.A.V.C., Cape Sect., 1940-41. Actual size* 2x2".

FIG. 16. — *Flash worn by S.A.V.C., A.C.F., Transvaal. Actual size* 2½x2".

FIG. 17. — *Flash worn by S.A.V.C., A.C.F., Natal. Actual size* 2½x2".

N.C.O. it is worn above the chevrons whether on tunic or shirt. If a W.O. Class II., whose badge of rank, a crown surrounded by laurel wreath, is worn on right lower arm, 1 inch above point of cuff, the horseshoe is *below* the crown and wreath. This refers to the tunic and great coat. When in shirt sleeves the arm badges are worn in the same position, but *above* the elbow joint.

A W.O. Class I. has exactly the same arrangement except that the badge of rank is the Union coat-of-arms. See Fig. 14.

The above arrangement applies to saddlers except that the technical badge is a bit.

Flashes[13]

(a) Diamond-shaped and maroon in colour, worn on the left side of the helmet during the German East African campaign.

(b) P.F. pattern 3 inches x 2 inches with square-cut edges. Maroon with 1 inch horizontal black stripe across the centre. See Fig. 15a.

(c) A.C.F. 1st Mobile Veterinary Section. The flash consisted of horizontal stripes of blue, maroon, and chocolate brown. See Fig. 17. Blue indicated the Natal origin of the section, maroon its veterinary nature and brown the fact that it was a departmental unit of the A.C.F.

(d) No. 1 Veterinary Hospital. The flash was similar to the above except that a green stripe, representing Transvaal, took the place of the blue stripe. See Fig. 16.

(To be Continued.)

[13] It is interesting to note that a flash on the helmet was worn by the Royal Artillery during the Anglo-Boer War (1899 - 1902).

LAMENESS IN A STUD CATTLE HERD POSSIBLY OF HEREDITARY ORIGIN

G. C. VAN DRIMMELEN
Bloemfontein

A number of cases of a progressive, unilateral lameness of a hind leg occurred in a herd of registered cattle in the course of three years. The pathology of this peculiar lameness is not fully understood, but the evidence points to an hereditary etiological factor.

As reports of obscure lameness in dairy cattle are by no means rare, the history of these cases is recorded for comparison.

Symptoms

These were very similar in all the cases described, but their duration varied as did the ages of the subjects. At first a slight stiffness of one hind leg was observed, noticeable when walking but not when trotting. This stiffness was of the nature of an excessive extension of the affected leg, which developed gradually or rapidly into a state in which the leg was not used at all. Secondary symptoms found were those of aseptic deforming arthritis of the hock, atrophy of the gluteal muscles, deformity of the feet due to unequal and abnormal wear, recurrent tympanitis and decubitus.

Various treatments were applied, mainly for diagnostic purposes, without success. Pathological investigations were not conclusive, notwithstanding the large amount of nerve, bone, muscle and other material examined.

A description of the cases encountered will now be given.

CASE 1: A three-months-old heifer developed a stiff hind leg a few days before transfer to another farm. In reply to my enquiries I was informed that she had subsequently been slaughtered, as she was believed to be suffering from a pelvic injury sustained during the train journey.

CASE 2: Another heifer became stiff in the right hind leg at the age of four months. I suspected muscular rheumatism and the animal received 15 g. sod. salicylate daily for 30 days. This had no effect on the condition and as a result of consultation with the Director of Veterinary Services, Onderstepoort, the possibility of a mineral deficiency was considered. The ration, however, proved to be perfectly balanced. The tuberculin test was then applied but with negative results. Vigorous massage for 21 days did not arrest the progress of the symptoms. A course of 4 g. pot. iodide per day for 14 days had no effect.

The typical symptoms were now plainly evident: The animal lay down most of the time, but could stand, walk and canter on three legs. In the standing position, the right hind leg was held slightly backwards, the toe resting on the ground, the hock and stifle extended. When walking this leg would be swung forwards and backwards, the toe scratching the ground, but the limb never carrying any weight. When running, both hind legs would move together, the right hind leg apparently assisting somewhat in propulsion. The tips of the hooves were worn, the hock surrounded by hard swellings and the gluteal musculature atrophied. The body otherwise showed nothing unusual, even the external bone measurements being perfectly symmetrical.

At nine months old the animal was killed for post-mortem examination and special attention was paid to: (a) the skeleton, which showed nothing unusual, (b) the joints which showed hypertrophic tarso-metatarsal ligaments in the right hock and (c) the muscles which showed slight right gluteal atrophy and marked atrophy in the right semitendinosus. Microscopically the latter showed wasting of the muscle fibres. There were degenerative changes in the fibres of all the nerve trunks in the right hind leg, and worn hooftips and hypertrophy of the subcutaneous connective tissue round the hock were observed.

CASE 3: A three-months-old bull calf became stiff in the right hind quarter. A 30-day course of 15 g. sod. salicylate daily had no apparent effect, but 14 days after completion of this treatment the condition passed off completely.

CASE 4: Twenty-two days after birth a bull calf became stiff in the left hind leg. 30 g. cod liver oil twice daily for 14 days had no effect. The tuberculin and contagious abortion tests gave negative results. The blood calcium was shown to be 10 mg. per 100 cc. and the phosphorus 5·9 mg.

The development of the symptoms was slow and they were carefully observed, the lameness increasing progressively. At four months old (see fig. 1) no patellar reflex could be demonstrated. Flexion of the leg by hand became more difficult and although the stage of a complete swinging leg lameness had been reached, yet the ability to scratch voluntarily with the left hind foot was retained. Muscular atrophy was slight but femoral nerve anæsthesia (local) using up to 10 cc. of a 2% solution had no effect on the lameness.

At nine months old this case was also slaughtered for post mortem. Microscopical findings were similar to those in case 2, except that no degeneration of the nerves could be detected. Multiple thrombi in the smaller arterioles of the lumbar pia mater and hæmorrhages in the grey matter of the lumbar cord were found.

CASE 5: Another bull calf at 25 days old showed stiffness in the right hind leg. At five months the leg was still used slightly when walking and more when running. At seven months it was no longer used. At eleven months, when the animal was forced to stand on

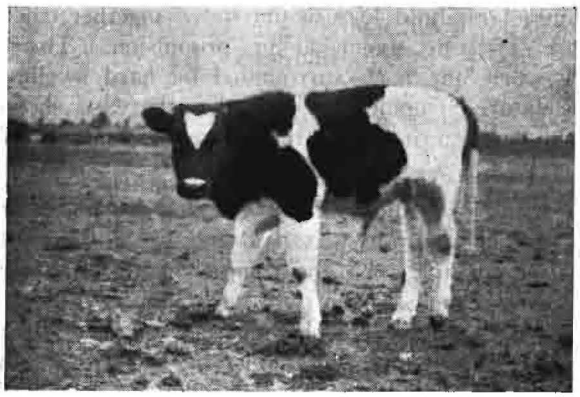

FIG. 1.

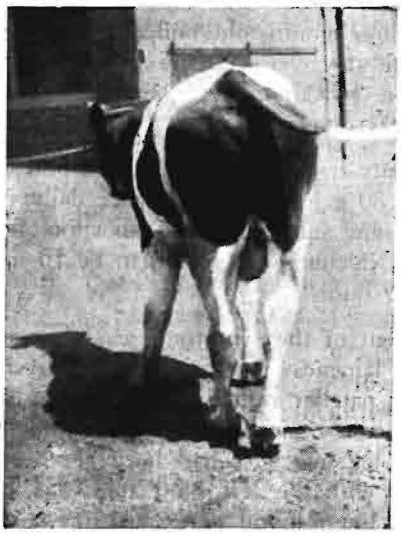

FIG. 2.

the affected leg by flexing and tying the left hock in a figure of eight with rope, the leg collapsed within a few seconds. This was repeated several times. Tying the healthy leg to the body was tried as well, yet at twelve months this animal was seen to scratch behind the right ear with the right hind foot!

When the animal was five months old femoral anæsthesia produced an effect in 30 minutes: the foot though carrying no weight was held in a normal position when standing. When walking total paralysis of the quadriceps muscles was evident. A week later saphenous nerve anæsthesia resulted in a striking release of tension in the hock. The leg was held normally when standing and flexed normally when walking. Tuberculin and contagious abortion tests gave negative results. Blood calcium was 9·3 mg. and phosphorus 4·7 mg. per 100 cc. When between 12 and 18 months old this bull was made to serve several grade cows, and two normal calves resulted. Unfortunately efforts to breed him to his mother or to other cows that had produced affected calves failed owing to the lack of interest of the authorities concerned.

