THE ARMY VETERINARY SERVICE IN SOUTH AFRICA

(1881—1914)

BY

H. H. CURSON

Jl. S.A.V.M.A.
VIII. 1937.

DEDICATED TO THE MEMORY OF ALL VETERINARY SURGEONS, BOTH REGULAR AND VOLUNTEER, WHO HAVE DIED DURING MILITARY SERVICE IN SOUTH AFRICA. *

They are :—

A.V.D.

Wilkinson, H.
Crawford, F. F.
Loughlin, J.
Richardson, A. E. †

C.V.S. ATTACHED A.V.D.

Hirst, W. H.
Ensor, E. T. C.
Kidd, W.
MacGregor, C.
Armstrong, R.
Sykes, G.
Davies, H. E. H.
Parker ,J. M. (Canada).

IMPERIAL YEOMANRY.

Fenner, E. A. L.
Barningham, D. C.

SOUTH AFRICAN VETERINARY CORPS.

Worsley, J. A.

With the exception of Wilkinson, Richardson and Worsley all died during the Second Anglo-Boer War.

God made our bodies of all the dust
That is scattered about the world,
That we might wander in search of home
Wherever the seas are hurled :
But our hearts He hath made of English dust,
And mixed it with none beside,
That we might love with an endless love
The lands where our kings abide.

HAROLD BEGBIE — *Britons beyond the Seas.*

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* In the May, 1932, issue of the *Jl. R.A.V.C.* is a request for information concerning the "place of burial of deceased officers connected with our Service."

† For note and photograph of Richardson's grave see *Vet. Rec.*, 28-11-36.

PREFACE

As it is essential that succeeding generations of veterinarians shall be familiar with the history of their profession, I have continued the task of historian.

Had it not been for the willingness of the Editorial Committee of the South African Veterinary Medical Association to assist me and for the generosity of the colleagues * mentioned below, this record could not have been published. In the circumstances I am particularly grateful for their co-operation.

Donations towards the cost of printing blocks were made by Dr. G. Martinaglia and Messrs. S. T. Amos, F.R.C.V.S., J. Chalmers, M.R.C.V.S., F. J. Dunning, F.R.C.V.S., F. C. Gavin, M.R.C.V.S., J. A. Maybin, M.R.C.V.S., A. McNae, M.R.C.V.S., B. Runciman, M.R.C.V.S., C. M. Sharpe, M.R.C.V.S., and N. F. Viljoen, M.R.C.V.S. The Professional Staff Fund, Onderstepoort, also kindly contributed.

H. H. CURSON,

Department of Native Affairs,
Pretoria.
30th January, 1937.

* Other colleagues have thoughtfully promised to give financial assistance should this become necessary.

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The Biology of Onchocerciasis in Man and Animals.*

By R. J. ORTLEPP, PH.D., D.SC., Helminthologist, Onderstepoort.

The subject of Onchocerciasis is of particular interest for two reasons : firstly, because representatives of the causal parasite affect both man and animals; and, secondly, because of the alarming incidence and spread of this disease among South African cattle, resulting in a considerable number of carcasses being condemned for human consumption and in considerable loss to the farmer.

Onchocerciasis in man has been known for some considerable time, having been discovered in natives of the Gold Coast in the early 'nineties; subsequently it has been found to extend through the whole of Central West Africa, embracing Sierra Leone, Cameroons, Nigeria, Congo Basin, Uganda, etc. In 1915 it was recorded as a common parasite in Guatemala and has since been found to be common in certain parts of Mexico. Leuckart named the African parasite *Filaria volvulus;* and Brumpt, considering the Central American form to be different, gave the name *O. caecutiens* to the American parasite. Sandground has recently been able to make a careful morphological study of numerous representatives of both the African and American parasite, and a comparison of this material led him to conclude that the parasites from the two continents were identical.

In animals the earliest record of this parasite is by Diesing, who in 1841 described as *Onchocerca reticulata* a parasite from the ligaments of the horse in Austria; it has since been recorded from several other parts of Europe and there is also a doubtful record from Egypt. In 1910 Raillet & Henry obtained from the nuchal ligament of a horse in France a filariid parasite to which they gave the name *Onchocerca cervicalis* : this parasite has since been found to be fairly common in various parts of Europe, where its presence has been associated with poll-evil and fistuous withers. These two parasites from the horse were considered to be distinct until Sandground showed that they were co-specific and that Diesing's name was the correct one.

A number of species of this parasite have been described from cattle, but it appears from later work that the number of genuine species is probably not more than four, namely O. *linealis,* originally described by Stiles (1892) from the perisplenic connective tissue of American cattle:

* Paper read before a combined meeting of the Pretoria Branch of the South African Medical Association and the staff of the Onderstepoort Laboratory, on the 8th September, 1936.

viviparous, each female being capable of producing innumerable larvae, The larvae are without a sheath and, contrary to other filariid larvae of man and animals, do not circulate in the blood but in the lymph stream.

Occurrence.

Both in man and animals, the adults are parasites of the connective tissues, where they eventually produce fibrous nodules in which they are intricately threaded. In Africa the human parasite produces nodules mostly on the trunk, the areas frequently affected being those where the peripheral lymphatics converge, *e.g.* the axilla, popliteal space, about the elbow, and intercosal spaces; occasionally nodules may be present on the head. In America, on the other hand, the predilection site is on the head, and only occasionally are nodules found on the trunk. The reason for this variation in distribution is not clear, one theory advanced being that the American natives usually wear hats, the pressure of which round the head may be responsible for hindering the migrations of the young parasites in this region, and the irritation thus produced being responsible for the formation of nodules in this region.

In cattle the nodules occur chiefly in the region of the brisket. This is especially so in Australia and the East Indies, where in most cases, after cutting out the brisket, the carcass is passed as fit for human consumption. In South Africa the affected areas appear to be more extensive : while the brisket is usually involved, other parts of the body may carry many more nodules, *e.g.* the infection may involve the head, neck, shoulder, thorax, flank; and hind limbs, extending as far down as the stifle; in fact, the whole of the subcutaneous tissues may be affected, the number of nodules present in one animal reaching several hundreds. This differs from what has been reported from Australia where an animal infected with more than 50 nodules appears to be a rarity.

In both man and cattle it is quite probable that mature parasites may be present in the host without the formation of nodules; in fact some authorities hold that nodule formation is not normal for the parasite, and that nodules are only produced when for some unknown reason the parasite is held up in its migrations and the consequent irritation of the adjacent tissues stimulates the formation of nodules. In proof it is shown that sections of the skin from humans, showing no clinical signs whatever of Onchocerciasis, have revealed the presence of numerous onchocerca larvae. This, however, does not exclude the possible presence of deep-seated nodules which would be missed on clinical examination. Unfortunately no thorough search has been made for free forms in humans. In cattle, however, free forms have often been found and these have been regarded as identical with those found in the nodules. I have not been able to find such free forms, although I have recently dissected out free

Onchocerca parasites from a carcass which also showed the presence of nodules; a comparison of the free parasites and those dissected out from the nodules showed that they were not the same; the parasites from the nodules were the typical *O. gibsoni,* whereas the free ones proved to be *O. gutturosa,* which is present in North Africa and has never been reported as producing nodules.

In South Africa, as in Australia, the nodules are limited to a subcutaneous position; where such is the case it is a comparatively easy procedure to remove these superficial nodules and pass the carcass as fit for human consumption. Unfortunately, a considerable number of nodules may be deeply embedded in the musculature, and to remove all these would necessitate considerable disfigurement of the carcass; this applies especially to those carcasses intended for export, and for this reason it is the general practice in South Africa not to pass for export any carcass which is infected even to a small visible extent.

Geographical Distribution.

As has been mentioned, the African human parasite is found in the western portions of Central Africa (Liberia, Cameroons, Gold Coast, Nigeria, Congo Basin, Uganda, etc.) and the American parasite in the Central American states of Guatemala and Mexico. It is generally accepted that the parasite was introduced into America from Africa, but when and how is not clear. It has been suggested that it was introduced by African negro slaves, and that the parasite has only been able to establish itself in those areas where conditions were also ideal for the existence of the necessary intermediate host. That this view is probably correct is supported by our knowledge that such typical African parasites as the Guinea worm and the rectal bilharzial parasite are also present in America and have only made their appearance in that continent in recent times. Another view which has been suggested is that the parasite was introduced into America by cattle infected with *O. gibsoni;* it is true that morphologically there is very little or no difference between *O. volvulus* and *O. gibsoni,* yet it must be remembered that in countries like India and Australia, where onchocerciasis among cattle is very prevalent, not a single case of onchocerciasis in humans has even been known.

The distribution of onchocerciasis in cattle is fairly extensive, being very prevalent in certain areas of Australia, East Indies, India, South Africa, and Rhodesia. In the Union of South Africa the condition occurs in Mafeking, Vryburg, Northern Transvaal, and Natal. It is probably also present in Swaziland. All these regions are low-lying bushveld areas traversed by a few streams. In Natal, along the Tugela valley near Weenen, the cattle are very heavily infected : in one herd of over 600 more than 90% of the cattle were found on inspection to have nodules visible on the surface.

Life History.

Although investigations into the life-history of the cattle parasite (*O. gibsoni*) have been carried out for a considerable number of years, especially in Australia, no clue has yet been found as to how the parasite is transmitted. It is quite probable that the mode of infection and spread will eventually be found to follow more or less the same lines as that found in *O. volvulus* of man and *O. reticulata* of horses, whose life histories have recently been elucidated by Blacklock and Stewart respectively. Blacklock, working in Sierra Leone from 1923 to 1926, found numerous larvae of *O. volvulus* in healthy and diseased skin of the natives and he also noted that subcutaneous nodules were very prevalent. In no case did he find larvae in the blood. He therefore concluded that if a bloodsucking vector was concerned, it would have to be one which does not make a clean puncture into the skin, as do mosquitoes, but one which, when feeding, rasps or tears the skin, with the result that the larvae would be dislodged from the skin into the wound and would then be taken up by the vector together with the blood. He then noticed that such a possible vector was very prevalent and that it was biting in great numbers along the small streams : this was the gnat *Simulium damnosum*. Arrangements were made that the native boys should catch such gnats as had fed on them, and on dissection Blacklock found that a number of these gnats contained typical active larvae of onchocerca in their gut. He thus satisfied himself that this insect was capable of taking in the larvae and that the latter remained alive and active in its gut. He carried out similar dissections on tsetse flies, tabanids, and the floor maggot after they had fed, but failed to find any larvae in the gut of these species except in the case of the tsetse flies; where, however, the larvae were motionless and apparently dead within an hour of feeding. Blacklock then continued his observations on Simulium, and after obtaining infected volunteers proceeded to capture gnats which had fed on them and to dissect these gnats at various intervals from 1 to 19 days, which was the longest time he was able to keep the gnats alive in captivity. He found that the percentage of infected gnats rose to over 80% and that the larvae were very active in the gut; after about a day they had pierced the gut and had migrated into the thoracic muscles, where further development proceeded; from the 5th day onwards he found that the larvae had migrated into the head; here further development took place until about the 9th day, when the larvae were about 0.75 mm. long and had apparently reached their mature infective stage. The shortest time between feeding and the appearance of the mature larvae in the proboscis of the gnat was 7 days. Unfortunately, Blacklock was not able to trace the further development of these larvae in a human host, but attempts were made to infect monkeys by placing the mature larvae under the skin. The monkeys, however, failed to show any signs of infection after some months.

That species of the genus Simulium are the true and probably only vectors of *O. volvulus* in man has recently been confirmed by the work of Strong in Central America. He found that three species of this gnat were able to take up the larvae of this parasite and that, in these, the larvae were able to develop until the mature stages appeared in the head and proboscis. He failed to get any developmental stages in any of the other biting insects which he dissected. The gnats were found to breed abundantly in the swiftly flowing mountain streams, the adults readily biting man.

With regard to the life-history of *O. reticulata* of the horse, Stewart (1932), working in England, was able to show that in this case it is not a gnat which acts as carrier, but a midge, viz. *Culicoides nubeculosus*. He first tried five species of gnats; but on dissection of these after they had fed on infected horses, he was not able to find any micro-filaria in their gut. He also obtained negative results from dissecting engorged tabanids, stomoxys, and various mosquitoes. With midges, on the other hand, he was able to find active larvae in the ingested blood for about 24 hours after feeding. From the 3rd to the 4th day the larvae are found in the thoracic muscles and here they continue their development and become larger until about the 22nd day, when they pass into the head and may be found as mature larvae in the proboscis on the 24th or 25th day after feeding. Compared with the development of *O. volvulus* in the gnat (*Simulium damnosum*) that of *O. reticulata* takes a much longer time to reach the infective stage in *Culicoides nubeculosus*, viz. 24 to 25 days as against 7 to 8 days in the case of *O. volvulus*. Otherwise the developmental stages of these two species appear to be identical.

Prophylactic Measures.

In all helminthic diseases the prophylactic measures to be adopted are based on our knowledge of the bionomics of the intermediate host, where such is necessary and known. Should the carrier of onchocerciasis in cattle prove to be a gnat or a midge it does not appear that really effective measures for the control of this disease would be possible. These insects are so minute that screening is practically impossible, besides being economically prohibitive. Attempts may perhaps be made to deal with the vectors in their breeding grounds, but as Simulium only breeds in water running swiftly over submerged rocks, its control also becomes extremely difficult. Fortunately, infected cattle do not appear to suffer any harm from the presence of the parasite and it would thus appear advisable that farmers living in those areas where onchocerciasis occurs should not breed cattle for slaughter purposes but should limit their activities to other types of farming, if possible, or to dairying.

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The Occurrence of Carotinoids in Animal Sera: the Danger of Mistaking Carotinaemia for Bilirubinaemia.

By CLAUDE RIMINGTON, M.A., Ph.D., B.Sc., A.I.C.,
Onderstepoort.