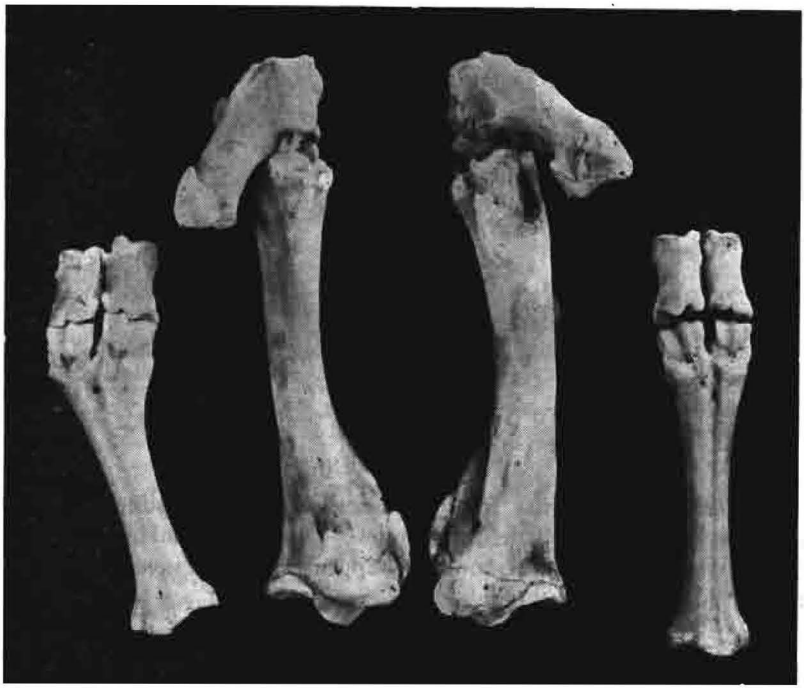

FIG. 3.

He was kept under observation until three years old, when his healthy hind foot was so deformed from carrying excessive weight and from his unnatural stance, that it would have served no useful purpose to have retained him. The toes of the affected foot had grown out and the hock was markedly deformed. (See fig. 2.)

Post-mortem findings were: severe arthritis chronica deformans of the right tibio-tarsal joint, apparently due to over-extension, and rarefying ostitis of the shaft of the tibia due to pressure by the tuber calcis. (See fig. 3.)

CASE 6: The sire of cases 2 to 5 as well as of 52 normal calves had a very slight lameness at the age of three years and nine months. A few months later he had lost the use of his right hind leg and muscular atrophy had set in (see fig. 4). He died a month later when almost four years old. The treatment consisted of massage and remedial measures for constipation, hoven and decubitus. The tuberculin test gave negative results. Examination of blood-smears revealed a neutrophilia (53%).

At post mortem nothing of interest was discovered excepting irregular extravasations in the grey matter of the lumbar spinal cord.

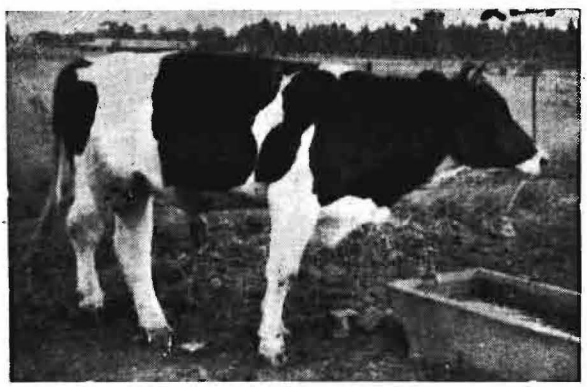

FIG. 4.

DISCUSSION

A striking similarity was encountered in the symptoms shown by the animals particularly in their course. The variation in the time of development of the total lameness appeared to be inversely proportionate to the age of the subject.

All were born without visible defects. At varying ages a noticeable incoördination of movement of one hind limb was seen. After varying periods of time the affected leg would, on the animal's rising from a lying position, become permanently and forcibly extended. Atrophy of the hip muscles would then commence and would be followed by permanent deformity of the foot, leg and joints of the affected limb with compensatory hypertrophy of the overburdened healthy leg. In one case death was due to secondary lesions sustained early in this stage.

The underlying cause of this affection could not be determined from the investigations carried out. However, it appears that hereditary influences may have played a major part. The points to be considered are: (1) the original female stock of this herd was derived from an

inbred herd established in isolation more than a century ago, (b) the breeding policy has been that of close line breeding for many years, (3) the cases occurred only in one bull and in about 8% of his progeny.

From the breeder's point of view the condition described must be considered as likely to occur very rarely. In the families from which the cattle were derived, close inbreeding has been practised with success, but has the disadvantage that it may cause some recessive condition to appear which may result in heavy financial loss to the breeder. In the case of the condition under consideration where the animals were valuable, the financial loss could not have been much less than £450 plus the cost of maintaining the animals.

Summary

1. A lameness in cattle, in which the principal feature was the forcible overextension of one of the hind limbs, has been described.

2. The secondary pathological changes were tibio-tarsal arthritis chronica deformans, atrophy of the stifle and hock flexor muscles, and other deformities.

3. The etiology is obscure but hereditary factors are apparently involved.

Acknowledgement

We appreciate the kind advice received from Dr. A. D. Thomas, of Onderstepoort, Dr. P. S. Snyman, S.V.O., Bloemfontein, and Dr. E. M. Robinson in connection with this publication.

SKAAPWURMS IN DIE KARO: DIE ROL VAN NEMATODIRUS

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Onderstepoort

Tot sowat nege jaar gelede het die Karo uitgeblink as dié deel van die Unie wat nie deur wurmparasiete van skape geteister is nie. Die oop grond tussen die karobossies en die relatief droë klimaat is baie ongunstig vir die meeste soorte wurms, wat veel beter in die grasveld aard, omdat die gras 'n bedekking oor die grond daarstel waarin die wurmeiers en larwes die skaduwee en vogtigheid vind wat hulle so nodig het totdat die taaier besmetlike stadium bereik is.

Met die groot droogte van 1933 egter, nadat baie trekkery plaasgevind en die karoskape allerlei wurms saam teruggebring het, of ook skape van elders wurmbesmetting in die Karo ingebring het, het oral moeilikhede en sterftes as gevolg van wurms voorgekom. Gedeeltelik was die moeilikhede ook daaraan te wyte dat die karoboere min of geen kennis van wurms en hul bestryding gehad het nie.

Nietemin was die verwagting dat die moeilikhede tydelik sou wees, omdat die Karo nie juis geskikte omstandighede vir die voortbestaan van swaar besmettings bied nie en goeie voeding, wat die weerstandsvermoë van die skape versterk, deur die karobossies gelewer word.

Nou is dit egter die algemene ondervinding dat die moeilikhede sedert 1933 gedurig erger geword het. Wat is die oorsaak hiervan? Die algemene omstandighede in die Karo het nie gunstiger vir die wurms geword nie en die enigste plekke waar besmettings werklik gunstige kondisies kry om te ontwikkel is om damme en oorlopende of lekkende drinkbakke, waar daar gras groei en altyd vogtigheid is, of ook op natleilande waar op die skape mag gebring word as die lande nog nie behoorlik droog is nie. Sulke plekke is 'n groot gevaar en moet vermy word. Maar tog skyn daar nog 'n faktor nodig te wees, en veral een wat die vatbaarheid van die skape verhoog, om die erge graad van besmetting wat nou dikwels voorkom te verklaar. Dit lyk baie waarskynlik dat *Nematodirus* hierdie faktor daarstel.

Nematoridus, die dunnek- of langnekwurm, is dun en sowat 2 sm. lank, ligrooi van kleur, en lê op die slymvlies van die dunderm met sy voorste helfte gewoonlik opgekrul. Die meeste wurms word gevind in de jejunum van omtrent 3 tot 20 voet agter die pylorus en kan maklik gesien word.

Die wurmopname wat 'n paar jaar gelede gemaak is het gewys dat *Nematodirus* uitsluitend in die Karo voorkom. Dat hy daar so

goed kan aard word verklaar deur die feit dat sy eiers, wat besonder groot is, nie uitbroei nie totdat die larwes tweemaal vervel en dus die besmetlike stadium bereik het, wanneer hulle baie taai is.

Hierdie parasiet word gewoonlik nie as erg patogeen beskou nie, maar uit die westelike dele van die V.S.A. is berig dat dit swaar besmettings, agteruitgang en selfs verliese onder jong skape veroorsaak en die Staatsveeartse van Middelburg, K.P. (mnr. Sutton) en Beaufort Wes (mnr. Ryksen) het herhaaldelik dergelike ondervindings gehad. By 'n onlangse besoek aan die Cradock distrik het die skrywer 'n aantal skape van verskillende plase ondersoek en in almal *Nematodirus* gekry, waarvan party swaar besmettings was.

'n Verdere opmerklike feit is dat juis in die Karo gedurende die laaste tyd die mening ontstaan het dat tetrachlooretileen doeltreffend is teen knoppieswurms, omdat skape wat 'n tydlank gereeld daarmee behandel is die wurms kwytraak. Etlike boere het hulself nou al oortuig dat dié middel nie knoppieswurms doodmaak nie. Die feit is dat tetrachlooretileen baie doeltreffend is teen *Nematodirus* (tegelykertyd ook teen haarwurm en bankrotwurm) en heel waarskynlik is die gevolg dan dat die skape, nadat die verswakkende faktor verwyder is en hul weerstand deur die goeie voeding van die karobossies versterk is, die knoppieswurms outomaties kwytraak, soos dit onder dergelike omstandighede welbekend is.