The carotinoids are a group of yellow pigments, formerly called lipochromes (Krukenberg) or luteins (Thudichum), which are universally distributed in the vegetable kingdom and are thus liable to be ingested in considerable quantities whenever green food is supplied to an animal. They also occur in egg-yolk and in milk. Since the colour they impart to the serum is almost indistinguishable from that caused by bilirubin, caution should be exercised in interpreting any yellow coloration of the serum as bilirubinaemia without further tests. The carotinoids, when present in abundance, are also liable to cause a yellowish pigmentation of the skin closely resembling that of clinical icterus.

During the course of some experiments on the pigment metabolism of animals, these facts were brought very forcefully to the notice of the writer, an experience which has prompted him to draw attention once again to the liability of confusion arising owing to an imperfect identification of the pigments present in any yellow-coloured serum.

Without entering at all systematically into the chemistry of the carotinoids, one may state that the two pigments most commonly encountered are carotin and xanthophyll. The former occurs in three isomeric modifications, known as α, β, and γ carotin respectively, and it is these substances which are the precursors of vitamin A. They are ether- and fat-soluble pigments, but are devoid of acid properties—a characteristic which facilitates their separation from bilirubin.

The carotinoids cannot be synthesised in the animal body; they are, however, retained to some extent by the different tissues after absorption. In different organic solvents they exhibit characteristic absorption spectra. They give no colour with the van den Bergh diazo reagent for bilirubin.

From observations on the human subject, it would appear that a high serum carotinoid value is rarely, if ever, associated with any particular pathological condition : the quantity of these pigments seems rather to fluctuate with the diet. This would also appear to hold good in the case of animals. Palmer has shewn that feeding foodstuffs rich in carotinoids increases the carotinoid content of the blood of many species. However, the pig, guinea-pig, and rabbit, and most probably also the dog never

exhibit carotinaemia. Sheep and cat sera contain the pigment to a slight extent, whilst in horses and particularly in cattle the quantity may be so great that the serum has a deep yellow colour (Hijmans van den Bergh, 1928; Lewin, Miethe, and Stenger, 1907; Palmer, 1922; Palmer and Eckles, 1914; Rösiö, 1929, *et al.*). Euler (1928) and his collaborators have found as much as 0.47 mgm. carotin per cent. in bovine blood serum.

Detection and Separation from Bilirubin.

In many instances it is possible to distinguish the absorption bands of the carotin spectrum by examining a carotin-containing serum with the ordinary hand spectroscope. The bands most clearly seen lie at approximately 490 $\mu\mu$ and 460 $\mu\mu$.

In order to extract the pigment, the serum should be treated with twice its volume of 96% alcohol, which throws down the serum proteins; and, since carotin is not soluble in alcohol of such a concentration as is here finally present, it is precipitated with the proteins, while bilirubin remains almost entirely in the supernatant liquid. After centrifugation or filtration, the protein mass is treated two or three times with ether and the combined ethereal extracts allowed to evaporate at room temperature until such a volume is reached that the characteristic absorption bands can be made out in the yellow solution. They lie approximately 475 $\mu\mu$ and 450 $\mu\mu$ in this solvent. Should there be any uncertainty, the ethereal solution may be shaken with a little very dilute sodium hydroxide solution to remove any remaining traces of bilirubin : if a yellow colour persists in the ether, the presence of carotinoids can be assumed. Where facilities permit, the ether solution may be evaporated (without warming) to dryness and the residue dissolved in about 1 ccm. of carbon bisulphide for measurement of the absorption bands in this solvent also (bands at 517 $\mu\mu$ and 482 $\mu\mu$).

The carotinoids give no colour with van den Bergh's diazo reagent; therefore, whenever a bovine or equine serum is found upon examination to be coloured yellow, the van den Bergh test for bilirubin should be carried out before any inference is drawn as to the nature of the pigment present.

In one case examined at this laboratory, a deep yellow bovine serum was found to contain 0.2 mgm. of carotinoids per 100 ccm., but not a trace of bilirubin. Had the van den Bergh test not been performed, an erroneous diagnosis of severe icterus might well have been made.

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OBITUARY.

Edoardo Peroncito.

One of the fathers of modern parasitology, Edoardo Peroncito, died at Pavia, Italy, on the 4th November, 1936, at the ripe age of 89.

It is mainly in the subject of helminthology that he distinguished himself, his greatest work being the elucidation of the cause of the "miner's anaemia" which so severely affected the labourers engaged on the construction of the St. Gottard Tunnel that this project almost came to a standstill. At that time little was known of the smaller worm parasites of man and it was especially from the results of careful autopsies that Peroncito came to the conclusion that the hookworm, *Ankylostoma duodenale*, described and named in 1843 by Dubini, was the cause of the anaemia. Thereupon he set to work to study the life-cycle of the parasite, on which he published a report in 1880. This contained information regarding the passage of eggs in the stools of infested persons, their development and hatching, and the further development and habits of the young larvae. It was not until later (1897) that Looss discovered the method of infection by skin penetration of the larvae; but Peroncito's work was sufficient to allow the institution of preventive measures which overcame a great deal of the difficulty and enabled the work on the tunnel to be completed.

Peroncito also wrote on bacterial and other diseases. He was one of the first to study the relationship between human and bovine tuberculosis. To him was due the discovery of the fowl cholera bacillus, and he confirmed Pasteur's work on swine erysipelas.

Of interest to South African veterinarians is the fact that Dr. Veglia, for many years helminthologist at Onderstepoort, was one of Peroncito's pupils and could relate many anecdotes of this famous Italian scientist.

Peroncito was professor of parasitology at the University of Turin for many years. He received several honorary degrees from universities in other countries, where the significance of his work and his profound understanding of the subject of parasitology were highly appreciated.

H. O. M. & E. M. R.

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The Physiology of the Udder.*

By J. I. QUIN, D.V.Sc., Onderstepoort.

From the purely structural point of view the mammary gland is a true skin gland, although functionally it is so closely associated with the reproductive system that it has to be considered as an accessory gland to that system. In the cow, the udder comprises four distinct quarters, each of which is provided with its own separate lactiferous sinus and teat.

Development.—The mammary gland arises as an invagination from the ectoderm, which at first forms a solid column of cells and which gradually becomes hollowed out to form the duct system. Further development before sexual maturity is limited mainly to an increase in fat and connective tissue. With each oestrum there is, however, some slight transitory budding of the glandular or alveolar tissue. In the bitch, where a distinct state of pseudo-pregnancy occurs, this development may be quite pronounced. However, it is only during true pregnancy that the udder maintains a progressive growth and proliferation of its glandular elements.

Control of Mammary Development and Lactation.—Nervous influences on the udder are of importance only in so far as they aid in the regulation of the blood flow to the mammae (vasomotor nerves). Thus, after parturition there is a mass shifting of the blood from the uterus to the mammary vessels.

Various hormones liberated into the bloodstream are intimately concerned with mammary function. Thus under the influence of *oestrin,* which is elaborated in the maturing ovarian follicle and later also in the placenta, marked growth and branching of the duct system in the udder takes place. The glandular tissue, however, is not stimulated by oestrin to any extent. The effect is well demonstrated by the injection of this hormone into experimental animals. If, together with oestrin, *corpus luteum* extracts, containing the hormone *progesterone,* are administered, pronounced glandular development takes place, to a degree comparable with that occurring in mid-pregnancy. However, no secretion from the cells takes place as yet. For actual secretion of milk to occur, the intervention of the *anterior lobe of the pituitary* is essential. Through the elaboration of a hormone termed *prolactin* this organ induces the glandular tissue, developed under the influence of the above-mentioned two ovarian hor-

* Paper read before the S.A.V.M.A. Congress at Onderstepoort on the 30th October, 1936.

mones, to secrete. This activity becomes evident only during the latter half of pregnancy and is continued subsequently throughout the whole lactation period of the individual.

Mechanical Factors.—Apart from the rôle played by the above-mentioned hormones, various mechanical factors are closely concerned with a continued milk flow. Of these, the effect of milk pressure within the udder appears to be of fundamental importance. The whole structure of the cow's udder is designed for the relief of pressure on the secreting alveoli : the elastic fibrous coat, the elastic fibres around the ducts, the thin and elastic skin, the presence of a large milk cystern for each quarter, the tone of the teat sphincters—all these tend to keep the internal pressure as low as possible.

The Normal " Letting down " of Milk.—From one milking to the next the pressure in the udder gradually rises. While at the onset of milking there follows a sudden and very large increase in pressure, this gradually falls as milking proceeds. At one time the theory was widely held that this sudden rise in pressure was due to a reflex secretion of milk, stimulated by drawing on the teats, half the total amount of milk being thought to be secreted during milking. However, there are serious objections to this conception. Firstly, there are no true secretory nerves supplying the mammae. Further, the quantity of milk recoverable from udders immediately after slaughter suggests that there is actually more milk in the udder than is obtainable at any one milking. Again, if this theory were correct, the udder would have to secrete sixty times faster during milking than during the intervals. According to Hammond, the sudden increase in pressure is due to a process of erection of the udder and teats similar to that occurring in the erectile tissue of the genital organs. This erection is responsible for a virtual " squeezing out " of the milk from the alveoli and ducts, through reflex engorgement of the very large number of blood-vessels and anastomosing veins. It is to be remembered that the veins of the udder, when filled, may contain as much as 50% of the total blood volume. When milk is continuously drawn off through catheters inserted into the teats, only 40% of the total yield is obtained, the remaining 60% being retained in the small ducts and the alveoli, and not being recoverable in the absence of active erection of the udder.

The " letting down " is therefore the active process, and not the " holding up", as is frequently assumed. Influences from the brain, however, can exert an inhibitory effect on the " letting down " reflex, *e.g.* in the case of a cow being frightened by a dog. With incomplete erection, the fat-content, as well as the milk yield, will be below normal, since the small fat globules, of lower specific gravity than the non-fatty solids and the water, are incompletely extruded from the small ducts, in which they are held up—*cf.* the richness of the " strippings " in fat content. The erection appears to be transitory, inasmuch as the yield from the quarter

first milked is usually higher than that from those milked subsequently, by which time the degree of erection may be subsiding : hence the importance of rapid milking.

The Effects of Milk Pressure on Milk Production.—The easiest way to dry off a cow is to stop milking, this effect being due, not to the accumulation and chemical influence of the secretion products, but to the pressure exerted by the milk in the udder. The latter, again, depends to some extent on the conformation of the udder : compare, for example, the small, fleshy, fibrous udder of low elasticity, in which a high pressure is soon reached, with the elastic pendulous udder of the heavy milker. It would even appear that one of the most important limiting factors to a still higher milk yield and butter fat content in many cows is that of milk pressure within the udder; hence the value of milking at short intervals : a 10% increase may be noted with the 8-hour as compared with the 12-hour milking interval. High milk pressure, through causing lymphatic obstruction in the udder, is responsible for the oedema at the time of calving. Changes in the size and shape of the alveolar cells, depending on the pressure to which they are subjected, may be demonstrated histologically.

The Effect of Milk Pressure on Milk Composition.—Milk drawn at short intervals usually contains a higher percentage of butter fat than that drawn at longer intervals. According to Hammond, it is again the milk pressure that is responsible for this difference, (1) through inhibition of secretion generally, and (2) through impeding the passage of the fat globules down the capillary ducts into the cystern. Thus milk drawn at short intervals may contain 0.5% more fat than that taken at longer periods. Furthermore, the erection of the udder appears to be important in dislodging the fat globules from the finer ducts, seeing that milk drawn by catheter has a uniformly lower (*e.g.* from 2.4% to 4.3%) butter fat content than has milk obtained by hand after withdrawing the catheter. Similarly, the fat content of milk drawn from amputated udders has been found to be only about half that of the milk obtained from previous milking, although the residual milk in the amputated udder actually contains a much greater quantity of fat. Suckling, through provoking a strong and rapid erection of the udder, results in the flow of a more fatty milk. Thus, according to the experiments of Crowther, rapid milking (like suckling) produces not only a higher milk yield but also as much as a 40% increase in fat yield, as compared with slow milking. By milking all four quarters simultaneously, Crowther found a 6% increase in fat as compared with the ordinary method. In the latter case, the quarter milked first yielded a fat content of 4.26% while for the quarter milked last the figure was 3.1%. Therefore any factor which tends to inhibit the erection mechanism of the udder may lead to increased variability in the quantity and quality of the milk.

The Formation of the Different Constituents of Milk.—Although our knowledge concerning the actual mechanism of mammary secretion is still very scanty, recent investigations conducted by Graham, Kay, and others have thrown considerable light on the nature and origin of the milk precursors derived from the blood supplied to the mammary gland. Until now, the main obstacle to investigation has been the difficulty of obtaining comparable samples of arterial and venous blood entering and leaving the gland. By the ingenious method of puncturing the external iliac artery per rectum, these workers succeeded in obtaining adequate amounts of arterial blood at the same time that venous blood was being withdrawn from the large mammary veins of lactating cows. Although, as was to be expected, comparative analyses of such blood samples shewed only small differences, the results are nevertheless significant in revealing the chief precursors essential to the formation of milk. Moreover, this work has clearly demonstrated the extreme sensitivity of the cow to such disturbances as may provoke significant changes in the blood composition, leading to even a rapid checking of the secretory activity of the udder, probably through reflex vaso-constriction : under such conditions, the usual differences between the arterial and the venous blood tend to disappear more or less completely. For this reason, the rapid and simultaneous collection of the different blood samples while the animals are at ease is important (less than 4 minutes for the whole operation). From the results so far obtained, the following conclusions may be drawn :—

(1) There is a rapid and voluminous blood circulation through the udder, calculated to be in the vicinity of 9,000 litres per day (6 - 7 litres per minute), or even more in heavy milkers.

(2) The formation of 1 litre of milk requires the passage through the udder of as much as 500 litres of blood : hence the close correlation existing between the diameter of the mammary veins and the milk yield.

(3) The fat of cow's milk is derived mainly from the neutral fat of the blood and not, as was previously held by many workers, from the phospholipin.

(4) The phosphorus compounds of milk originate mainly from the inorganic phosphate of the blood plasma, the organic phosphoric esters and phospholipins constituting no appreciable source of phosphorus.

(5) The blood sugar (glucose) is the direct source of the milk sugar (lactose), the amount of glucose extracted by the udder depending largely on the sugar level of the arterial blood and probably also on the volume of milk secreted.

(6) Thyroxin in daily doses of 0.02 mgm. per Kilo has a marked effect in increasing milk secretion, milk fat, and non-fatty solids.

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The Incidence and Diagnosis of Streptococcic Mastitis. *

By S. W. J. VAN RENSBURG, M.R.C.V.S., Onderstepoort.

Of all the diseases of bovines there is certainly none which plays greater havoc with the economic side of dairying than does mastitis; but owing to the insidious nature of this disease and the inability of the layman to diagnose any but the most advanced cases, the dairy farmer and the consumer of dairy products both remain in blissful ignorance of the true extent of its ravages.

The detrimental effects of mastitis may be considered under the following heads :—

1. *Direct Loss of Milk.*—It is estimated that the milk yield of a cow affected with mastitis in one or more quarters is on an average reduced by 21 per cent. That would give an average loss of approximately 100 gallons per infected cow per annum.

The disease has up to now received most attention in Germany and in America. In Germany it is estimated that anything up to 50 per cent. of the cows are infected, and the annual loss resulting from this is regarded as being between £9,000,000 and £14,500,000.

In New York State alone we find that out of 1,330,000 cows over 57,000 are annually eliminated from production of milk as a result of mastitis. The annual loss of milk in this one State alone is considered to be about £10,000,000 and the total loss of cows and milk £14,000,000.

In Canada the number of individual cows found to be infected in the separate herds varies from 19% to 97%. The average number infected is estimated in the neighbourhood of 40 per cent. This is approximately the same percentage as in England, where the disease has also been the subject of special attention within recent years.

No extensive mastitis survey has as yet been carried out in South Africa, but such observations as have been made tend to show that the position here is no better than in any of the countries previously mentioned. We have a cow population of approximately 2,000,000. Only half of these can be regarded as dairy cows. Assuming that the incidence of mastitis among the latter is as low as 20%, there would be 200,000 infected cows,

* Paper read before the S.A.V.M.A. Congress at Onderstepoort on the 30th October, 1936.

representing an annual loss of 20,000,000 gallons of milk. At 1/- per gallon, this means a direct loss of £1,000,000 per year.

Streptococcic mastitis is one of the diseases of domestication, and many authorities believe that it does not arise spontaneously, but is usually introduced by an infected cow. It is therefore not so prevalent among self-contained herds in rural areas as in the dairy herds of towns, where the vicious circle of continually selling off infected cows—often to other unsuspecting dairymen—and replacing them with new introductions is in operation. It is certain that in many of the dairy herds in the larger towns of South Africa the incidence of mastitis is well above 50%.

2. *Reduction in the Lactation Life of Cows.*—In infected herds there is a noteworthy decrease in the lactation life of infected cows. Animals that normally would have lactation lives of 8 or 9 years frequently have to be prematurely retired and disposed of to a butcher after the third or fourth calving on account of mastitis. It is remarkable to note in an infected herd how many heifers actually have the disease after their first or second calving. By the time they reach their third or fourth lactation period, when normal cows generally attain the height of their milk production, such animals are suffering from such extensive fibrosis that they have to be discarded as uneconomical.

3. *Physical and Chemical Changes in Milk.*—No other disease produces such marked physical and chemical changes in milk as does mastitis. Milk from infected quarters is (except in mild cases) thin, watery, and flocculent. It shows a decrease in lactose, casein, potassium, calcium, phosphorus, and magnesium; and an increase in water, chlorine, sulphur, catalase, leucocytes, and bacteria.

As a result of the increase in bacterial and cellular contents the holding quality of mastitis milk is reduced to a marked degree, as may be demonstrated by the methylene blue reductase test.

Such milk is also much less valuable for cheese-making. The decrease in casein retards or may even completely prevent the formation of curd when rennet is added. When mastitis milk is used for cheese-making, the curd is usually soft and correspondingly more difficult to handle. The texture of the cheese is abnormal. There is a tendency for it to be sandy, to retain moisture abnormally and to become mouldy and discoloured. There is also a greater loss of butter fat during the manufacturing process, resulting in less economical cheese-production.

4. *Unsuitability of Mastitis Milk for Human Consumption.*—The public health aspect of mastitis would itself provide sufficient material for a lengthy paper. It can receive but scant consideration here.

While according to present knowledge some strains of mastitis streptococci are non-pathogenic for human beings, there is on the other hand conclusive evidence that many are capable of setting up disease in man

and are especially responsible for streptococcic infections of the upper air passages and of the gastro-intestinal tract.

The consumption of mastitis milk is to be condemned not only because of the possibility of transmitting disease, but also on account of the pronounced physical and chemical changes, and the quantities of pus in the milk. Meat contaminated with pus is rightly condemned as unfit for human consumption. Why should not milk be similarly treated? There is no difference between the two except that in the latter case the pus is obscured by the similar colour of the milk. Assuming that the incidence of mastitis in urban areas is as low as 35% and that the average amount of sediment given by such milk is only 0.03 ccm. per 10 ccm. milk, then in a town like Pretoria or Port Elizabeth, where the daily consumption of milk is about 7,000 gallons, the inhabitants consume at least 5 or 6 gallons of pus every day.

Unfortunately the control and examination of milk in South Africa are still in a primitive stage, and are confined to matters which are, after all, of only minor importance. Great attention is devoted by local authorities to comparatively insignificant details, like the overalls worn by milkers or the shape of the buckets, but the most important factor in the production of clean healthy milk, namely the health of the cow producing the milk, is totally disregarded. So dairymen are frequently prosecuted and heavily fined for adding a little harmless water to the milk, while on the other hand there is no attempt to limit the amount of pus in the milk.

Diagnosis.

There is probably no other disease for which so many diagnostic tests have been devised as for mastitis. Yet there is no single test which can at this stage be recommended as being 100 per cent. successful. All have limitations, and unfortunately with few exceptions the majority of them do not reveal more than 50 or 60% of cases.

Whatever line of treatment is adopted, success in ridding a herd of infection or preventing its introduction into a clean herd can only be attained if we have a method of diagnosis which will detect every case.

The various tests are based upon the following factors :—

A. Cultural characteristics of the causal organisms.
B. Pathological changes in the udder tissue.
C. Physical and chemical changes in the milk.
D. Microscopic appearance of the milk.

A. *Cultural Characteristics of Organisms.*

Diagnosis based on these, of course, involves bacteriological examination for the purpose of determining the presence or absence of the causal organisms in cultures grown from milk samples from the suspected cows.

It is certainly one of the most reliable methods, but has the drawback that its application is limited to the laboratory. *

B. *Pathological Changes in the Udder Tissue.*

Clinical examination for the detection of pathological changes in the udder is still one of the most reliable methods of establishing the presence or absence of mastitis, although it cannot give any indication of the nature of the mastitis and of the organisms responsible.

The greatest objection that can be raised against physical examination of the udder as a means of diagnosing mastitis is that its success depends to a very large extent on the human factor. Great patience and extensive experience are necessary for a proper clinical examination, which also involves the clinician in considerable physical discomfort.

The examiner must first of all be thoroughly acquainted with the appearance and consistency of the normal udder before he can appreciate the slight variations which are sometimes encountered in mastitis. Proficiency can only be acquired by careful examination of a large number of normal udders.

The examination of the udder must be very thorough, and should not be confined to running the hand casually over the udder while standing beside the cow.

The first essential is that the cow should be milked out thoroughly before manual palpation is attempted. No veterinarian, however skilled or experienced, can detect fibrosis (unless very marked) in a fully distended udder. The cow should be properly restrained in the same manner as she is accustomed to for milking, so that the examiner need not labour under the constant fear of being kicked.

Information which may have an important bearing on the interpretation of the findings obtained by clinical examination, and which must therefore be obtained from the owner, relates to breed, age, date of last calving, and history—the latter especially with regard to a previous attack of acute mastitis, since such an attack may be responsible for atrophy of the quarter in question.

One then proceeds to inspect the udder by viewing it from behind and from the sides in order to note its symmetry.

Next one literally has to " get down to it," sitting under the cow, as if for milking. Lift up each quarter separately with both hands, noting the size, weight, and consistence, and compare the four quarters with one another as regards these factors. At the same time examine the teats for scars and the presence of fibrotic tissue inside the teat.

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* Prof. Henning having dealt with the bacteriological aspects of mastitis at the Conference, no details were entered into in this paper.

Superficial palpation is then applied by running the palm of the hand over all four quarters and noting whether they are smooth or lobulated. Lobulation should not invariably be regarded as an indication of mastitis : it may be present in many normal quarters, but in such cases there is uniformity of lobulation and the projections are small and regular.

Finally, deep palpation is carried out by pressing the fingers firmly into the glandular tissue to determine the degree of elasticity or induration and the nature of the lobulations.

C. *Physical and Chemical Changes in the Milk.*

Advantage has been taken of these changes to devise a large number of tests for mastitis. Only those most commonly used can be discussed here. These tests are all carried out on milk from individual quarters.

1. *Strip Cup and Black Cloth Test.*—The strip cup consists of a container over which a 100 to 120-mesh wire screen has been placed. Three or four streams of the fore-milk are milked through the screen. Instead of a wire screen a black cloth can be used.

The appearance of flakes or small clotted masses upon the screen or cloth is a definite indication of mastitis. On the other hand, the absence of clots or flakes does not justify a negative diagnosis, and the greatest objection to this test is the fact that it only shows up those cases in which the changes in the milk are well marked.

2. *Sediment Test.*—This test is dependent on the fact that mastitis milk contains a variable quantity of pus, which consists mainly of degenerated neutrophiles, erythrocytes, epithelial cells, and bacteria.

Ten ccm. of milk from individual quarters is centrifuged in a Tromsdorff tube. This is a special centrifuge tube the lower part of which forms a capillary end and is graduated to indicate 0.01 ccm. and over. In milk from normal quarters there is no deposit or only traces. In mastitis milk the deposit varies from a trace up to 4 or 5 ccm. per 10 ccm. milk (*i.e.* up to 40 or 50%). A sediment of 0.01 ccm (*i.e.* 1 : 1000) is usually taken as positive for mastitis.

This test is not very reliable and cannot be depended on to show up more than about 60% of positive cases.

Centrifuging does not precipitate all the cells in milk and a fair proportion of cells rises with the cream. Also a large number of cases of mastitis show a deposit of well under 0.01 ccm. Like the strip cup, the sediment test only detects well advanced cases.

3. *The Rennet Test.*—This test is based upon the principle that the enzyme rennin causes the casein of milk to coagulate. There is a decrease of casein in mastitis milk, which therefore does not coagulate so quickly, if at all, on the addition of rennin.

The milk should be quite fresh for the test. The testing solution consists of one part fresh commercially prepared fluid rennet extract in 50 parts distilled water. A *fresh* solution must be made each time. To 5 ccm. of milk is added 0.1 ccm. rennet solution in a test tube. The combination is mixed by inverting the test tube and allowing to stand 1 hour at room temperature (72°—82° F.). Normal milk will coagulate within this period. Mastitis milk will not.

The rennet test is as reliable as most others, but cannot be depended on alone. Some cows which are quite normal are inclined to produce soft curd milk and such cows would therefore be wrongly classed as infected if this test only were applied.

4. *Alkalinity tests.*—The fact that mastitis increases the alkalinity of milk has led to the use of various indicators, such as brom-cresol-purple (dibromo-thymol-sulphonphtalein), and the Universal Indicator prepared by British Drug Houses, to determine the degree of alkalinity of the milk.

Two or three drops of brom-cresol-purple are added to 3 ccm. milk. Normal milk (pH 6.5) shows a pale dove-grey The colour becomes more and more purple as alkalinity increases.

When brom-thymol-blue is used, 1 drop of a 0.5% solution in 10% alcohol is added to 1 ccm. milk. With this, normal milk gives a greenish yellow, while mastitis milk is shown as a bluish green at pH 7.0.

With the Universal Indicator, normal milk is yellow (pH 6.5). Mastitis milk is indicated as a greenish yellow (pH 7.0 to 7.5), passing through to green (pH 8.0) in bad cases.

This is a very simple test, but unfortunately is not sufficiently sensitive. Of the three indicators mentioned the Universal appears to be the best for this purpose and it reveals 50 to 60% of cases.

5. *Catalase Test.*—Catalase is an enzyme which is believed to be derived from the leucocytes, which are greatly increased in the milk in cases of mastitis. The test is based on the fact that catalase breaks down hydrogen peroxide into oxygen and water.

It is carried out by adding 5 ccm. of a 1% solution of hydrogen peroxide to 15 ccm. of milk in a fermentation tube, and incubating the mixture for 3 hours at 37° C. The gas liberated collects in the closed arm of the tube. If this is more than 1.5 ccm. it is regarded as abnormal.

A rapid catalase test can be conducted in the stable by placing a large drop of milk on a dark background and adding a drop of 9% hydrogen peroxide. Mastitis is indicated by a large number of bubbles forming in the milk.

This is a fairly reliable test, but here again the line of demarcation between positive and negative is arbitrary, and at its best this test can only

be used in support of some of the others and is not to be relied on by itself.

6. *Lactose Test.*—Normal milk contains 4–5% of lactose. This sugar content is markedly reduced or even completely lost in mastitis. The test is carried out by adding 1 ccm. of a 25% solution of sodium hydroxide to 2 ccm. of milk, and boiling in a flame for about half-a-minute. Normal milk will then give a deep red colour; the less lactose present the less colour is produced.

This is qualitative test and therefore not very accurate or reliable.

7. *Chloride Test.*—In case of mastitis the chlorides in milk are appreciably increased, even to the extent of being sometimes detectable by the salty taste of the milk. The normal chloride content of milk varies from 0.09 to 0.14%, and in the absence of any other factors to explain an increase above 0.14% such increase may be regarded as indicative of mastitis. The following is a simple and reliable test for chlorides :—

To 10 ccm. milk is added 40 ccm. distilled water and 10–12 drops of 10% potassium chromate solution. A N/10 solution of silver nitrate is then allowed to drop in slowly, the mixture being stirred with a glass rod or shaken until the yellow colour takes on the first permanent reddish brown tint. The number of cubic centimetres of silver nitrate used is multiplied by 0.035 to give the percentage of chloride present in the milk.