Gevolglik moet aanbeveel word dat skape in die Karo gedurende die droë seisoen af en toe met tetrachlooretileen („tetrol") behandel word en gedurende die groen weiveld tydperk elke drie weke met knoppieswurmmiddel wat, sê elke tweede keer, deur tetrachlooretileen vervang word.

Summary

The dry Karoo with its highly nourishing scrub vegetation is not very suitable for the propagation of worm parasites of sheep. The fact that worm infestations, introduced into this area through large-scale movements of sheep to and from other areas during the 1933 drought have persisted there, is ascribed to *Nematodirus,* which normally occurs only in the Karroo. Although not very pathogenic itself, *Nematodirus* lowers the resistance of the sheep and so favours infection with other worms. Treatment with tetrachlorethylene is recommended.

A REPORT ON THE FAILURE OF ACAPRIN TO CURE *BABESIA BIGEMINA* INFECTION IN BOVINES

H. P. STEYN
Onderstepoort

For years it has been fairly generally accepted here that *B. bigemina* (*Piroplasma bigeminum*, Smith and Kilborne, 1893) infection can be cured by a single injection of acaprin and although there have been a number of field reports to the contrary, no definite proof of the failure of acaprin has been presented. The observations here recorded are not based upon a planned experiment, but it is considered that they were sufficiently well controlled to warrant the conclusion that acaprin may, at times, not be a reliable treatment for *B. bigemina* infection.

The cows upon which this report is based were the property of a small dairy at Pretoria North. They had been recently purchased from a part of the Orange Free State where piroplasmosis is unknown. After arrival at the dairy they were kept in a small tick-free paddock and most of them inoculated with the Onderstepoort redwater and gallsickness vaccine. About 14 – 21 days after inoculation they were allowed out on the veld to graze. They then became very heavily tick infested and one animal died of heartwater. After this death the whole herd was confined to the original small paddock. It was during this exposure to tick infestation that the first relapses occurred. One animal became infected and reacted during the period of exposure.

CASE 1: Breed, Friesland; age, about 5 years; weight, about 1,000 lbs.; condition, fair.

22.4.42. Inoculated with B. bigemina and Anaplasma centrale blood.

30.4.42. Reacted to B. bigemina and 6 cc. acaprin injected. Smears positive for B. bigemina.

2.5.42. 6 cc. acaprin injected. There was marked improvement and apparent recovery. On about 18.5.42 there was a rise in temperature and general illness and although a smear was not examined a relapse of redwater was diagnosed and 6 cc. acaprin injected.

6.6.42. Animal again obviously ill, off feed and milk yield dropped markedly. Smears were found positive for B. bigemina and 20 cc. gonacrin injected intravenously.

16.6.42. Animal reported to be seriously ill and very weak. 20 cc. gonacrin again injected intravenously. B. bigemina very frequent in smear, marked anæmic changes in blood.

17.6.42. 100 cc. piroblue injected intravenously and blood transfusion given. Animal died some hours later.

Smears taken on the 16th were controlled by Mr. Neitz and the diagnosis confirmed.

CASE 2: Breed, Grade Friesland; age, ± 8 years; weight, ± 800 lbs.; condition, poor.

22.4.42. Inoculated with blood from same bottle as case 1.

8.5.42. Reacted to B. bigemina and given 6 cc. acaprin.

23.5.42. Relapse to redwater. Treatment with acaprin repeated.

24.5.42. 6 cc. acaprin administered.

1.6.42. Temperature 104°F, obviously ill and off feed, weak and anæmic. 6 cc. acaprin injected.

22.6.42. B. bigemina seen in smear. Anæmia marked and animal still sick. 1 gm. piroblue in 100 cc. saline administered intravenously. Improvement now set in but was slow and 5 grains of arsenious oxide per day was prescribed. Steady improvement and recovery followed.

CASE 3: Breed, Grade Friesland; age, ± 2½ years; weight ± 700 lbs.; condition, poor.

22.4.42. Inoculated with redwater and gallsickness vaccine.

24.5.42. Animal reported sick and B. bigemina infection diagnosed. 6 cc. acaprin injected.

25.5.42. Acaprin treatment repeated.

1.6.42. Evening temperature 106°F, marked anæmia. 20 cc. gonacrin administered intravenously.

13.6.42. Unthrifty, anæmic, in very poor condition and off feed. Gonacrin treatment repeated. Thereafter there was slow but steady improvement. Occasional smears taken were negative.

CASE 4: Breed, Grade Jersey; age, ± 3 years; weight, ± 800 lbs.; condition, good.

13.6.42. Animal reported sick and B. bigemina seen in smears. 20 cc. gonacrin injected intravenously.

29.6.42. Very anæmic, poor in condition, off feed and obviously ill. Numerous B. bigemina and a fair number of Spirochæta theileri were seen in smears, the diagnosis being confirmed by Mr. Neitz. Marked anæmic changes in blood. Piroblue 1 gm. injected intravenously. Improvement was slow and unsatisfactory and after four or five days 5 grains arsenious oxide were given daily per os for a week. This was followed by slow but steady recovery.

In each of the four cases recorded the initial treatment was followed by improvement in the general condition and appetite but in no case was recovery complete. After a period of improvement there was a gradual loss of appetite and slow weakening. Although temperatures were not regularly recorded they were not usually very high after the initial attack, reaching from 103°F to 104°F and often being much lower. The condition can best be described as chronic piroplasmosis.

Discussion

There can be no doubt that acaprin has given fairly good results in the treatment of B. bigemina for many years, but the author had an experience similar to that recorded here in an imported Shorthorn bull in the Vryburg area during 1937. This bull, however, did not become as seriously ill as the cows reported upon here and recovered after the treatment with acaprin had been repeated following a relapse.

It is apparent from the results obtained in these cases that acaprin failed to bring about permanent recovery in three cases and that gonacrin similarly failed in one case. Piroblue, however, brought about recovery in three of the relapsed cases. The use of arsenic in some of these cases appears to have been beneficial. In Case 1 in which piroblue failed the animal was in extremis when treated and died in spite of a small blood transfusion given at the time of treatment.

The question now is, why should acaprin have failed so dismally when up to the present it has given fairly good results? There are certain factors which may have predisposed to these severe relapses, viz. that these were all highly susceptible lactating cows which had been exposed to exceptionally severe tick infestation. These factors may be extremely important in that the author has found that lactating cows may suffer from some degree of anæmia (work to be published) as a direct result of lactation when the nutritional state is poor. Severe tick infestation of an animal which is already bordering on a state of anæmia must adversely affect such an animal's resistance to blood parasites such as B. bigemina and these factors probably played an important part in causing the persistent relapses here recorded.

The piroblue used was manufactured by Sandoz Chemical Works, Switzerland, and although it cannot definitely be stated whether it was T 18 or T 19 it is reported to be the same product as that with which Theiler (1930) worked and which was subsequently sold as piroblue.

Reference

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INCIDENCE OF TUBERCULAR MASTITIS IN DAIRY COWS

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The significance of bovine tuberculosis in public health is well recognised overseas and it is only necessary to quote a single investigation to show that the problem can be a grave one. In a report published by the People's League of Health (1932) it is stated that in Great Britain 4,000 new cases of the bovine type of tubercular infection of man developed annually and that about 2,000 deaths (chiefly amongst children) occur yearly from this cause. In South Africa, however, although tuberculosis of man is prevalent enough, there are only two cases on record [Harington and Emmerson (1939) and du Toit and Buchanan (1942)] where the infection was due to the bovine type of tubercle bacillus. This point is always used by obstructionists when the question of compulsory pasteurisation of milk is mooted and when schemes for the eradication of bovine tuberculosis are considered.

Up to the present, few figures have been published concerning the extent to which South African milk supplies are contaminated with tubercle bacilli and the object of this article is to present such information as is available. The figures reported here are abstracts from the reports on the routine milk control carried out by the Veterinary Department of the Johannesburg Municipality. This work was started in 1931 by Martinaglia, G. and later was carried on by Robinson, M. C. and Pullinger, E. J., and the information given represents the combined findings of all three workers. Thanks are due to Martinaglia, Robinson and to the Director of the Municipal Abattoir and Livestock Market, Johannesburg, for permission to publish the data. It should be understood that the figures quoted do not include all cases of bovine tuberculosis encountered during the period 1931 – 1942, but only those cases which were adequately investigated and reported upon. Furthermore, the investigations refer only to herds situated on the high veld.

In the cases quoted a diagnosis of tuberculosis was never based upon a clinical examination alone, but confirmation was effected by microscopical and biological examination of milk, pus, sputum, etc. The precise technique used has varied over the years, but all samples recorded in Tables 3 and 4 were examined as indicated by Pullinger (1934).

TABLE 1.

*Examination of milk from cows suspected of suffering from tubercular mastitis.**

No. Cows examined	CAUSE OF MASTITIS					Percentage Tubercular Mastitis
	Tubercle	Strep.	Staph.	C. pyogenes	Cause undiagnosed. Not tubercle	
419	21	211	15	16	156	5·0%

* This list only includes cases highly suggestive of clinical tubercular mastitis.

TABLE 2.

*Post-mortem examinations of tuberculous cows, the udders of which did not show clinical signs of tuberculosis.**

Extent of Tubercular Infection	No. Cases	No. cases showing:—		% cases with T.B. udders
		Non-tuberculous udder	Tuberculous udder	
Generalised ...	75	61	14	18·7%
Pulmonary ...	32	32	—	—
Abdominal ...	2	2	—	—
Local Glandular	11	11	—	—
TOTAL CASES	120	106	14	11·6%

* Tubercular infection of supra-mammary gland is taken as presumptive evidence of tubercular mastitis.

TABLE 3.

*Ten-gallon churn samples from individual herds examined biologically for Tubercle bacilli.**

No. samples examined	Tubercle bacilli		Percentage samples contaminated with Tubercle bacilli
	Present	Absent	
40	1	39	2·5%

* Such samples contain milk of several cows which has mixed with more milk on the cooler.

TABLE 4.

*Composite pooled samples from wholesale depots and milk shops.**

Number samples examined	No. farms supplying milk	Tubercle bacilli		Percentage samples contaminated with Tubercle bacilli
		Present	Absent	
40	234	1.	39	2·5%

* The smallest bulk was a composite sample from one herd, the largest contained the milk of 59 herds, but the majority lay between 3 – 10 herds.

DISCUSSION

The bulked samples which were examined (see Tables 3 and 4) constitute a fair cross section of the class of milk consumed by the Johannesburg public: the large composite samples of 10 or more producers represent the milk handled by the large pasteurising depots, the samples of 1 to 10 producers represent the milk supplied by producer-retailers and milkshops, whilst the 10-gallon churn samples represent the supplies of small milk shops. Incidentally the 234 producers covered by the survey represent about half the producers permitted to bring milk into Johannesburg.

Over the whole series of 80 samples the degree of contamination with tubercle bacilli is 2·5%, a comparatively low figure compared with those from overseas which vary from 2 to 21% for herd samples, and 30 to 100% for composite samples from city supplies (see Hammer 1938 for American figures and Pullinger 1934 for those referring to Great Britain). This low degree of contamination may explain the rarity of cases of bovine infection in man in South Africa.

It is not clear why our bulk supplies should so seldom be contaminated with tubercle bacilli since tuberculosis is widespread throughout our dairy herds. It cannot be that tubercle bacilli are "diluted out" when large supplies of milk are pooled, for in London 100% of 63 3,000-gallon rail tanks were found to contain tubercle bacilli, whilst the milk of tuberculous cows was diluted one million times without loss of infectivity for guinea pigs (Pullinger 1934). It would seem, therefore, that the incidence of tubercular mastitis amongst South African dairy cows is comparatively low. In Great Britain it is estimated (People's League of Health, 1932) that 0·2% of all cows are affected with tuberculosis of the udder. In the present investigation, five of the bulk samples represented the composite milk of about 7,000 cows. Nevertheless, none of these samples contained tubercle bacilli whereas on the British basis 14 of these cows should have had tuberculous udders.

The figures given in Table 1 lend colour to the contention that tubercular mastitis is rare, for only 5% of samples of milk collected

from diseased udders contained tubercle bacilli. If it is remembered that these samples came from udders showing changes closely resembling tubercular mastitis it will be realised how low the percentage of positives is. But is must also be understood that by far the majority of cows concerned were apparently healthy, except for udder disturbance, and certainly showed no obvious signs of a generalised disease such as tuberculosis.

In Table 2 are given the results of post-mortem examinations of cows showing clinical signs of tuberculosis. The 21 cows noted in Table 1 are not included in this group. It will be seen that the udder was involved in 18·7% of 75 cases where the disease was well established and it was not involved in the 45 cases where the disease was comparatively localised. These figures suggest that generally speaking, infection only appears in the udder when tuberculosis is well established throughout the system, and under high-veld farming conditions where cows lose condition every winter as the result of drought, cold, tick infection and under-feeding it seems likely that tuberculous animals cease to be profitable milk producers long before there is time for their udders to develop infection.

Although the incidence of tubercle contamination of milk supplies is low, this is no reason for complacency. So long as bovine tuberculosis is allowed to spread through our dairy herds practically unhindered, there is every probability that tubercular mastitis will become more prevalent as dairying methods become more intensive. In any case, the present trend towards centralisation and pooling of milk supplies will aggravate the public health problem, a problem which can immediately be answered by controlled and efficient pasteurisation. The ultimate answer, of course, lies in a country-wide campaign for the total eradication of bovine tuberculosis.

Summary

Only 2·5% of samples of bulked milk in Johannesburg were found to be contaminated with tubercle bacilli. This low figure is thought to be due to the fact that tuberculous cows are usually culled from dairy herds before their udders develop infection.

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SULPHONAMIDES — THEIR COMPARATIVE EFFICACY IN BACTERIAL AND OTHER INFECTIONS, RELATIVE TOXICITY, DANGERS AND PROPHYLAXIS

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1. Introduction

As sulphonamides are being very extensively used in the treatment of many different diseases, it was thought advisable to record here briefly, in the light of recent literature, the most important points in regard to their comparative efficacy in the treatment of bacterial and other infections, their doses, methods of application, relative toxicity, prophylaxis, dangers, and the treatment of cases of poisoning with them. A large amount of useful information is available as a result of the extensive application of sulphonamide preparations in the treatment of human diseases. Wherever doses for man only are available these could be calculated for the different kinds of animals on the basis that a dog, approximately the size of an Alsatian, receives the same dose as an adult person and that sheep and goats receive about four times, and horses and cattle about sixteen to twenty times this dose.

In this review special attention is paid to sulphanilamide, sulphapyridine, sulphathiazole, sulphaguanidine, and sulphadiazine, as these comprise the most extensively used sulphonamides.

II. Comparative efficacy of sulphonamides in the treatment of bacterial and other infections

(a) *Virus diseases*

Information about the effect of sulphonamides on virus diseases is scanty and much more experimental work is needed before definite conclusions can be drawn. This group of drugs is valuable, however, in the treatment of secondary infections in virus diseases. Very good results have been reported in the treatment of the meningo-encephalitis of distemper in dogs with sulphanilamide, whilst in the general treatment of cases of distemper with this drug and sulphapyridine less favourable results were achieved.

Favourable results with sulphonamides have been claimed in the following virus diseases: the common cold, lymphocytic choriomeningitis, canine distemper, influenza, and lymphogranuloma inguinale (Findlay, 1939).

(b) *Bacterial diseases*

In *in vitro* experiments the effect of sulphaguanidine and sulphanilamide as growth-inhibitors of Bacillus coli was found to be 64 times less than that of sulphathiazole.

On the basis of equivalent blood concentrations, sulphadiazine was as effective as sulphanilamide in streptococcal infections and as sulphathiazole in staphylococcal infections, but less effective than sulphathiazole in pneumococcal infections. Good results are reported in the treatment of severe hæmolytic streptococcus infections in horses with sulphanilamide. Sulphanilamide appears to be of no value in streptococcic mastitis.

According to Barlow and Homburger (1939) " sulphathiazole and sulphamethylthiazole prolong the life of mice infected experimentally with a highly mouse virulent strain of Staphylococcus aureus and prevent the development and allow healing of abscesses in kidneys and other organs in a significant number of animals." " Clinical and experimental experience has shown that Staphylococcus infections are either influenced or at least slightly affected by sulphanilamide, sulphanilyl dimethyl sulphanilamide, prontosil, uleron, di(p-formylaminophenyl) sulphone and di(p-butylaminophenyl) sulphone. On the other hand, sulphanilyl sulphanilamide and sulphapyridine seemed to have some curative effects on experimental staphylococcal infections in mice." The bactericidal action of sulphathiazole on different strains of Bacillus coli may vary markedly, fortunately the number of resistant strains is small.

The bactericidal effect of sulphanilamide is greater in alkaline than in acid urine. However in high concentrations it is also bactericidal in acid urine. The acetylated form of sulphanilamide does not act bactericidally in urine at a pH of 7·8 in a concentration up to 200 mg. per 100 cc., whilst free sulphanilamide at the rate of 57 mg./100 cc. of urine has a good bactericidal action at pH 7·5 (Helmholz and Osterberg, 1937).

In Bacillus Friedländer (Klebsiella pneumoniæ) infections in mice the protective effects of sulphanilamide were found to be very slight and those of sulphapyridine a little better.