This is perhaps the most reliable of all the chemical tests and it shows up the slightest changes in the udder to a remarkable degree. In fact the greatest objection to this test is that it is too sensitive and too easily influenced by factors other than mastitis. It cannot be relied on in the case of milk taken within the first ten days after calving, nor in advanced lactation or when the cow is being dried off, since the chlorides are normally high in such cases. Further, injuries or bruises of any quarter will tend to cause a hyperaemia and are also likely to produce a temporary increase of chlorides.

D. *Microscopic Appearance of Milk.*

Microscopic examination of milk from suspected quarters is carried out with two objects in view :—

(1) To determine the leucocyte count.

(2) To detect the organisms responsible for mastitis.

(1) *Leucocyte Count.*—The number of leucocytes present in milk usually shows an appreciable increase in cases of mastitis and attempts have been made to establish the presence or absence of mastitis by means of the leucocyte count. While it is a comparatively easy matter for one experienced in the examination of milk smears to detect any noteworthy increase in the number of cells and so to suspect mastitis, this method cannot, however, be applied as a routine test in practice. There is so much

variability that it is not possible to lay down a definite standard. Some would regard 250,000 leucocytes per ccm. milk as indicative of mastitis, while others regard anything under 3,000,000 as normal.

(2) *Detection of Causal Organisms.*—Miscroscopic examination of milk smears for streptococci can be carried out on smears made from—

(a) fresh milk,

(b) the sediment of centrifuged samples, or

(c) milk samples incubated at 37° C for 12 to 16 hours.

(a) The examination of smears made from fresh milk can be discarded as useless since this will show the organisms in a very small percentage of cases.

(b) Smears prepared from the sediment of centrifuged milk reveal a far bigger percentage of cases, but even this method is not very satisfactory.

(c) If milk collected under sterile conditions in a sterile tube or bottle and incubated for 12 to 16 hours at 37° C and smears are then made, the long chained streptococci of mastitis may be detected with remarkable regularity by microscopic examination. One would not be prepared to state at this stage what percentage of cases will be revealed by this method, but the indications at present are that this may yet prove to be the simplest and most reliable method of diagnosing streptococcic mastitis.

For staining milk smears, Newman's method is recommended. This combines fat extraction, fixation, and staining. The slide is immersed for ½ to 4 minutes in the following solution :—

Methylene blue powder	2.0 gm.
Ethyl alcohol, 95%	60.0 ccm.
Xylene	40.0 ccm.
Glacial acetic acid	6.0 ccm.

There is no danger of overstaining. On removal from the solution, the slide is allowed to drain until the smear is thoroughly dry (about 5 minutes). It is then washed in water and dried.

Staining and examination are greatly facilitated by marking four divisions on the slide and making in each of these a smear from a different quarter of the udder.

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Personal Notes.

Members will be glad to hear that our President, Mr. S. T. Amos, F.R.C.V.S., is well on the way to recovery after his operation. At the time of going to press, we learn that he is about to leave the nursing home in England.

* * *

Dr. C. C. Wessels has returned from the U.S.A. to Bredasdorp.

* * *

Mr. C. H. Flight, B.V.Sc., has been transferred from Cape Town to Oudtshoorn.

* * *

Mr. J. H. L. Cloete, B.V.Sc., has been transferred from Ermelo to Onderstepoort, where he has been appointed Lecturer on Anatomy.

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Abattoir and Cold Storage Supervision.

By W. HAY, M.R.C.V.S., Lobatsi.

The veterinarian, especially if young and recently qualified, is apt to believe that in turning to abattoir and cold storage supervision as a means of livelihood he has chosen a fairly stereotyped and therefore trouble-free occupation. It may happen that such hopes are realised : he may at the beginning of his career have the advantage of being " shaded " by an experienced superior or he may have the good fortune to find a smooth-running routine of inspection already established by a predecessor whose system he can follow until such time as he has developed confidence in his own judgment.

On the other hand, circumstances may place him where he cannot enjoy such preliminary advantages. He may find that his professional training has not been sufficiently comprehensive—as indeed it scarcely could be—to enable him to meet the many problems which are likely to confront him. He will probably find it necessary to enlarge his knowledge of trade customs and of storage methods if he is to perform his duties adequately and defend his actions with confidence. This is especially true of new appointments in areas where supervision has to be spread over a number of killing and storage centres.

The writer, who has had experience at both inland and coastal export abattoirs (at Lobatsi, B.P., and Durban, respectively) and also in municipalities where there are multiple privately-owned abattoirs, feels that the following notes may be helpful especially to members of the profession who contemplate taking up such work.

The practice of meat inspection originated almost exclusively at old-established abattoirs in large cities. Conditions were fairly static, and for long it had been customary to appoint a butcher as superintendent. Veterinary overseership is a relatively recent innovation : the public still expect from the incumbent of such a post—as they did from the old type of superintendent—advice on matters which relate purely to the trade. This aspect of his appointment is often overlooked by the recent graduate.

It is interesting to recall that in one case (Edinburgh) it was the scandal of the introduction into the city of an anthrax carcass for sale that roused the civic authorities to an appreciation of the necessity for veterinary supervision—this within living memory. However, the older tradition still prevails in many quarters; but it may be assumed that this

state of affairs will gradually be remedied. Thus it is probable that there will be an increasing number of vacancies for veterinarians in the sphere of abattoir supervision, especially as a part-time occupation. Many of such municipal and semi-rural appointments, demanding as they will a considerable knowledge of the trade, will provide a very stiff test of the veterinarian's competence and tact.

Municipal Appointments.

In South Africa, most of those municipalities which do not employ a veterinarian have a Medical Officer of Health and a subordinate staff of Sanitary Inspectors, one or other of whom may have qualified in meat inspection. On his first being appointed, the veterinarian may perforce have to work under the control of the medical officer; but provided that his personal conduct and the performance of his duties prove satisfactory, such supervision is bound to diminish. The veterinarian who is placed in such a position must educate his public and especially the Public Health Committee of the Town Council in regard to the services he is rendering. The medical officer will generally be found to be only too willing to forego the responsibilities of meat and milk control if he is satisfied of the veterinarian's competence and tact.

Before such an appointment is taken up, it is well to spend at least a month on the floor of an abattoir as well as some period with a butcher so that trade practices (jointing and preservation, etc., of meat) may be learned. This time will be well spent if deftness is acquired in the use of both flensing and stabbing knives, in the sharpening of these on stone or steel, and the making of the necessary cuts. An old meat inspector I knew used to demonstrate his own deftness, as well as the keenness of the edge he maintained on his knife, by opening in one swift downward stroke all the posterior mediastinal glands. While such proficiency is not essential, the slaughterhouse staff is likely to be unfavourably impressed on first acquaintance with a supervisor who in his use of the knife betrays a recent association with the handling of meat.

The veterinarian will probably find the local by-laws insufficiently complete or precise, or too out-of-date to give him that full control of the trade which is essential from the standpoint of health requirements. Amendments must be made which will bring the following three essentials within his control: (1) inspection of all animals before as well as after slaughtering; (2) stamping of all carcasses; and (2) times of slaughter and removal of meat.

(1) Ante-mortem inspection will allow the detection (among other causes for temporary rejection) of animals which are scouring badly—a most important consideration and one which is frequently overlooked.

(2) Stamping of carcasses should be done with an ink of colour

and sheen not easily imitated (e.g. Giemsa stain) and with a stamp of fairly intricate pattern.

(3) The control of slaughtering and removal times is most essential. In private abattoirs it is a common practice to slaughter and deliver meat in the evening or early morning hours; unless legally protected, the Inspector may have to turn out at any hour of the day or night, including Sundays and public holidays—in fact, at any time that happens to suit the owner's convenience.

It is important to ascertain the bacteriological count of the water supply, especially where the meat is to be retained in cold storage.

Shops are more likely than are abattoirs to contain long-standing dirt, particularly in the cold cabinet or store room at the rear : it is wise to pay an occasional call to see that the meat is being properly handled at this end, even though such inspections are usually left to the Sanitary Inspector. European and Indian butchers of the poorer class are frequently the worst offenders against health requirements.

Municipal and Private Cold Storages.

The veterinarian may consider himself fortunate if he has to deal with up-to-date cold storage plant and equipment staffed by experienced technicians. But especially as regards private firms it is too often the case that the installation is second-hand or old and unreliable. Paradoxically, although such plants require the best technicians to maintain them in good working order, it is almost invariably the case that their supervision is left to artisans of poor type. The reasons for this are, firstly, that a competent refrigerating engineer will not accept an appointment where his duties involve continual fitting work; and, secondly, that the plants in question are generally small and cannot carry the cost of properly trained personnel. Under such conditions cold storage supervision can be an extremely onerous and vexatious responsibility. It becomes necessary for the veterinarian, if he has not previously done so, to acquire a knowledge of refrigeration if he is to decide the many problems that will be presented to him. It is advisable to be sufficiently well acquainted with the principles of refrigeration and with the plant itself to be able to understand the purpose of the various gauges, and an occasional visit to the engine room or compressor house should be made for the purpose of inspection. Human nature being what it is, poor coal or leaking valves will often be blamed by the engineer or attendant for inefficient refrigeration, when the true cause is a shortage of ammonia in the system, due perhaps to an ill-advised attempt on his part to save his employer's pocket or to qualify for a bonus in reward for cheap working.

The greatest and commonest difficulty with cold storage plants is to keep the floors, walls, and atmosphere of the rooms clean and wholesome

under unsatisfactory or inconstant conditions of maintenance. This is especially true of the chilling or hanging rooms, which, owing to the higher temperatures which there prevail, rapidly become sour if not regularly scoured. The lighting of such places is often poor, and reliance may have to be placed on olfactory rather than on visual inspection, especially when, as occurs under conditions of almost continuous operation, the room is rarely empty.

In my experience, overloading of the plant is a radical cause of hygienic difficulties : rooms cannot be cleared often or regularly enough to allow of radical cleansing. In the larger abattoirs there is usually sufficient reserve space, power, and equipment to make possible the routine cleansing and (if necessary) defrosting of the rooms in regular rotation. But in trying to maintain a reasonable standard of hygiene under the cramped and overloaded conditions of many small plants, the veterinarian will find his resources and patience tried to the limit. Unless legal powers to control the whole operation of such places are in his hands, bone-taint and other complications are likely soon to arise, making inspection doubly onerous. Where such powers have not been obtained, an inefficient or obdurate proprietor often does not hesitate to blame the inspector for what in reality are the consequences of his own negligence. If the position becomes acute and legal powers (perhaps because of local politics) are not forthcoming, the veterinarian may have to await an opportunity when meat, unsound through the causes mentioned, has been delivered at retail premises; when he may seize such meat and initiate a prosecution for exposure for sale. The publicity which such proceedings threaten is usually sufficient to frighten recalcitrants into submitting to instructions for altered arrangements.

The recent developments in cold storage cabinets for butchers' shops make it necessary for the veterinarian to extend the scope of his inspections to include these. Cabinets in which, as is commonly the case, all sorts of meat and other food products are stored can easily become incubators of various infections.

Export Abattoirs.

Where meat is being prepared for consumption overseas, the responsibility of the veterinarian is greatly increased; for the product is likely to be reinspected at its destination, where any defects will be noticed. Further, the retail butchers to which it eventually finds its way are, on account of the intensity of the competition prevailing in this trade, quick to complain of defects in quality or preparation.

The bulk of the meat exported from South Africa is for the Italian Contract, and since the usual price of the product is about 2*d.* (or less) per pound, the standard of the product and of its preparation is considerably lower than the English chilled meat standard—the most severe of its kind.

The writer has handled boned beef and edible offals in large quantities for the English market, and a few remarks on the supervision of this traffic may be of interest.

In all cases, irrespective of the destination of the carcass, a full routine examination according to English chilled meat importation requirements is conducted; these requirements specify incision of the prescapular, superficial inguinal (or supramammary), precrural, ischiatic, and internal iliac lymphatic glands, in addition to the ordinary cuts. In the case of boned beef, the incised prescapular and popliteal glands must be brought to the outside of the meat and skewered with cut surfaces exposed. During inspection of the head, it is advisable to open the retropharyngeal glands, since the usual transverse cut makes it difficult subsequently to skewer these glands in the approved manner. After detachment of the tongue, both the retropharyngeal and the submaxillary glands are incised longitudinally and skewered to show the incised surfaces. In each case the two glands are coupled on one skewer, the former on the upper surface and the latter on the lower surface of the base of the tongue.

I find, in practice, that a routine inspection according to English chilled meat standards allows many defects to be detected which might otherwise pass unobserved. For example, since it is necessary, in order to incise the lymphatic glands of the hindquarter, for the examiner to work from a raised platform, it is easy to palpate for croup and anal abscesses, which are occasionally found. It is remarkable too, how often the prescapular gland is swollen or infected in the absence of any other demonstrable lesion.

In regard to products destined for the overseas markets, the veterinarian should concentrate on (1) absolute cleanliness, *i.e.* freedom from abattoir dirt or contamination, and (2) rapid freezing.

(1) To ensure cleanliness, continuous supervision is required, since the average native slaughterman concerns himself only over the output and the earliest completion of the day's killing. A common cause of soiling lies in overhead rails which are too low. They should be high enough to allow of easy severance of the pluck from its cervical attachments, not compelling the slaughterman to bend almost double in order to effect this.

It should be remembered that it is impossible to cleanse a carcass completely of bowel contents after drying, at least not without leaving some tell-tale stains or dressing marks.

Of edible offals, the liver probably requires the most careful handling. It is wellnigh impossible to remove dried dirt or stains without rupturing the capsule, and the peculiar sheen of the latter is in any event destroyed by washing. To remedy this, egg-white is sometimes used as a dressing—a questionable procedure.

The boning of meat is usually carried out on the day following

slaughter, the meat having been allowed to set overnight in the chilling room. During this operation the continuous attendance of the inspector is required. It once happened (in spite of the fact that no smoking is allowed) that a native's pipe was inadvertently wrapped up in a boned quarter. Nothing further transpired until an irate English butcher sued the company's agents for the price of a new mincing machine.

In the storage rooms also, rigorous supervision must be maintained. It is not unknown for the works engineer to attempt to hide a deficiency of ammonia by diverting the circulation to the rooms alternately. The floor-ice begins to thaw and the pipes to drip, and stains appear on the meat due to splashing and blood-drip, etc. In such cases the veterinarian must act promptly and, if necessary, suspend further slaughtering, since the management may prefer to take a chance with the product rather than to throw the staff into idleness. He must be prepared to stand by his judgment of values in deciding whether or not the product (and perhaps his own reputation) deserve priority over the company's immediate interests.

When working with a subordinate staff, make it a rule that each storage room is opened daily for the purpose of taking the temperature, and keep a record of the latter. Tampering with the ammonia circulation will thereby be discouraged.

(2) Rapid freezing allows the product to retain much of its original bloom. The difference in appearance between rapidly frozen and slowly frozen meat has to be seen to be fully appreciated : the former retains its bright attractive aspect while the latter becomes dark and opaque. Generally speaking, rapid freezing demands a reserve of space and power which is commonly not available : most works are taxed to capacity, and further most of the old cold storage plants were under-piped, rapidity of freezing not having been a prime consideration save for export purposes. The rate of freezing depends of course upon the relation between the area of the cooling surface and the capacity of the space to be cooled, in other words, the ratio between the length of standard piping and the cubic capacity of the room. There are cases in which one might be justified in enforcing a limitation of slaughtering for the sake of the quality of the product. The problem arising under conditions of unsatisfactory management or with an inefficient plant are, in fact, never ending. Even under conditions which ordinarily are unexceptionable, difficulties may occur at peak periods when there is a tendency to overwork both plant and staff.

Transport to the coast from inland centers introduces possible complications for the veterinarian. Especially when the plant is working to capacity, the sequence of operations under his control can be as completely upset by the non-arrival of the expected insulated trucks as by a breakdown in the engine room. He may perhaps be pressed by the management to allow slaughtering to proceed, on the assurance that on the morrow

the trucks and subsequently, therefore, an empty freezing room will be available.

The veterinarian has to assess the effect of an overcrowded chilling room in which carcasses are so closely packed that the overhead fans are virtually useless in maintaining the air circulation and in which the temperature remains at a dangerously high level.

With the development of the export trade in so perishable a product as meat, especially in our subtropical climate, the veterinarian's responsibility must increase. Signs are not wanting that if he is not to find the position lost through his own default, he must be prepared to insist on exercising the authority which is his prerogative.

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A Zoological Survey and Its Significance to the Veterinarian. *

By A. D. THOMAS, D.V.Sc., Onderstepoort.

A zoological survey, to put it a little crudely perhaps, amounts to a stocktaking of the fauna of a given country or locality, taking into consideration the geographical distribution, concentration, and life history of each species. In a wide sense its aim is firstly to find out which animals are useful to mankind, to protect them, favour their reproduction, and generally to improve their usefulness. Secondly—and perhaps this is of greater importance—it attempts to determine which animals are harmful or inimical to man and to devise ways and means of destroying, combating, or controlling them and in every way possible to minimize the damage they do directly or indirectly.

The recognition and application of these principles is of course not new. They have been practised, in part at any rate, from time immemorial, in fact ever since man first thought of selecting and domesticating animals for his own use.

The great possibilities which now commend the wider application of this type of research are, however, of more recent origin, following the impetus received by biology as a science and the patent success which has attended the solution, whether partial or complete, of some of our greatest pest problems. While a great deal of work of this nature has thus been done already, the efforts made have been directed usually to the pressing solution of one or other specific biological problem, such, for instance, as the mosquito, locust, and rodent problems, etc., rather than to the systematic study of the fauna as a whole. However, the scientific importance and advantages to be derived from such a systematic study or survey of the fauna of a country has not been entirely overlooked. In the U.S.A. as far back as 1885 a modest movement was started by amateurs with a view to collecting and pooling information regarding useful and harmful birds. This has now developed into an elaborate and active state organisation—the Biological Survey—which deals with every possible phase of wild life research.

The reason why similarly specialised organisations do not exist in many other civilised countries is probably the fact that the fauna in closely populated countries is almost negligible. Some excellent work on the

* Paper read before the S.A.V.M.A. Congress at Onderstepoort on the 30th October, 1936.

acclimatisation and selection of suitable wild animals has, however, been done in Russia.

In the Union, as in other parts of Africa where growing farming communities have to struggle for existence side by side with what is undoubtedly the most varied and extensive fauna in the world, it is not surprising that serious biological clashes arise and that the need for their solution at times becomes acute. Agriculture and stock-raising have been possible only with continuous and assiduous destruction of predatory and destructive vermin and slaughter of game to make room for stock and abate disease. After all these years the fight is still being waged at great expense : pests and vermin are still with us, while many relatively harmless species are threatened with extinction. Thus the time is overdue to review the whole situation and to attack these various questions on a sound scientific basis.

As long ago as 1920 a strong movement was afoot to launch a Zoological Survey of the Union. Sir Arnold Theiler was among the most active protagonists of the scheme, and judging by the records of his stated views no one realised better than he did the possibilities of such a project in regard to veterinary science and how it should be organised and carried out.

A meeting of heads of divisions, museums, universities and other bodies was actually convened by the then Minister of Mines and Industries and took place at Pretoria. Elaborate resolutions and suggestions were framed, but the project was never even started. Failure to stress the economic side of such an undertaking and lack of enthusiasm and agreement between members who served too many and varied interests were probably some of the immediate causes of its premature abandonment.

During the cycles of financial depression which followed, efforts were made to revive the proposal, but nothing came of them. It was only in 1935 that with the alarming increase of rabies among the small veld carnivores, the extensive outbreaks of African swine fever and its relationship to wild pigs, and the prolonged difficulties attending tick eradication in connection with East Coast fever, the Director of Veterinary Services succeeded in impressing on the Government the need for exhaustive research in the bionomics of these various vectors of disease.

A small sum of money for this purpose has accordingly been placed on the estimates this year (1936) as a beginning. It is being administered by the Director of Veterinary Services in consultation with certain members of the staffs of museums and other institutions, who have special knowledge and interest in this kind of work.

It is indeed gratifying to learn that the necessity for this type of research has at last been realised by the Government and what is more that the work will be initiated and carried out under the auspices of the

Division of Veterinary Services. In this way not only can the best use be made of the opportunities offered—by attacking first the most pressing economic problems, but by vesting the administration of the scheme in a single authority there should be better co-ordination of the work. It is hoped that the co-operation of museums and other interested bodies will be obtained and that later the work will be correlated through an advisory committee representative of their various interests.

To those who may have misgivings at the outset as to whether this work should be entrusted to veterinarians there can be but one answer. The veterinarian who takes his vocation seriously is first and foremost a zoologist and therefore eminently suited to the task. Not only has he a special knowledge of disease and its treatment, but by virtue of his studies in anatomy, physiology, genetics, dietetics, and parasitology, he is well equipped to promote the breeding, feeding, and health protection of domestic animals and birds : Why therefore should he not apply his science with equal success to wild animals as well ? The work in itself is very interesting. It calls for ingenuity, resourcefulness, excellent judgment, and keen observation. It has this further advantage—that every one of us can take part in it all over the country. As the work progresses and practical results are achieved there is little doubt that new avenues of employment will be created for enthusiasts in this subject.

It is impossible to foresee all the benefits that will accrue from a well organised zoological survey and especially to assess in terms of money the value of such work, since much of it will be of very long range, and some of it purely of aesthetic and educational significance for the future. However, one may give examples of the more obvious practical possibilities, if only to show the extreme ramification of the work.

Useful Animals.

From the point of view of man, his stock, crops, and other pursuits, one may ultimately classify wild animals as *useful* or *harmful.*

In order to protect and even propagate those falling in the useful class, we must first know which and where they are, and in what way they are useful. In other words, although man has turned to his use many animals and insects, the possibilities of finding more are by no means exhausted.

Of primary importance in this category are (a) animals utilised for food, *e.g.* game, birds, fish. In connection with the last a special Marine Survey already exists. In the vast semi-arid pastures of this country, where stock can scarcely exist but where antelopes are known to subsist, it is quite possible, for instance, that game farming with a view to "chilled venison export" might be made to pay.

Attempts at crossing some of our large game animals (eland and S.A.

buffalo) with cattle are even now in progress here at Onderstepoort with a view to incorporating in cattle some of the desirable disease-resisting and other characteristics they are known to possess. Even if hybridisation is not successful why should the results sought for not be achieved by selective breeding ?

(b) The sources of commercial animal products, *e.g.* skins, furs, feathers, hair, ivory, eggs, guano. How do we know whether some new source of raw materials is not awaiting exploitation ?

(c) The valuable allies in combating pests of all kinds (insectivorous animals and various birds, *e.g.* bats, anteaters, locust and tick birds).—A number of these are protected, but the list could probably be added to. Parasites, bacteria, and viruses are also being pressed into service in the so-called biological control of certain pests. Unfortunately the results in general are disappointing; but further research is necessary.

(d) Game animals and birds for sporting purposes.—Large sums of money are collected annually from shooting licences. In return for this revenue and to prevent abuse of this sport most of our present game laws are enacted. More precise information regarding the feeding and breeding habits of game will greatly help in fixing closed shooting seasons and determining where over- and under-stocking has taken place.

(e) The fauna in its entirety as preserved in our reserves and sanctuaries has now earned public recognition as a national asset which, apart from its intrinsic worth, has an important commercial value to be reckoned with as a tourist attraction. It has been said, and quite rightly so, that after the destruction of feeding and breeding grounds and commercial slaughter, disease and parasitism rank first as destroyers of game. In our parks and reserves to-day, protection from the first three calamities is as complete as possible. Obviously then it remains to guard against disease and for this a veterinary service will have to be provided and prepared in good time. It is evident that medication must play only a minor rôle in the fight against diseases of wild animals, so that this service will have to depend largely if not entirely on the efficacy of its preventive measures.

Harmful Animals.

The list of animals, birds, insects, etc., which can be classed as harmful to man is indeed a most formidable one. Practically each species presents a problem in itself, and experience clearly shows that haphazard methods of control have never yet achieved much progress.

(a) Most important in this category are those animals, birds, insects, etc., which propagate and disseminate disease, acting either as *vectors* or as *reservoirs*. On this aspect a great deal of research has already been done in the Veterinary Division. Most of our diseases are too well known to require detailed description here. Rabies and swine fever have been

singled out because their spread is connected with *known* wild animal carriers, control measures for which it is desired to establish as soon as possible. Other diseases like heartwater, East Coast fever, anaplasmosis and trypanosomiasis require no less attention as regards their natural reservoirs and vectors—especially ticks. Actual experiments (such as those of Neitz) have shown that some antelopes are capable of carrying many of our stock diseases, and point to many interesting possibilities.

Apart from these specific problems it seems fairly clear that the systematic study of disease in all animals can lead only to good. The discovery of new parasites and the elucidation of their life-histories often shed light on other parasites whose life-habits may have been obscure or difficult to work out.

Looking for obscure virus diseases in wild animals is not so much waste of time, since it may lead to the discovery of strains that may have valuable immunising properties. Conversely, regarding a given disease, if we can find new susceptible animals the work is often greatly facilitated and advanced. Experiments with protozoan blood parasites on antelopes have already yielded useful results, as instanced by the isolation of a pure vaccine strain of anaplasmosis by passage through blesbuck and duikers (Neitz and du Toit).

Before leaving the subject of disease I should remind you of the difficulties and anomalies which attend the state veterinary medical and legal aspects of game in relation to the Stock Diseases Act. Two examples will serve to illustrate my point. (a) Great precautions are prescribed by law regarding the disposal of cattle dead from anthrax. When antelopes die of anthrax (as they have been known to) no such rules apply. (b) In regard to rabies more than once the ludicrous position has arisen that the domestic animals on an infected farm have been in quarantine and even under restraint, while the wild carnivors which actually carry the disease have been roaming about at will. This unhealthy state of affairs is closely bound up with the vexed question of ownership of game—one which is, of course, the great stumbling block in fixing responsibility for any unpleasant duties to be performed. It is believed that a zoological survey will go a long way to assist in rectifying these anomalies by providing precise information regarding the presence of disease in, and methods of control of such animals.

Again, our regulations provide for quarantine at ports of entry to prevent the introduction of infectious diseases from outside. It is perhaps not generally known that one of the important functions of veterinarians at such ports of entry is also to prevent the introduction and acclimatisation of healthy, yet undesirable animals which may (as has happened more than once in other countries) become pests.

Further harmful animals include (b) the predatory group (*e.g.* jackal, leopard, hunting dog, mongoose, and certain birds of prey), and (c)

destructors of crops and grains, *e.g.* baboons, wild pigs, birds, rodents, and the many insect pests. All of these but, most of all, the rodents need attention.

The rodent problem in South Africa is usually associated with bubonic plague. Although admittedly serious, this connection and the losses from plague are considered by experts as trifling when compared with the collossal aggregate losses due to destruction of crops, grain, and stores by rodents all over the country. If it is worth while controlling rodents where plague is threatened, it would seem infinitely more rational and economical to destroy rodents on a much larger scale and over larger areas purely because of the damages they do. Such a campaign, being doubly justifiable, could be pushed far more and to better purpose than is at present possible. If farmers cannot see this yet, it is still possible that facts and figures could be provided by actual survey work which might prove convincing.

Plan of Campaign.

Research on all these aspects can obviously not be started all at once. In the beginning the work will be confined to the study of those species of wild animals which, for reasons already mentioned, need urgent investigation. With the money and staff available at the start, it will take some hard work to produce tangible results within a reasonable period. At the same time it is desirable that the work and organisation shall be so arranged that they can be extended at any time and so that the fullest possible use shall be made of material and opportunities as they occur : while the urgency of the problems will determine where the work is to be started and carried out, once a given locality has been selected as much information as possible will be collected there. It would, for example, be important to determine the amount of food available for carnivorous animals.