Intramuscular injections of neoprontosil failed to protect mice against Clostridium welchii infections.

Sulphanilamide and sulphapyridine are fairly effective in the treatment of mice infected with fatal doses of Listerella monocytogenes, but, if anything, helped to hasten death in mice infected with Erysipelothrix.

Sulphapyrazine and sulphadiazine are equally effective against experimental pneumococcal infections and the former drug was somewhat more effective than sulphapyridine and sulphathiazole.

Sulphonamide compounds have an inhibitory effect upon the development and growth of phage-resistant bacteria.

Steyn (1940) reports a fair degree of success in the treatment of strangles in horses with sulphanilamide, while Sterne (1942) achieved limited success in the treatment of virulent anthrax in sheep and rabbits with sulphapyridine. Sulphanilamide, sulphapyridine and 4:4'-diaminodiphenyl-sulphoglycoside had no influence on the course of anthrax in mice. Sulphaguanidine is stated to have yielded favourable results in the treatment of dysentery carriers. Favourable results are reported in the prevention of gas-gangrene infections in wounds by the local application of sulphonamide compounds and in the treatment of cholera with sulphaguanidine.

III. Size of therapeutic doses and methods of application

(a) *Sulphanilamide*

Oral doses for man: 1 g. every 4 hours for 48 hours and then 0·5 to 0·66 g. every 4 hours for a few days.

Oral doses for small animals: 0·2 g/kg. body weight for the first two days, then 0·13 g/kg. for three days and then 0·06 g/kg. for five to seven days.

It is recommended that dogs be given orally 0·5 g/kg. for the first 24 hours; the dose to be subdivided into five or six parts and to be reduced after the first 24 hours or 48 hours to such an extent as to maintain a blood concentration of 15 mg. sulphanilamide per 100 cc.

Oral doses for horses (450 kg.): 60 g. of the powdered drug per nasal tube daily for five consecutive days; no further administration for nine days and then another 60 g. daily for five consecutive days.

Steck recommends for horses suffering from strangles a dose of 80 g. of sulphanilamide on alternate days until the fever disappears. It is considered that in horses a concentration of 5 to 10 mg. of the sulphonamides per 100 cc. of blood must be maintained until the fever disappears.

(b) *Sulphapyridine* (Dagenan, M. and B. 693)

Oral doses for man: Initial dose of 4 g., then 1 g. every 4 hours until the temperature is normal for 48 hours. From reports of Strickler *et al* (1941) it appears that an intravenous dose of 0·06 g/kg. body weight of sodium sulphapyridine monohydrate is dangerous for man.

Klein *et al* (1941) administered sulphapyridine in daily doses of 0·08, 0·1, 0·12, 0·14, 0·16 and 0·18 g/kg. body weight to six cows orally for six days. Each dose was divided into three fractions and given at 8-hourly intervals. All the cows showed anorexia, constipation

and a decrease in milk yield, but these symptoms disappeared in one or two days after the last dose. No serious toxic effects were noticed.

(c) *Sulphathiazole*

Oral doses for man: Initial dose of 4 g., then 1 g. every four hours day and night until the temperature is normal for 72 hours.

Oral doses for cattle: 0·13 g/kg. body weight daily for two days, then half this dose daily for two to four days depending upon the progress toward recovery.

(d) *Sulphaguanidine*

Oral dose for man: 0·1 g/kg. body weight given every four hours. As much as 12 to 14 g. can be given within a 24-hour period. (Stickney *et al*, 1942).

Oral dose for pigs: 0·1 g/kg. body weight four times daily for five days; in severe cases of enteritis the dose can be increased to 0·2 g/kg. (Cameron, 1942).

Thorp and Shirley (1942) report good results in the treatment of calf scours with sulphaguanidine in the following oral doses:— "Calves 75 lb. and over, 0·15 to 0·17 g. per pound of body weight per day, divided into three equal parts and given morning, noon and night; calves under 75 lb., 0·13 g. per pound of body weight, given in the same manner. The dosage for all calves should be reduced on the third day by 0·05 g. per pound of body weight and not over 0·06 g. per pound of body weight should be given on the fourth day." An overdose of sulphaguanidine causes severe tubular nephritis.

(e) *Sulphadiazine*

Oral doses for man: 2 to 4 g. and even 6 g. as an initial dose, followed by 1 g. every four hours until the patient has been afebrile for three to five days. The usual treatment lasts six days with a total dose of 40 g. or less (Editorial, 1941). Three per cent. solutions of sulphadiazine sprayed at short regular intervals on wounds are said to be effective in the prevention of infections, especially with streptococci and staphylococci.

(f) "*Sulphonamide E.O.S.*" (Prepared by I.C.I.)

Green and Coplans (1942) recommend this preparation in the treatment of common colds and as a general safe prophylactic because it is relatively non-toxic. It appears to be of great value in infants suffering from broncho-pneumonia. The infants, which ranged in age from one to thirteen months, received 4-hourly doses of 0·5 to 1 g. It is far less toxic than sulphanilamide and clinical trials were very successful. White mice tolerated very well as much as 1·25 g/kg. body weight administered *per os* daily for several days. It has a strong bactericidal action and is hydrolysed in the system into sulphanilamide.

(g) Macartney *et al* (1942) found the new sulphonamide, sulphamethazine, of high therapeutic efficiency in lobar pneumonia, meningococcal meningitis and gonorrhœa. It is less likely than other sulphonamides to cause urinary calculi, nausea, vomiting and cyanosis.

(h) *Uleron (Diseptal A)*

Neitz of Onderstepoort achieved good results in the treatment of heartwater (Rickettsia ruminantium) in cattle and sheep. The following doses are recommended: Cattle and sheep: 10 cc. of a 10% aqueous solution of sodium Uleron per 50 kg. body weight intravenously. The dose can be repeated four times at 24-hourly intervals. Neitz has also achieved good results in the treatment of Rickettsia canis infection in dogs with Uleron; the dose is the same as that recommended for cattle and sheep. Care should always be taken to observe whether biliary fever relapses occur. Carmichael of Uganda had good results in the treatment of Rickettsia canis infection with soluseptasine and proseptasine.

Five per cent. sulphanilamide pastes and ointments prepared with equal parts of zinc oxide or iodoform and liquid paraffin; or 12 g. of sulphanilamide powder in 120 cc. of cod liver oil and made up to 8 oz. with lime-water are recommended in the treatment of wounds. The author had most excellent results in the treatment of fresh and infected wounds and cases of blood poisoning by dusting sulphanilamide (prontosil) powder on the wounds or applying pastes and liniments prepared with sulphanilamide powder and cod liver oil. Neither the fibroblastic inhibition nor the toxic action on muscle is sufficient to contraindicate the local application of sulphanilamide, sulphathiazole and sulphapyridine on open wounds. They do not appear to retard the healing of wounds. The drug should be used in the solid form as solutions are rapidly absorbed. The solid sulphonamides are hardly absorbed to any extent from wounds, consequently there is little risk of poisoning. Nevertheless, it is advisable to exercise caution in the application of the solid drug to very large wounds. Furthermore, sulphonamides are of value in the treatment of metritis and vaginitis; 90 to 120 g. of sulphonamide -ρ can be introduced into the uterus at a time. The administration of sulphonamides should be combined with the usual treatment (irrigation, etc.) of metritis and vaginitis.

After operations in cases of perforated appendices, Gardiner (1942) introduces 10 g. of finely powdered sulphapyridine suspended in 120 cc. of saline into the peritoneal cavity before suturing the wound. On the other hand Taylor (1942) warns against the implantation of sulphonamide powders in the powder form at the site of an appendicectomy as adhesions may result, due to irritation and inflammation caused by such solid powders. Taylor furthermore states that sulphanilamide, sulphapyridine, sulphathiazole, sulphadiazine and sulphagu-

anidine are quite irritant to tissues. They should not be placed in wounds which are then sewn up, as they cause inflammation of such wounds. Sulphathiazole and sulphadiazine actually cause the formation of abscesses even if quantities as small as 1 g. are placed in wounds, which are then sutured. Sulphanilamide caused the least damage in such wounds. Taylor explains that his remarks do not concern the application of sulphonamides to open wounds.

Trockmorton introduced powdered sulphanilamide, sulphathiazole, sulphapyridine, sulphadiazine, sulphamethyldiazine and sulphanilylguanidine at the rate of 1 g/kg. body weight into the abdominal cavity of white rats (weighing 100 gm.) after laparotomy. The drugs were evenly distributed in the peritoneal cavity and the abdomen sutured. No toxic symptoms developed and no deaths occurred. Some of these drugs, however, caused quite severe local inflammation and peritoneal injury. Sulphanilamide can be considered harmless to the peritoneum, whilst sulphathiazole causes a marked and immediate foreign body reaction. Sulphapyridine cannot be recommended for intraperitoneal use as it causes inflammation and peritoneal injury. He states: "Regarding sulfadiazine, sulfamethyldiazine and sulfanilylguanidine, their intraperitoneal application seems possible without permanent peritoneal injury, should the occasion warrant."

For further information concerning the uses and dosage of sulphonamides Milks (1940), Cushny (1941) and the booklets issued from time to time by the respective manufacturers should be consulted.

IV. Relative toxicity and dangers in the use of sulphonamides

Sulphonamide preparations used in the treatment of various diseases have been responsible for many cases of acute yellow atrophy of the liver, hydronephrosis, urinary calculi, gastritis, anuria, polyneuritis, peripheral neuritis, hæmaturia, intestinal hæmorrhage, cyanosis, methæmoglobinæmia, sulphæmoglobinæmia, acute hæmolytic anæmia, urticaria agranulocytosis, conjunctivitis, acidosis, various forms of rashes resembling those in measles, scarlet fever, urticaria, erythema (macular, papular, and nodular) and porphyrinuria, which resulted in severe photosensitization of those parts of the body exposed to direct sunlight (Rimington and Hemmings, 1939; Marks, 1940). Rimington and Hemmings found no quantitative relation between therapeutic efficiency and prophyrinuric action and that the latter runs roughly parallel with general toxicity. Blum (1941) states that "sulphanilamide in high concentration sensitizes the human skin to ultra-violet radiation of λ shorter than 320 mμ producing a response similar to severe sunburn." He continues: "Patients should not be exposed to direct sunlight or to mercury or carbon arcs during treatment with sulphonamides, since some individuals may develop untoward reactions or become sensitized in the course of treatment."

In experiments upon rats, which were injected intraperitoneally, Antopol *et al* (1941) found that death occurred within three hours with 1·5 g. of sodium sulphapyridine per kg. of body weight, within 11 hours with 1·22 g. of sodium sulphathiazole per kg. of body weight, and within 20 hours with 0·77 g. of sodium sulphamethylthiazole per kg. of body weight. Even in deaths as early as two to three hours after injecting a single dose of sodium sulphathiazole there were large amounts of precipitate of the free compound in the renal papillæ, ureters, and bladder in every case. The greatest caution is necessary in the use of sulphamethylthiazole since it is apt to cause severe lesions in both the liver and kidneys. Sulphathiazole appears to be more apt to produce renal damage than sulphapyridine. All three of these preparations affected the adrenals, causing pronounced vacuolation and minute foci of necrosis in the medulla.

Richardson (1941a), who used white mice in his experiments as they are most sensitive to blood changes with sulphanilamides, states: "(2) On the basis of drug intake, sulfanilamide was the most injurious to blood, being 2·1 times as injurious as sulfapyridine, 4·3 times as injurious as sulfathiazole and 10·9 times as injurious as sulfanilylguanidine. (3) When corrections were made for differences in absorption, excretion, molecular weight, and partition between erythrocytes and plasma, all four compounds were of the same order of injuriousness. (4) Cyanosis, characterized by sulfhæmoglobinemia, was observed only with higher doses of sulfanilamide and sulfapyridine."

Peripheral neuritis has been caused by disulphanilamide (sulphanilyl sulphanilamide) and di-methyl-di-sulphanilamide.

The hydrochloride of sulphanilamide is stated to be four times as toxic as the base and diethylene glycol and sulphanilamide appear to have synergistic effects. Neoprontosil administered orally is less toxic than sulphanilamide.

Hydroxylamine (*p*-hydroxylaminobenzenesulphonamide) is not very toxic, but produced methæmoglobinæmia when injected intravenously into dogs at the rate of 20 mg/kg. of body weight, while rabbits tolerate 30 to 50 mg/kg. of body weight, (Bratton *et al*, 1939).

Carbonic anhydrase, which catalyses the reversible reaction $H_2CO_3 \rightleftharpoons CO_2 + H_2O$, is specifically inactivated by the unsubstituted sulphonamide group of sulphanilamide and this is probably the cause of acidosis produced by it. Sulphapyridine and sulphathiazole do not cause inactivation of this enzyme *in vitro* (Mann and Keilen, 1940; Locke *et al*, 1941).

The L.D. 50's by oral administration to mice, i.e. killing 50%, of the isomers of sulphanilamide are: ortho 3·84, meta 12·45, and para 4·61 gm. per kg. of body weight. There is no correlation between their solubility and toxicity (Laug and Morris, 1939). Sulphanilamide

causes in mice an increased susceptibility to pentobarbital, diethyl ether, divinyl ether and chloroform. Sulphapyridine potentiates the anæsthetic action of papaverine in rats and rabbits; this is not due to an increased concentration of the drug in the blood.

Haerem (1940) reports two cases of spindle cell sarcoma at the site of injection, in 2 out of 20 white mice injected subcutaneously with sulphanilamide. On the other hand Zomecnik and Koletsky (1939) and Lewis failed to find any carcinogenic potency in sulphanilamide and "prontosil soluble" in mice.

The use of sulphanilamide in rheumatic fever is warned against, as its toxic action far outweighs its beneficial effect.

In patients treated with sulphonamides the use of drugs (purgatives), which render the contents of the colon liquid, facilitate the development of sulphæmoglobinæmia. Liquid paraffin is the only safe laxative and should be combined with enemata in severe constipation. Liquid colon contents facilitate bacterial growth and purgatives cause less food to be absorbed. Hence more material (proteins), which is capable of being decomposed into sulphuretted hydrogen, finds its way into the colon. It is probable that all drugs containing $C_6H_5N<$ are capable of facilitating the formation of methæmoglobinæmia and sulphæmoglobinæmia. Such drugs probably act as catalysts in combining sulphuretted hydrogen with the hæmoglobin of the red cells. Chickens receiving sulphanilamide in a high sulphur diet developed cyanosis associated only with methæmoglobin (Richardson, 1941).

A high protein diet protects against sulphanilamide poisoning, whilst rats on high fat and high carbohydrate diets are more susceptible to this drug. The protective effects of high protein diets is most probably due to increased execretion of the drug as the urinary output is very much increased on such diets.

Various authors advise care in the use of sulphonamides during pregnancy, as these drugs pass through the placenta into the foetus. Speert (1941) in experiments upon rats proved the harmful effects of sulphanilamide on the foetus. Sulphonamides are also execreted in the milk, but not to such an extent as to be harmful to the young.

When injected subcutaneously the following are the lethal doses for mice, expressed in grams per kg. of body weight, of various sulphonamides: sodium sulphacetamide 9·7 to 10, sodium sulphapyridine 1, sodium sulphathiabole 1·95, sodium sulphamethylthiazole 0·86, sodium sulphaphenylthiazole 0·9, sodium sulphanilamide 2·92, and sodium sulphadiazine 1·5 to 1·75 (Donovick and Henderson, 1941).

Injected intraperitoneally into rats, "the toxicity of sodium sulfamethylthiazole is almost twice as high as that of sodium sulfathiazole, which in turn is slightly greater than the toxicity of sodium sulfapyridine. The toxicity is influenced by the strong alkalinity of the sodium salts of sulfapyridine and the thiazole derivatives, which

differs despite the presence of the same amount of sodium (Na sulfapyridine 2% – pH 10·7, Na salts of the thiazole derivatives in same concentrations – pH 9·4)," (Lehr *et al*, 1941). It is obvious that in large doses these drugs will disturb the acid-base balance of the body, especially Na sulphapyridine, and produce a serious degree of alkalosis.

Sulphathiazole, administered *per os*, is less poisonous than sulphapyridine for monkeys. Autopsy revealed hæmaturia, urinary calculi, degenerative changes in the tubular epithelium, pyelitis and cystitis.

Sulphaguanidine is less harmful than other sulphonamides. There is a report that it causes goitre in rats, (Editorial, 1942).

Sulphadiazine is a promising new sulphonamide, which is much better tolerated than sulphathiazole or sulphapyridine by man. Sulphadiazine is reported to be also less harmful than sulphathiazole. Renal complications have been noted more frequently in the use of sulphapyridine than in sulphathiazole. However this may be due to the fact that the latter drug is a more recent introduction into therapeutics.

Krems *et al* (1941) report the development of tolerance to sulphanilamide in rats injected intraperitoneally twice daily with this drug. Other investigators again found increased susceptibility to this drug when it is administered orally. Krems and his collaborators report that female rats are more susceptible than males to sulphanilamide.

In man Uleron (Diseptal A) has been responsible for fatal " softening " of the spinal cord, paralysis of peripheral nerves, collapse, polyneuritis, nephritis, and various types of skin eruptions.

Animals appear to be fairly resistant to the toxic effects of sulphonamides. The following symptoms of poisoning have been described in animals:— drowsiness, anorexia, a state of, nephritis, incoördination of movement, paresis, paralysis, diarrhœa and hæmolytic anæmia.

(To be Continued.)

BOOK REVIEW

Spontaneous and Experimental Leukæmia in Animals
By Julius Engelbreth-Holm

This is a comprehensive review of the literature on and our present knowledge of the leukæmic diseases of animals, a field in which the researches of the author and his Danish school of co-workers are known the world over. As such, it calls for notice rather than for detailed criticism.