In this way the aims of the survey as a whole will not be lost sight of, and gradually material and data will accumulate which can later be worked up.

Procedure.

In general the procedure will be as follows :—

The animals required will be obtained and destroyed in the most humane way convenient (by shooting, gassing, poisoning, etc.) so as to be available for examination as soon after death as possible.

A complete autopsy will then follow, with as detailed a description of the animal and findings as found expedient.

The *material to be collected* includes :

i. Ectoparasites—ticks, lices, fleas, etc.

ii. Smears—of blood, spleen, kidney, and lesions if any.
iii. Pathological specimens, when found.
iv. Endoparasites—worms, and faecal matter for their eggs.
v. Anatomical specimen—to include in every case the complete skin (with digits) and the skull for identification of the subject.
vi. In special cases live ticks, snails, flies, and other vectors for transmission tests. Fresh blood, organs, or lesions for inoculation and bacteriology.
vii. Stomach contents.

The *information to be particularly noted* includes :

i. Local vernacular name(s) of the subject.
ii. Sex, age, and general *signalement.*
iii. Nature of stomach contents (in detail).
iv. State of pregnancy, if a female.
v. Known feeding habits, especially if destructive.
vi. Habitat.
vii. Details of locality.
viii. Any other peculiarities.

Receptacles, preservatives, special labels, and forms will be available for collectors. The forms are drawn up in such a way that most of the information required has merely to be filled in under headings, thus minimizing the amount of descriptive writing. On the reverse of each form will be found concise directions for the collection, preservation, and despatch of specimens.

Staff and Assistance.

In many fields of science, where it is out of the question to employ a paid staff to do all the observation and collection (*e.g.* in astronomy and in the U.S.A. bird survey, where an enormous field has to be covered and a very great number of minor yet important observations have to be made over long periods) it has been found that teams of amateurs, when properly organised, can render invaluable services. Incidentally, the amateurs themselves derive considerable pleasure and instruction from such a hobby.

In this country also it should be possible to enlist the co-operation of persons with naturalistic tendencies in order to accelerate the collection of data. It is inevitable that a certain amount of the information obtained in this way would be unreliable and useless and would have to be discarded; yet if only a few enthusiastic and reliable collectors do co-operate, the work would certainly benefit greatly at very little cost to the state.

As far as permanent staff is concerned, only a few men will be available at the start. They must be interested in the work and well equipped. A motor lorry for travelling and field research is at present on order. No

doubt the development and expansion of staff will be dependent on the progress achieved.

It is hoped also that veterinarians all over the country who are particularly favourably situated for collection will collaborate as occasion arises. Even one or two specimens a month would mean a lot.

In short, then, a zoological survey will afford a means of fostering a better general understanding of the wild life around us, whether it be for the preservation and protection of useful animals or for the better control of economic pests, vermin and disease. Incidentally it also offers new opportunities to the veterinarian to extend the sphere of his activities. We should make the most of such opportunities by taking an active lead in these matters.

Acute Tulp Poisoning in a Clydesdale Mare.

By N. F. VILJOEN, M.R.C.V.S., Bloemfontein.

On the 22nd November, 1936, a pure-bred four-year-old Clydesdale mare, "Sandyknowse Rose," went, along with a number of other mares belonging to the Bloemfontein Municipality, to graze in one of our paddocks. At 4 p.m. the grooms, on fetching the animals in for the evening grooming and stabling, noticed that "Rose" was off colour. Two hours afterwards she developed a violent colic. I was unavailable that afternoon, but the stable foreman immediately administered the following drench, which it is my practice to keep for emergencies : tinct. opii 1 ounce, tinct. hyoscyami 1 ounce, aquam ad Oi.

This had scarcely any carminative effect. On visiting the mare at 8.30 p.m., I at once administered morphia, with very little result. An hour later another drench consisting of hyoscyamus and chloral hydrate was given. This quietened the patient, and she then lay down and "dozed" for a couple of hours. However, at midnight she again went into violent spasms of agony, and she died five minutes later.

At post mortem, I was surprised to find in the stomach almost a handful of poorly masticated fresh tulp. A very severe gastro-enteritis was present. Severe inflammation of the colon, with a sero-gelatinous coating and a purple discoloration of its mucosa was the most prominent feature.

Although cases of acute tulp poisoning in equines are very rare in this country, this is not the first on record. The Johannesburg Municipality in 1910 suffered the loss of no fewer than 25 mules from this cause. Steyn

(1934) mentions that tulp poisoning under natural conditions is somewhat rare in horses, but not unknown.

This mare was imported from Scotland about twelve months ago. When on the day of her arrival in Bloemfontein I was examining her, the native groom, with the customary carelessness of his kind, let her head go, and I noticed that she made straight for a nearby patch of tulp. I warned the stable staff to be very careful of tulp where imported mares were concerned, since, although South African bred horses would not deliberately eat this plant, this could not be assumed to hold good in the case of imported horses, to which tulp was an unknown plant.

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STEYN, D. G. (1934). *Toxicology of Plants in South Africa*, p. 557. C.N.A., Johannesburg.

Book Reviews.

Professor Gage's *The Microscope* * has gone through sixteen editions since 1908. It contains more than 600 pages crammed with information about microscopes and microscopical technique. Special chapters deal with the polarizing microscope, the micro-spectroscope, and the dark and bright field microscopes. In addition there are sections on the ultra-microscope and on the comparatively new technique of micro-incineration.

In spite of its size, the book is probably adapted more to the needs of the student than to those of the specialist, as it gives useful information about a number of things rather than an exhaustive consideration of any one subject. The student would do well to carry out some of the simple exercises which are described : they will give him an understanding of the microscope and a certainty in manipulation that many mature workers lack.

The arrangement of the book leaves something to be desired, there being some difficulty in tracing general information. For example, the theory of the microscope, which could well have come at the beginning, is scattered through a number of chapters—part in the chapter on the bright field microscope, part in the introduction, a good deal in the chapter on the polarising microscope, and so on. In such a large volume this lack of arrangement is apt to be a nuisance. Among minor defects, the book

* *The Microscope*, by Simon Henry Gage, Emeritus Professor of Histology and Embryology, Cornell University. 16th Ed., 1936. Pp. viii + 617, figs. 313. Comstock Publishing Company, Ithaca, New York. Price $ 4.

suffers from a plethora of somewhat pointless illustrations and unnecessary repetitions of illustrations, which needlessly increase its size.

The ideal that this work is designed to foster is most commendable, *viz.* that everyone who uses a microscope should be an intelligent microscopist.

M. S.

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Brook's *Studies of the Spine of the Dog* † is an interesting publication which will undoubtedly encourage the use of specialized diagnostic methods for selected cases. The work is based on the technique of puncture of the *cysterna magna* for the purpose of collecting samples of cerebro-spinal fluid for examination, and of injecting radio-opaque substances to facilitate photography of the vertebral column.

The anatomical details and the technique are so clearly described and well illustrated that after a certain amount of practice other workers should be able to undertake the clinical application of these procedures. Particularly useful are the figures for the cellular content of the cerebro-spinal fluid and the photographs of normal dogs provided by the author.

Those interested in such specialized examinations will look forward to further clinical studies by Dr. Brook of various canine diseases, especially those that are characterised by nervous symptoms.

The book is divided into two parts. Part I deals with the *cysterna magna*—its anatomy, surface markings, the actual puncture, the effects of puncture, observations on the normal cerebro-spinal fluid, and the clinical application of this knowledge. Part II contains the radiographical technique and the interpretation of photographs. Regarding the latter, an appendix assists the operator in becoming familiiar with the normal. The work is completed by a number of excellent photographs illustrating the technique of puncture and of iodized oil injections.

The book is a well-arranged and clear description of a field of diagnostics which in the past has received but little attention.

B. S. P.

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† *Experimental and Clinical Studies of the Spine of the Dog,* by Geoffrey B. Brook, D.Sc. (Edin.), F.R.C.V.S., Centenary Post-Graduate Research Fellow, Royal (Dick) Veterinary College, Edinburgh, 1927–28. Pp. ix + 122, Figs. 50. London : Balliere, Tindall & Cox, 1936. Price 5/-.

THE ARMY VETERINARY SERVICE IN SOUTH AFRICA, 1881—1914.

CHAPTER I.

The Army Veterinary Department, 1881—1899.

(a) Introduction (Tables I and II). (b) Narrative: (i) Bechuanaland Expedition 1884–5; (ii) Zulu Rebellion 1888; (iii) Matabeleland Rebellion 1896. (c) List of Army veterinarians after departmentalisation in 1881 (Table III). (d) Statistics relating to Army horses and mules (Table IV). (e) Conclusion.

(a) Introduction.

The history of the Army Veterinary Service having been described *up to March* 1881 (1), when the regimental system (except for the Household Cavalry) was replaced by the Army Veterinary Department (2), it is proposed to continue with the record of the Army Veterinary Service in South Africa until the outbreak of the Great War (August 1914), when the Imperial troops returned to the Mother Country.

As before, the history of the A.V.S. is largely the history of the cavalry, and for this reason all movements of cavalry regiments have been summarised in Table I. Since arrivals and departures up to the *end* of 1881 have already been published—see Column 1—(1) particular attention should be paid to Columns 2, 3 and 4, which deal with movements from 1882–97, 1898–1901, and 1902–1914 respectively. The table, as it stands, gives at a glance all cavalry movements from 1795–1914. Veterinary surgeons (3), it must be emphasised, were also attached to other units, *e.g.* Artillery, Army Service Corps, Remount Department etc., but no tabulated statement of the movements of these has been prepared, a task that could only be undertaken in London (4). According to Smith (1927) the veterinary establishment of the Royal Field Artillery prior to 1854 was three, but in that year it was increased to eleven, in 1856 to twenty-two, and in 1861 to twenty-eight, including, it would appear, six appointed for the first time to the Royal Horse Artillery. In 1878 when departmentalisation of the A.V.S. was commenced, veterinary officers were transferred from the Artillery to the Veterinary list (p. 58).

Be that as it may, the most important information, *i.e.* the periods of

(1) CURSON, H. H. (1935). Matters of Veterinary Interest, 1795–1881. *Jl. S.A.V.M.A.*, 6: 19, 196, 274.

(2) In 1903 was created the Army Veterinary Corps, which until 1906 consisted of N.C.O.'s and men, the officers remaining in the A.V.D. In 1906 amalgamation took place with the title Army Veterinary Corps. In 1918 the prefix "Royal" was conferred.

(3) By the Royal Warrant of October, 1891, the designation was amended to Veterinary Officer (Smith 1927, p. 192).

(4) From 1905, as a result of reduction of establishment, veterinary officers were allotted to stations and not to units (Smith 1927, p. 211).

service of officers of the Army Veterinary Department in South Africa is available, thanks to the courtesy of Major G. E. Oxspring, R.A.V.C., who has furnished these details.

Since the greatest event during the period 1881–1914 was the Second Anglo-Boer War (1899–1902), it is considered advisable to narrate the events as follows : (a) from 1881–1899, (b) 1899–1902 (Second Anglo-Boer War), and (c) 1902–1914.

The chief source of information is the *Annual Statistical and General Report of the Army Veterinary Service,* but an official account of the Bechuanaland Campaign (1884–5) by Duck and *Reports on African Horse-sickness* (1888) by Nunn are available. The history of the (b) 1899–1902 period has been fully described by Sir Frederick Smith (1912–14) in his *Veterinary History of the War in South Africa* 1899–1902, and it is not proposed to refer at length to this campaign. Reference to this will be indicated simply by the page number, *e.g.* (Smith, p. 306). His *History of the Royal Army Veterinary Corps,* 1796–1919 also proved invaluable. Contributions from A.V.D. officers to the periodical literature will be listed in the final chapter.

It is deemed advisable to include in the Introduction a summary giving the names of the Principal or Senior Veterinary Surgeons (3) in South Africa along with other details, *e.g.* period of service. See Table II.

The difficulty of compiling this record will be obvious when it is explained that no set of the *Annual Statistical and General Report of the Army Veterinary Service* is obtainable in South Africa. Apart from a few private copies, the necessary reports were kindly lent by Dr. Fred. Bullock, and to him cordial thanks are extended. Dr. Bullock has as usual gone to much trouble in supplying not only literature but also information of all sorts. To Dr. P. J. du Toit, Director of Veterinary Services (Department of Agriculture), Pretoria, special thanks are due, for he allowed members of his staff to assist with the typing and photography.

Explanatory Notes to Table I.

Not only are the country of origin and month and year of arrival indicated, but also the country of destination and month and year of departure. If horses were brought to South Africa, the country of origin is set in black type, *e.g.* 1st D.G. in 1879; in case of doubt in italic type, *e.g.* 16th Lancers in 1900.

Horses sometimes accompanied the regiment on departure, *e.g.* 12th Lancers in 1912, but more frequently horses were left in South Africa, either (a) for the use of the relieving unit, *e.g.* 20th L.D. 1806, 12th Lancers, 1853, 17th Lancers, 1879, 15th Hussars, 1881, 6th Dragoons, 1890, 11th Hussars, 1892, 9th Lancers, 1898 and 1910, or (b) to be sold, *e.g.* those of the 1st D.G. in 1880.

It must be emphasised that in some cases only part of the regiment was in South Africa, *e.g.* 20th L.D. in 1806, and 1st D.G. at the end of 1880 and beginning of 1881. In other cases only a proportion of the horses accompanied the men, *e.g.* 20th L.D. in 1906.

TABLE I.

Imperial Cavalry Regiments in South Africa, 1795—1914.

Regiment.	1. From	Arrived 1795—1881.	To	2. From	Arrived 1882—1897.	To	3. From	Arrived 1898—1901.	To	4. From	Arrived 1902—1914.	To
25th Light Dragoons	U.K.	1796	India	—	—	—	—	—	—	—	—	—
27th do.	U.K.	1796	India	—	—	—	—	—	—	—	—	—
28th do.	U.K.	7/1796—1/1799	India	—	—	—	—	—	—	—	—	—
Household Cavalry	—	—	—	—	—	—	U.K.	12/1899—11/1900		—	—	—
1st Dragoon Guards	**U.K.**	4/1879—early 1881	India	—	—	—	**U.K.**	1/1901—10/1903 (T)	U.K.	—	—	—
2nd do.	—	—	—	—	—	—	**U.K.**	12/1901—12/1907 (T)	U.K.	—	—	—
3rd do. (a)	—	—	—	India	1892—1895	U.K.	**U.K.**	2/1901—7/1904 (O)	U.K.	—	—	—
4th do. (b)	—	—	—	—	—	—	—	—	—	*India*	1905—1908 (C)	U.K.
5th do. (c)	—	—	—	—	—	—	**India**	10/1899—3/1902	India	*India*	3/1904—11/1908 (O)	U.K.
6th do. (a)	—	—	—	—	—	—	**U.K.**	11/1899—8/1902	India	**India**	10/1908—12/1912 (O)	U.K.
7th do. (b)	U.K.	6/1843—4/1848	U.K.	—	—	—	**U.K.**	1900—1904 (N)	U.K.	—	—	—
1st Dragoons	—	—	—	—	—	—	**U.K.**	11/1899—9/1902	U.K.	India	11/1911—8/1914 (T)	U.K.
2nd do.	—	—	—	—	—	—	**U.K.**	12/1899—1904 (T)	U.K.	—	—	—
3rd Hussars	—	—	—	—	—	—	**India**	12/1901—10/1902	India	*India*	12/1907—11/1911 (T)	U.K.
4th do.	—	—	—	—	—	—	—	—	—	India	11/1905—11/1909 (T)	U.K.
5th Lancers (g)	—	—	—	—	—	—	**India**	3/1898—10/1902	U.K.	—	—	—
6th Dragoons (c)	**U.K.**	2/1881—10/1890 (N)	U.K.	—	—	—	**U.K.**	11/1899—1902	U.K.	—	—	—
7th Hussars	**U.K.**	4/1881—3/1882 (N)	U.K.	India	10/1895—10/1898	U.K.	**U.K.**	12/1901—11/1905 (T)	U.K.	—	—	—
8th do.	U.K.	11/1796—2/1803	Egypt India	—	—	—	**U.K.**	3/1900—10/1903 (T)	U.K.	—	—	—
9th Lancers	—	—	—	**U.K.**	9/1896—3/1898	India	**India**	10/1899—3/1902	India	*India*	11/1906—11/1910 (T)	U.K.
10th Hussars	—	—	—	—	—	—	**U.K.**	12/1899—9/1902	India	India	11/1912—8/1914 (T)	U.K.
11th do.	—	—	—	U.K.	8/1890—10/1892	India	—	—	—	—	—	—
12th Lancers	U.K.	10/1851—11/1853	India	—	—	—	**U.K.**	11/1899—9/1902	India	India	11/1910—12/1912 (T)	U.K.
13th Hussars (d)	—	—	—	*India*	end 1884—10/1885	U.K.	**U.K.**	12/1899—9/1902	U.K.	—	—	—
14th do. (e)	**India**	3/1881—11/1881 (N)	India	—	—	—	**U.K.**	1/1900—3/1903 (O)	U.K.	—	—	—
15th do. (f)	India	1/1881—11/1881 (N)	U.K.	—	—	—	—	—	—	India	11/1909—12/1912 (T)	U.K.
16th Lancers (g)	—	—	—	—	—	—	**India**	1/1900—10/1904 (T)	U.K.	—	—	—
17th do. (h)	**U.K.**	1879	India	—	—	—	**U.K.**	3/1900—9/1902	U.K.	—	—	—
18th Hussars (d)	—	—	—	—	—	—	?	1898—1902	?	—	—	—
19th do. (f)	—	—	—	—	—	—	**India**	10/1899—1/1904 (O)	U.K.	—	—	—
20th do. (e)	**U.K.**	1806	S. America	—	—	—	*India*	12/1901—3/1903 (O)	Egypt	—	—	—
21st Lancers (h)	U.K.	1806—1817	India	—	—	—	—	—	—	—	—	—
Cape M.R.	—	1827—70	—	—	—	—	—	—	—	—	—	—

TABLE II.

The subjoined list gives the names of those in charge of the A.V.S. in South Africa from 1881 to 1914.

The dates marked with an asterisk were supplied by Sir Frederick Smith (letter of March 3rd, 1927).

Designation.	Name.	Period.	Headquarters.	P.V.S. (War Office) or Director-General – from 1891.	Head, Remount Dept. 1898—1913.
P.V.S. (local)	J. D. Lambert	1881	Pietermaritzburg	J. Collins (1876–1883)	During the period 1898—1913 the A.V.D. was subordinate to the Head, Remount Department, whose title was altered five times in 13 years.
S.V.S.	F. Duck	*1882—85	,, ...	G. Fleming (1883–1890)	
S.V.S.	F. F. Crawford	*1886	,, ...		
S.V.S.	R. Rowe (5)	*1887—90	,, ...		
S.V.O.	Vety.-Major J. C. Berne (6)	*1891—95	,, ...	J. D. Lambert (1890–1897)	
S.V.O.	Vety.-Major R. T. Frost (6)	*1896—98	,, ...		
P.V.O.	Vety.-Lieut.-Col. I. Matthews	1899—1903	Period of 2nd Anglo-Boer War	F. Duck (1897–1902)	Maj.-Gen. E. A. Gore (1898—99).
P.V.O.	Vety.-Lieut.-Col. F. Smith	1903—05	Pretoria (Artillery Barracks)	H. Thomson (1902–1907)	Maj.-Gen. W. R. Truman (1900).
P.V.O.	Vety.-Lieut.-Col. J. A. Nunn	1905—06	,, ,, ,,		
P.V.O.	Vety.-Lieut.-Col. L. J. Blenkinsop	1906—09	,, ,, ,,	F. Smith (1907–1910)	Col. C. E. Beckett, C.B. (1901—2). Maj.-Gen. F. W. Benson, C.B. (1903—7). Maj.-Gen. C. E. Heath, C.V.O. (1908–11).
P.V.O.	Vety.-Lieut.-Col. A. F. Appleton	1909—13	,, ,, ,,	R. Pringle (1910–1917)	Col. J. Fowle, C.B. (1912).
Asst. Dir. Vet. Serv.	Vety.-Lieut.-Col. C. F. Nuthall	1913—14	,, ,, ,,		Col. W. H. Birkbeck, C.B., C.M.G. (1913).

The G.O.C. Imperial Troops had his headquarters at the Castle, Cape Town, until 1902, when he transferred to the Artillery Barracks, Pretoria.

The Office of the Assistant Director of Veterinary Services, at any rate in Colonel Appleton's time, was that on the extreme left of the ground floor of the Artillery Barracks—see Fig. 20. (Information supplied by F. E. H. Appleton, Esq., Onderstepoort.)

From 1881—Nov., 1901, the A.V.D. fell under the control of the Adjutant-General and thereafter under the Quarter-Master-General. The supreme heads were therefore: *Adjutants-General.* Gen. Sir C. H. Ellice (1-11-76—1-4-82), Lt.-Gen. Sir G. J. Wolseley (1-4-82—1-8-82), Lt.-Gen. R. C. H. Taylor (1-8-82—1-11-82), Gen. Garnet J., Lord Wolseley (1-11-82—18-2-85), Lt.-Gen. Sir A. Alison (18-2-85—1-10-85), Gen. Garnet, Lord Wolseley (1-10-85—1-10-90), Gen. Rt. Hon. Sir R. H. Buller (1-10-90—1-10-97), Gen. Sir H. Evelyn Wood (1-10-97—1-10-1901) and Lt.-Gen. Sir T. Kelly-Kenny (1-10-01). *Quarter-Masters-General.* Gen. Sir C. M. Clarke (16-9-99—27-4-03), Lt.-Gen. Sir Ian Hamilton (27-4-03—2-2-04), Maj.-Gen. H. C. O. Plumer 2-04—18-12-05), Gen. Sir W. G. Nicholson (18-12-05—2-4-08), Lt.-Gen. Sir H. S. G. Miles (2-4-08—3-6-12), and Gen. Sir J. S. Cowans (3-6-12—15-3-19). (Letter 27-8-35 from A. S. White, Esq., c/o Library War Office.)

(5) Col. Rowe wrote (letter of 24-2-27) that he was S.V.O. for the period 1886—1892.

(6) With this exception photos are available of all officers who were in command A.V.S., South Africa. Biographical notes and photos are given by Smith (1927) of Collins, Fleming, Lambert, Duck and Thomson. Biographical notes concerning Smith and Pringle are contained in Smith (1927), and photos are given by Curson (1935) and Goldbeck (1908) respectively.

Again, the titles of some of the regiments have been changed on several occasions, *e.g.* (i) the 8th Hussars at the time of the First Occupation (1795–1803) having been 8th L.D., (ii) the 20th Hussars in 1806 having been 20th L.D. and (iii) the 21st Lancers in 1806 having been 21st L.D. Not only were these alterations of designation effected after the Peninsular War, but certain regiments, *e.g.* 25th, 27th and 28th L.D., ceased to exist at the same time. Certain amalgamations took place in 1922 and these are indicated by the same letter (a–h) after the regiments concerned. The Cape Mounted Riflemen of 1827–70 although consisting largely of Hottentot troopers was an Imperial regiment.

Up to 1853 all garrison stations during peace were in the Cape Colony, from 1795 to 1810 only in the present Western Province and later chiefly along the stormy Eastern Frontier. After 1853 and until the 2nd Anglo-Boer War cavalry regiments were stationed in Natal, first at Pinetown (until May, 1886) and then at Fort Napier (Pietermaritzburg).

After the Peace of Vereeniging (May 31st, 1902) the Colony or Province is represented by the capital letter C., N., T. or O., designating Cape Colony, Natal, Transvaal, or Orange Free State respectively, each of which is the initial letter of the territory in question.

The compilation of the table has only been possible owing to the courtesy of many of the officers commanding the various regiments. Their kindness is much appreciated.

(b) Narrative.

1881. It has already been shown that at the end of 1880 there was no Army Veterinary Surgeon in South Africa, but owing to the First Anglo-Boer War (7) probably seven veterinarians with James Lambert as P.V.S. were in Natal by the end of March, 1881 (8). Duck's movements at this time are not clear, but according to the *Quarterly Journal of Veterinary Science in India* (1882–1883, p. 213), he took to Bombay 650 mules, used in the Zulu and First Anglo-Boer Wars, as a nucleus of the Mule Transport Corps. They included South African and South American mules which were described by the *Times of India* as "without exception the finest body of animals which have passed through Bombay of recent years."

The chief cavalry centre was Pinetown (until May, 1886), but Mounted Infantry, Artillery and Transport were stationed at Fort Napier, Pietermaritzburg, where the P.V.S. had his headquarters.

A reference of interest is a notice from the Military Department of India [*Natal Government Gazette* 3-5-81 (9), Government Notice 174 (1881)] drawing attention to the proposed purchase, by Army remount

(7) For a veterinary history see *Vet. Jl.*, Feb., 1934.

(8) These appear to have been F. Duck, J. Reilly, G. Durrant, W. D. Sartin, W. Pallin, C. Rutherford and T. Caldecott.

(9) This Gazette contains a report by the Colonial Veterinary Surgeon (S. Wiltshire) on Bluetongue of sheep. See Govt. Notice 169/1881. In Wiltshire's *Ann. Rept.* for 1881 reference is made to the "considerable difficulty" in obtaining suitable remounts for the Police "owing to the requirements of the military authorities."

agents at 15 centres in India of 900 horses. Of these 771 are described as "walers", 69 as "country breds", and 60 as Arabs and Persians. The prescribed age was 4–7 years, except for "country breds" which might be 2½–3½ years. Horses and mares were accepted provided the latter were not in foal, and the average price was 550 rupees ([10]). This notice recalls the prosperous years of 1840–60 when the Indian Government stationed a remount officer at the Cape.

The *Natal Government Gazette* of July 26th, 1881, (Govt. Notice 315/1881) contains "with the permission of the Military Authorities" a paper by P.V.S. James Lambert on Glanders and Farcy, which he

Government Notice No. 315, 1881.

WITH the permission of the Military Authorities, His Excellency the Administrator of the Government directs the publication, for general information, of the following Paper on Glanders and Farcy, by the Principal Veterinary Surgeon to the Forces, James Lambert, Esquire, F.R.C.V.S.

By His Excellency's command,

M. H. GALLWEY,
Attorney-General,
For Colonial Secretary.

Colonial Secretary's Office, Natal,
July 22nd, 1881.

"Glanders and Farcy," by James Lambert, F.R.C.V.S., Principal Veterinary Surgeon to the Forces, Natal.

Glanders and Farcy are far too common in South Africa, and especially in Natal. It is therefore very desirable that all interested in horses should have some knowledge of the symptoms of these destructive diseases, and of the means best calculated to prevent their diffusion.

Glanders and Farcy attack many different kinds of animals, including man, but we shall here only consider them as affecting horses, mules, and asses, in which the symptoms are the same.

Glanders and Farcy are contagious, that is, "catching" diseases, therefore no animal suffering from either should be

Fig. 1.

GOVERNMENT NOTICE 315 (*Natal Government Gazette*, July 26th, 1886).
An early military contribution on Glanders.

describes as "far too common in South Africa and especially in Natal". In regard to a suspicious case he advises that it be shown "to a veterinary surgeon without delay". Symptoms, differential diagnosis, and preventive treatment are given. In regard to differential diagnosis he refers to a condition called "Natal sores", affecting equines in poor condition. On improvement of condition the sores "very often heal completely". This disease was probably mange.

The *Natal Government Gazette* of November 1st, 1881 (Govt. Notice 448/1881) contains a "Paper upon Horse-sickness or Anthrax in South Africa", again by P.V.S. Lambert, published by direction of His Excellency the Governor (Sir Evelyn Wood). Lambert believed, as did

([10]) Regarding remounts, a letter in *Nature* (Feb. 16th, 1935) from Herbert Maxwell states that docking was practised during the Peninsular War so that British horses could be distinguished from those of the French Cavalry!