By far the greater part of the book deals with the experimental aspects of fowl and mouse leukæmias and relatively little space is devoted to the spontaneous leukotic diseases of domesticated mammals. While this of course merely reflects the trend of research, the opinion may be expressed that a large field of pathological research into spontaneous leukæmic diseases, especially of the dog, still awaits serious attack.

Dr. Engelbreth-Holm writes with great authority on the subjects of transmission and heredity in leukæmias — aspects on which a very great deal of experimental data has accrued both in the United States of America and in Denmark. As is desirable in reviews, the author never unduly intrudes his personal opinions at the expense of giving a balanced assessment of the literature. One has little doubt, however, that he is a member of that growing group of authorities who regard the leucotic diseases as essentially neoplastic and that the discussion of them cannot be separated from the general problem of tumours. In common with most other workers, the author concludes that lymphoid leukosis of fowls is not transmissible and unrelated ætiologically to the transmissible myelogenous leukosis and erythroleukosis. It is accepted that certain strains of virus may not only produce either of the two latter conditions, but also sarcoma and endothelioma. The status of neurolymphomatosis, which some authorities are strongly inclined to regard as a neoplastic disease pathologically closely related to lymphoid leukosis but in which the nervous system is exclusively or chiefly involved, is left open.

The weakest side of this excellent book is the detailed histopathology of the diseases dealt with. Again this largely reflects the extent to whcih this aspect has been neglected by investigators. One feels there is great need of accurate definition and characterisation of the various cell types involved in these proliferations, so that uniformity may be achieved in diagnosis and nomenclature among different workers.

Mention should be made of the quite extraordinary ability displayed by the translator (C. L. Heel) from the Danish. From the beginning to end of the book there is not the slightest clue to the fact that the author did not write his manuscript in English. This facility of style is an unusual and refreshing feature in the translation of Continental scientific works.

This book can be unreservedly recommended to veterinary research workers. The practitioner, however, will find little of use to him, and indeed could not expect to do so considering the present status of our knowledge.

Spontaneous and Experimental Leukaemia in Animals, Julius Engelbreth-Holm, PpXVIII - 245. Edinburgh and London. Oliver & Boyd. 1924. Price not stated.

MOVEMENTS OF OFFICERS

Transfers of government veterinary officers during the year September, 1941, to September, 1942:—

J. P. Moll appointed to Umtata 26.1.42.
J. D. Smit appointed to Umtata 21.2.42.
S. L. Snyders appointed to Kokstad 29.1.42.
F. E. Cavanagh appointed to Dundee 2.2.42.
N. C. F. Steenkamp transferred from Kokstad to Grahamstown 18.2.42.
S. G. Turner transferred from Dundee to Port Shepstone 2.3.42.
P. H. Brown transferred from Port Shepstone to Umtata 5.3.42.
J. A. Thorburn transferred from Grahamstown to East London 2.3.42.
G. L. Muller transferred from Umtata to Malmesbury 2.5.42.
J. F. Fick transferred from East London to Umtata 20.4.42.
W. B. Allchurch transferred from East London to Port Elizabeth 3.3.42.
P. J. Goosen appointed to Nongoma 22.5.42.
C. F. B. Hofmeyr resigned 30.6.42 to take up private practice in Pretoria.
J. M. Fourie transferred from Vryburg to Armoedsvlakte.
T. F. Adelaar transferred from Vryburg to Pretoria.
P. H. Brown transferred from Umtata to Bedford 1.7.42—19.12.42 to replace W. Jones on leave.
P. P. Hugo transferred from Eshowe to Rustenburg 17.7.42.
V. R. Kaschula appointed to Dundee 9.9.42.

VETERINARY FACULTY.

The following students at Onderstepoort obtained the B.V.Sc. degree at the examinations held in November, 1942:—

C. H. Basson, F. B. W. du Casse, R. J. Heydenrych, G. K. Mager, T. N. Osborn, R. A. Painter, D. E. Truter and P. L. Uys.

OBITUARY

GERHARD GOTTFRIED KIND

Dr. G. G. Kind was born at Schwanden, Glarus, Switzerland, on July 19th, 1893, and died at his residence in Craighall, Johannesburg, on July 18th, 1942, in his forty-ninth year. He endured the excruciating pain of his long illness with grim pluck and determination which were the keynotes of his character and which were manifested in his work.

Dr. Kind passed his state examination in Zürich and came to South Africa in 1919. He worked at Onderstepoort until 1922 when he resigned to take up private practice. During his period at Onderstepoort he was engaged on bacteriological work and was instrumental in introducing the spore vaccine for anthrax into practice. For this alone he deserves great credit and was granted his doctorate in Zürich for the work. He commenced to practice in Pretoria but soon moved to Johannesburg. He

began his work on the Rand under great difficulties with his limited knowledge of English and Afrikaans and even more limited financial means. It was indeed an uphill struggle in the beginning, but his fanatical devotion to his work and sound scientific knowledge could not but be rewarded. He built up a remarkable practice especially among dairy farmers, whom he deeply impressed with his skill and capable handling of sick animals. His skilful and scientific treatment of cases generated a widespread respect for veterinary practice on the Rand, and future practitioners will reap the benefit of his labours. Many are the stories of his kindness to clients in poor circumstances and very often they did not even receive an account. Charitable institutions and orphanages obtained his services free of charge. Reserved and modest by nature he had a strong and buoyant personality. Few will forget that big muscular frame and those deep-set eyes, the eyes of an idealist. He was also a keen and interested student of the political and social problems of the day. He remained a student and kept himself informed of all developments and progress in veterinary science.

He leaves a widow and five young children to whom we tender our deepest sympathy in their sad bereavement.

J. G. B.

THE ASSOCIATION

Council Meeting held at Polley's Hotel, Pretoria, on 22nd September, 1942, at 8 p.m.

Present: Mr. S. T. Amos (President), Col. C. J. van Heerden, Dr. P. J. du Toit, Mr. A. C. Kirkpatrick, Dr. P. S. Snyman, Dr. D. G. Steyn, Dr. E. M. Robinson, Dr. A. D. Thomas, Dr. R. Alexander and Mr. S. W. J. van Rensburg (Hon. Sec.-Treas.).

Apologies for Absence: Capt. N. F. Viljoen.

(1) *Minutes* of meeting held on 19th May, 1942, were confirmed.

(2) *Arising from these minutes:*

(a) *Students' Loan Fund*: The Chairman of the Finance Committee (Dr. Alexander) submitted the following report drawn up by that Committee:—

"Council at its previous meeting instructed the Finance Committee to submit a report on the method to be adopted to administer this account with a view to obtaining uniformity and continuity for the future.

It is recommended:—

(1) Under no circumstances will loans be granted to students in their first or second year.
(2) According to the fund available, not exceeding 50% of the amount in credit, priority will be given to students in their final or fourth year of study.
(3) Only in exceptional circumstances will advances be made to students in their third year, each application to be dealt with strictly on its merits.

Points to be taken into consideration in making an advance are:—

(a) The previous academic career of the individual to be obtained by report from the Faculty.

(b) The suitability of the applicant for a career in veterinary medicine to be obtained from the Faculty and from individual members of the profession with whom he/she has come in contact.

The amount of a loan should not exceed £50 p.a., the total loan to be repaid in not more than six years after qualifying. Council should take such steps as it deems necessary in every case to ensure repayment of the loan. It is suggested that cession of an endowment insurance policy for the amount involved be made, the payment of the premium to be a liability of the Association until the official notification of qualification of the individual. Loans to be free of interest up to date of qualification, thereafter to bear interest at a rate of interest not less than 6%."

The report was accepted in principle. Considerable discussion ensued on the question as to whether the money for this purpose should be derived from the General Fund or from the Benevolent Fund, also whether Council is legally justified in investing Association funds in other than gilt-edged securities.

It was ultimately resolved that legal opinion be obtained and that, if necessary, certain alterations to the Constitution be submitted to the next General Meeting.

(b) *Witwatersrand Branch*: The desirability of a constitution for this branch was discussed. It was decided that a copy of the constitution of the Natal branch be obtained and submitted to Witwatersrand.

(c) *Hormone Treatment of Race Horses*: The President reported that since the last meeting certain developments had taken place which resulted in a legal action. It was resolved that as this case was now *sub judice* no action be taken until after the verdict of the Court has been given, when a copy of the evidence is to be obtained for consideration by a special meeting of the Council.

(3) *New Members*: It was unanimously agreed to recommend acceptance of the following by the General Meeting: R. K. Loveday, B. M. McIntosh, I. Mowat, W. J. Nixon, D. E. Osbourn, J. S. van Heerden.

(4) *Animal Welfare Society*: The Secretary submitted copies of correspondence which had passed between himself, a member and the Secretary of the Society regarding certain advertisements. The grounds for complaint have now been removed and the action taken by the Secretary was confirmed.

Charges of covering were also considered, but it was decided that available evidence was insufficient to warrant further action.

(5) *Standing Committees*: The following were elected for the ensuing year:—

Editorial: E. M. Robinson, P. J. du Toit, C. Jackson, H. P. Steyn and R. Clark.

Finance: R. Alexander, H. H. Curson, B. S. Parkin and A. D. Thomas.

Library: E. M. Robinson, D. G. Steyn, G. de Kock, A. D. Thomas and C. Jackson.

General Purposes: R. Alexander, C. J. van Heerden, A. C. Kirkpatrick, P. J. J. Fourie and P. S. Snyman.

(6) *General*:

Subscriptions: The question of suspending subscriptions of members who are interned or abroad on military duty was discussed. Dr. Alexander and the Secretary were asked to frame a resolution for the General Meeting.

Library: A letter from the Chairman of the Library Committee was considered and the writing off of certain missing books and journals was authorised.

National Health Services Commission: Dr. Snyman expressed the desirability of Council arranging for evidence to be given before this Commission on the veterinary aspect. It was resolved that a sub-committee consisting of Col. van Heerden, Dr. du Toit, Dr. Fourie and the Secretary be appointed to consider the terms of reference of the Commission and to prepare a memorandum for submission to the Commission.

Alleged Closing of Faculty: In reply to Dr. Snyman, Dr. du Toit stated there was no truth in the rumour that the Faculty was to be closed.

Auditing: Council agreed to Finance Committee's recommendation of an honorarium of £10 10s. for the auditor.

The meeting closed at 10.30 with a vote of thanks to the President.

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

THE ASSOCIATION

37th General Meeting held at Onderstepoort, Wednesday, 23rd September, 1942.

Present: S. T. Amos (President), P. S. Snyman, D. Coles, J. M. Fourie, J. S. Watt, P. J. du Toit, M. W. Henning, J. R. Scheuber, H. Theiler, R. Clark, C. J. van Heerden, N. C. Starke, H. P. Steyn, T. F. Adelaar, P. J. Meara, H. P. de Boom, J. W. A. Brookes, B. M. Horwitz, M. Sterne, H. H. Curson, C. F. B. Hofmeyr, J. H. R. Bisschop, R. Alexander, J. Nicol, G. de Kock, E. M. Robinson, M. M. Neser, D. G. Steyn, A. D. Thomas, J. G. Boswell, O. T. de Villiers, J. Quinlan, Miss R. Datnow, D. Haig, R. du Toit, J. G. Keppel, W. G. van Aswegen, B. S. Parkin, J. H. Mason, D. T. Mitchell, V. Cooper, P. J. J. Fourie, H. O. Mönnig, J. I. Quin, K. Schulz, J. G. v. d. Wath, and S. W. J. van Rensburg (Hon. Sec.).

Apologies for Absence: W. G. Barnard, N. F. Viljoen, and J. G. Williams.

Obituary: The President referred to the loss the Association has sustained through the death of Dr. G. G. Kind and asked the meeting

to stand in silence for a minute as a mark of respect.

(1) *Minutes* of the General Meeting held on 5.11.41 were taken as read and were confirmed.

(2) *Matters* arising from these: Nil.

(3) *Election of New Members*: The following were elected: F. E. Cavanagh, (Miss) R. Datnow, P. J. Goosen, V. R. Kaschula, R. K. Loveday, B. M. McIntosh, J. P. Moll, I. Mowat, W. J. Nixon, D. E. Osbourn, J. D. Smit, S. L. Snyders, J. I. Taylor, J. S. van Heerden and F. J. Veldman.

(4) *Election of Council*: The following were declared elected: J. G. Boswell, P. S. Snyman, D. G. Steyn and A. D. Thomas. The Council for 1942 - 43 therefore consists of:—

President:	S. T. Amos.
Vice-President:	C. J. van Heerden.
Hon. Sec.-Treas.:	S. W. J. van Rensburg.
Members:	R. Alexander, J. G. Boswell, J. Dickson, P. J. du Toit, A. C. Kirkpatrick, P. S. Snyman, D. G. Steyn, A. D. Thomas and E. M. Robinson (Editor of the Journal).

(5) *Presidential Address*: In commencing his address, Mr. Amos said that he had not written out a set speech, but would just make a few extempore remarks. In thanking the members of the conference for their attendance he paid a special tribute to the executive officers of the Association, referring particularly to the Secretary, and, congratulating the new members of Council, Drs. Thomas, Steyn, Snyman and Mr. Boswell on their election. Mr. Amos was in a reminiscent mood and referred to the circumstances which led him to come to South Africa and to his early years in Natal as a government veterinary officer. His experiences of those days had made him love South Africa and decide to make it his permanent home.

He then paid a tribute to Col. van Heerden for having taken over control of the inspection of food materials of animal origin being supplied to the Army in South Africa. This must have an influence in gaining for the veterinarian his proper place in the future in food inspection work and allowing him to play his proper role in municipal administration.

In discussing the growth of private practice associated with the treatment of pet animals, Mr. Amos had a complaint to make. Owing to war conditions it is now almost impossible to obtain vaccine for immunization of dogs against distemper and he considered that the research authorities in South Africa should have considered the matter of its local production in order to assist the private practitioner. He stressed the importance of small animal practice, particularly in view of the numbers of veterinarians who would be qualifying in the future and the necessity for research into the diseases of the dog and cat in particular.

In conclusion Mr. Amos made an appeal for the suppression of personal feelings and for the utmost co-operation for the advancement of the profession, avoiding anything which may be in the slightest degree unprofessional so that the profession may at least have an equal status

with the medical in the eyes of the public. He then thanked the members for their indulgence in listening to his address and declared the conference open.

Dr. P. J. du Toit, in thanking Mr. Amos, said we all enjoyed the address given by Mr. Amos who speaks with great authority and experience. He paid tribute to the President's leadership during difficult times.

Dr. du Toit said the greatest development that has resulted from the war, as far as the veterinary profession is concerned, is the important part now played by the veterinary officers of the S.A. Veterinary Corps in meat and milk inspection. This will establish the veterinarian in his proper position in the inspection and control of food in future.

Dr. du Toit further outlined the many difficulties connected with the production of distemper vaccine and stated that this was the reason why distemper serum and virus have not yet been produced at Onderstepoort.

(6) *The Reports of Standing Committees* were submitted and accepted. The Chairman of the Finance Committee drew attention to one very unsatisfactory item on the balance sheet, namely the large amount outstanding as arrear subscriptions. An appeal was made to all members to assist in effecting a considerable reduction in this amount.

(7) *The Secretary proposed* on behalf of Council that the following resolution passed by the 26th General Meeting on 29.3.34, namely, "That the annual subscription for members be increased to £2 2s. p.a. and that 5s. of this sum be earmarked for benevolent purposes," be amended by substituting "10s." for "5s." This was unanimously agreed to.

Dr. M. W. Henning proposed "that in future no member be required to pay any further subscription after he has been a member of the Association for 24 years." This was seconded by Dr. du Toit and after discussion a suggestion by Dr. de Kock that this proposal be submitted to Council for further consideration was agreed to.

(8) *General*:

(a) *Courtesy Title*: Mr. Boswell proposed "that a referendum be held as soon as possible on the desirability or otherwise of adopting the courtesy title of 'Doctor'." Dr. Steyn seconded.

Dr. Snyman pointed out that if this were done immediately those members not present would not be acquainted with all aspects of this question, and he proposed the appointment by Council of a Committee to investigate fully and to report to Council; further that members who wished to do so be permitted to submit their views. This was seconded by Dr. du Toit and agreed to by the meeting.

(b) *Subscriptions*: Dr. Alexander proposed on behalf of Council "that the annual subscription to the S.A.V.M.A. be suspended for members who (a) have been placed in internment camps, and (b) are outside the Union as members of the military forces, the period of suspension being based on completed cycles of 12 months irrespective of the financial year of the Association." This was unanimously agreed to.

This completed the business portion and the following papers and demonstrations were next considered:—

Some aspects of sex physiology and their relation to fertility of animals: Dr. J. Quinlan.

Semen examination; supported by microphotographs: Mr. N. C. Starke.

Discussion and demonstration on the arsenic-resistant blue tick: Messrs. P. M. Bekker and R. du Toit.

Mules operation on sheep: Dr. J. Quinlan.

After lengthy discussion on all aspects of this operation, the following resolution moved by Dr. Thomas was passed, there being only one dissentient:

"This Association recognises that the so-called Mules operation has undoubted advantages in the prevention of fly strike in sheep and also that it constitutes a drawback in that it may delay or render difficult the selection and breeding of plain breeched sheep which is a definitely preferable means of achieving the same object. This Association's attitude is that the operation may be performed on lambs up to weaning as in them pain and suffering are not so severe. The application of rudimentary surgical measures is also strongly recommended on humane grounds."

The meeting concluded at 4.15 p.m.

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