Wiltshire (*Natal Almanac*, 1878) and Hutcheon (*Report of the C.V.S. on Horse-sickness*, A. 73–1881, dated Bedford, May 17th, 1881, Cape Colony), that horse-sickness and anthrax were the same disease, probably not only on account of the greater prevalence of both during the summer, but also owing to their both being of an acute and communicable nature. This view prevailed until 1887, when Joshua Nunn established that the two diseases were distinct. Lambert, in point of fact, actually appears to have encountered horse-sickness, the nature and causes, prevention, incubation, symptoms, and treatment of which he describes. Horse-sickness, he mentions, occurred at Fort Napier "last season" (*i.e.* late summer, 1881), and he adds that in the dangerous season farmers in the Thorns (Low Country of the large river valleys) "send troops of brood mares . . . to Weston (Mooi River) so that they may avoid it" (11). It was for this reason that the Remount Department established a depôt at Mooi River on the outbreak of the Second Anglo-Boer War.

1882. Early in 1882 Lambert returned to England and was succeeded by S.V.S. Francis Duck who had seen service in the Ninth Kaffir (1878), Second Sekukuni (1878) and Zulu (1879) campaigns.

"During 1882 *Regulations for the Army Veterinary Department*" were issued, including "an appendix on *Hints for the Selection of Remounts*, which was written by General Fitzwygram" (Smith, 1927, p. 176). In this year also appeared "a new form of confidential report on veterinary officers . . . the Commanding officer" being "called upon to report on the officer's strict sobriety". This provision was not deleted until Sir Evelyn Wood (Adjutant-General) took action ten years later (Smith, 1927, p. 176).

1883. In June, 1883, retired James Collins (1830—1895), who by his enthusiasm and energy brought about the creation of the A.V.D., the first real step in the development of the military side of the profession. "He brought his period of administration to a close by obtaining the removal of the restrictions against Presentation at Court". (Smith, 1927, p. 176.)

Duck, whose headquarters were in Pietermaritzburg (probably Fort Napier), deputised for Samuel Wiltshire, Colonial Veterinary Surgeon, from August, 1883, to March, 1884, during leave of absence in Great Britain.

(11) In a Report . . . on the different forms of Anthrax in the Colony, Wiltshire (*Natal Govt. Gazette* 30-5-82, Govt. Notice 228/1882) includes not only horse-sickness, but also redwater and quarter evil. He adds that horse-sickness material supplied during the Zulu War to Dr. Greenfield of the Brown Institution, London, was injected into mice which contracted the disease! Biliary fever also seems to be included in the term anthrax, an opinion corroborated by the *Ann. Rept. of the Colonial Veterinary Surgeon* for 1883 written by S.V.S. Duck.

Smith (1927, p. 179) records that the Zulu War "demonstrated
1884. the rottenness of the contract system . . . the impossibility of dragging heavy forge carts with an army over unmade roads . . . the impossibility of forging shoes in the field in sufficient numbers to meet wear and tear". P.V.S. Fleming, who had succeeded James Collins, appreciating these difficulties, drew the attention of the Authorities in 1884 to a system whereby light forge carts could be employed and

Government Notice No. 448, 1881.

HIS Excellency the Governor directs the publication, for general information, of the following Paper upon Horse Sickness or Anthrax in South Africa. By JAMES LAMBERT, Esq., F.R.C.V.S., Principal Veterinary Surgeon to the Forces, Natal.

By His Excellency's command,
C. B. H. MITCHELL,
Colonial Secretary.

Colonial Secretary's Office, Natal,
October 29, 1881.

Horse Sickness or Anthrax in South Africa. its Nature, Causes, PREVENTION, Symptoms, and Treatment, by JAMES LAMBERT, F.R.C.V.S., *Principal Veterinary Surgeon to the Forces, Natal.*

In this paper the use of technical and scientific terms is, as far as possible, avoided, and what to many persons may appear to be very simple matters, are explained at some length, and sometimes with reiteration, so that there may be no trouble in fully understanding what we wish to convey to the minds of our readers.

Horse Sickness is Anthrax, therefore

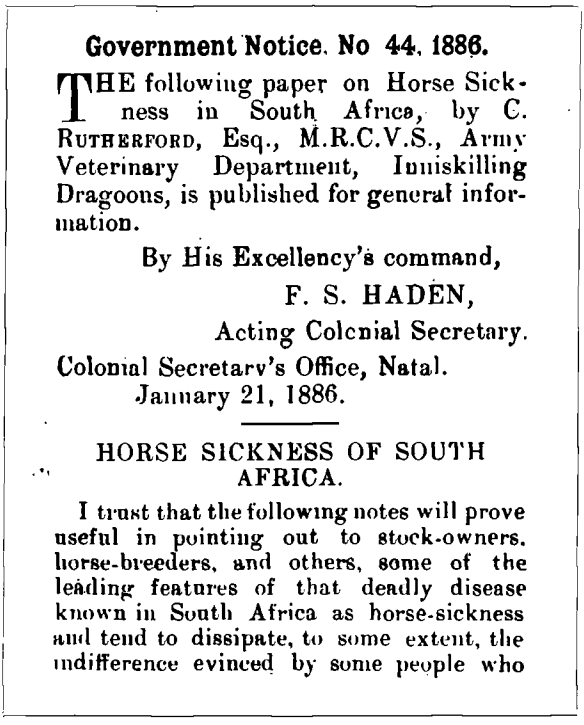
Fig. 2.

GOVERNMENT NOTICE 448 (*Natal Government Gazette*, November 1st, 1881).
An early veterinary paper on Horse-sickness.

machine-made shoes and nails used. At the same time a new concave shoe, replacing the seated pattern of 1854, was introduced. Reference to these features will be encountered in S.V.S. Duck's veterinary report on the Bechuanaland Expedition.

(i) *The Bechuanaland Expedition* 1884–85.

The above expedition, frequently known as Warren's Expedition, was merely an incident in " the scramble for Africa ", characterising the final quarter of last century. Owing particularly to tribal strife, envious eyes had been cast on Bechuanaland, the gateway to the North, by several countries; but when two Dutch republics, Stellaland and Goshen, were established by Transvaal burghers in the early eighties, two of the rivals became at once interested. The Cape Government, cautious after the Basuto War of 1880–81, was unduly slow, and it was left finally to the Imperial Government to take action. It was, of course, realised that Dutch



Government Notice. No 44. 1886.

THE following paper on Horse Sickness in South Africa, by C. RUTHERFORD, Esq., M.R.C.V.S., Army Veterinary Department, Inniskilling Dragoons, is published for general information.

By His Excellency's command,

F. S. HADEN,

Acting Colonial Secretary.

Colonial Secretary's Office, Natal.
January 21, 1886.

HORSE SICKNESS OF SOUTH AFRICA.

I trust that the following notes will prove useful in pointing out to stock-owners, horse-breeders, and others, some of the leading features of that deadly disease known in South Africa as horse-sickness and tend to dissipate, to some extent, the indifference evinced by some people who

Fig. 3.

GOVERNMENT NOTICE 44 (*Natal Government Gazette*, 21st January, 1886).

V.S. Rutherford had an extensive clinical experience of Horse-sickness.

extension to the west would interfere with the British scheme of progress northwards.

Accordingly, preparations were made at the end of 1884 to occupy the two republics and to place Bechuanaland under the Union Jack. Actually Sir Charles Warren marched through the country early in 1885 without opposition, and in September of the same year a British protectorate was proclaimed.

For the expedition not only were the military resources in South Africa utilised, *e.g.* the 6th Dragoons were transferred from Natal, and volunteers recruited, but men and equipment were also brought from England.

In November, 1884, the 6th Dragoons proceeded from Zululand to Durban and there embarked for Capetown, encamping on arrival at Wynberg near Capetown. The regiment reached Barkly West early in 1885 and on January 27th left that village for a march through Stellaland (capital Vryburg) and Goshen, reaching Mafeking on March 11th. Three mounted volunteer corps were also created, the 1st (Methuen's Horse) Mounted Rifles, the 2nd (Carrington's Horse) M.R., and the 3rd (Gough's Horse) M.R. The 1st and 2nd M.R. were recruited in the Cape Colony, while the 3rd M.R. was composed entirely of volunteers from Great Britain. Serving in Methuen's Horse as troopers were Messrs. J. B. A. Hare and E. W. Larnder, who were later distinguished officers in the Royal Army Veterinary Corps (Hare, 1930).

Veterinary Organisation. Thanks to an excellent veterinary report on the Expedition by 1st Class V.S. Francis Duck (dated December 29th, 1885, Aldershot), who acted as Local Inspecting Veterinary Surgeon, it is possible to give a résumé of veterinary arrangements. The report appeared in the *Quarterly Journal of Veterinary Science in India* (VI, p. 355) and was again published in the *Veterinary Record* of 7-1-1911.

The report consists of (a) letter of transmittal, from the Principal Veterinary Surgeon (George Fleming), War Office, to H.R.H. the Duke of Cambridge who was Field Marshal Commanding-in-Chief, (b) the report itself, (c) remarks on South African horses and mules and English horses, (d) extract from Duck's Annual Report of 1883 to the Government of Natal ([12]) on Horse-sickness, and (e) five appendices, being statistics of animals that had been sick, or had died or been destroyed.

The percentages are :—

Admissions into sick lines (excluding hired transport oxen)—25.91%.
Return of horses died (Dec., 1884—Aug., 1885)—5.54%.
Return of horses destroyed (Dec., 1884—Aug., 1885)—1.33%.
Return of mules died and destroyed (Dec., 1884—Aug., 1885)—5.58%.
Deaths among Government oxen—5%.

Duck states that "the entire staff of the Department came from England" and that "with one exception they were unaccustomed to the

([12]) At this time the 13th Hussars were in Natal, having arrived from India at the end of 1884. They were mounted on horses brought from Cairo and belonging to the 19th Hussars, then in the Sudan. The 13th Hussars returned to the United Kingdom in Oct., 1885. At least three veterinary surgeons were also in Natal, probably J. A. Woods, J. E. Elphick, and J. Finlayson.

Cape horse and to horse management in South Africa . . . Five officers of the A.V.D. were employed, one administrative and four executive". Duck was the administrative officer, while the executive officers were C. Rutherford (6th Dragoons), J. H. Cox, J. C. Berne, and another whom Smith (1927, p. 258) states was J. A. Woods, but which according to *Vet. Jl.*, March, 1885 (XX, p. 213) was F. F. Crawford ([13]). Duck also mentions that "three additional veterinary surgeons were attached to the Department for duty, Messrs. Rangeley, Brown, and Gentle, who were in the volunteer regiments. Although nominally attached to regiments, all performed general duty."

Reports on African Horse-Sickness.

HORSE GUARDS, WAR OFFICE,
27*th October*, 1888.

MY LORD,

I have the honour to submit, for the information of His Royal Highness, the Commander-in Chief, the Right Honourable the Secretary of State for War, and the Right Honourable the Secretary of State for the Colonies, three reports from First Class Veterinary Surgeon J. A. Nunn, F.R.C.V.S., Army Veterinary Department, on the destructive disease that appears among horses in certain parts of the African continent, and which, in South Africa, is known to the English Colonists as the "Horse-sickness," to the Boers as the "Paard-ziekte."

In consequence of the diversity of opinion with regard to the nature of the disease, the great mortality it occasions, its terribly rapid course, and the impotency of all attempts hitherto made to cure it, my attention has been directed to it for some time.

Fig. 4.

OPENING PARAGRAPH OF P.V.S. FLEMING'S LETTER FORWARDING V.S. NUNN'S REPORTS ON HORSE-SICKNESS.

These three reports (Bluebook 116/Cape/35) were transmitted to General Viscount Wolseley, Adjutant-General of the Forces.

Herbert Rangeley came out as a trooper, having some time previously (22-1-78) retired from the A.V.D. (private letter from Sir Francis Duck, January, 1927), but it is not known whether he was in the 1st or 2nd M.R. He graduated at London in 1870 and died in 1889. Alexander Gentle (private letter dated 6-12-26) writes that he was in the 3rd M.R., having joined as a trooper. He proceeded from Barkly West to Taungs with his regiment, where it was divided. Half the regiment, including Gentle, remained at Taungs, where, apart from a few cases of lung-

sickness among the transport oxen, the only casualties were due to falls, bruises, fractures, colics, and colds. In both the 1st and 2nd M.R., however, cases of glanders were discovered. Gentle, born April 25th, 1851, qualified at Edinburgh in 1880. Unfortunately, beyond the surname, nothing is known of Brown.

To continue with Duck's report : He states that the total number of horses used (including those of the 6th Dragoons and " C " Battery, 1st Brigade, R.A.) was 3,071 and of mules 824. The contract for horse buying was given to one firm — Messrs. Fraser & Co. — and the price was £33, which he adds " was unnecessarily high ". Col. Cottingham, R.A., of the Remount Department, assisted by 1st Class V.S. Cox, was responsible for the purchasing, *viz.* of 2,521 horses and 187 mules. Twenty farriers and shoeing smiths were sent out from England and "in lieu of the regulation field forges, light 'rivetting ' forges were . . . issued . . . and . . . proved most useful ". Horse-shoes provided by the horse contractor were of inferior metal " and could not be worked without forges ". Later " horse-shoes of the new concave pattern were . . . issued and a severe test . . . proved their efficiency . . . being readily fitted either hot or cold . . . and lasting in South Africa over two months". The saddlery supplied to the volunteer units " was of good pattern . . . and lighter than the service saddle "; but ". . . the proper material for restuffing . . . was often hard to get and freshly clipped wool was occasionally issued ". English horse blankets were issued and eye-fringes were necessary during the summer. Mealies and oats were issued as grain. Owing to various causes there was often delay in obtaining veterinary stores, e.g. bandages, horse-shoes and nails. In discussing diseases, Duck confuses horse-sickness and anthrax, giving the losses as 1.3% for horses and 0.97% for mules for the period December, 1884, to August, 1885. Eighty-four mules are reported to have died in August from horse-sickness (or anthrax fever), but it is more likely anthrax was responsible. Glanders also occurred—two cases at Setlagoli and three at Mafeking. Duck mentions that " nearly all the mares bought proved to be in foal "; also that at the close of the expedition the horses were returned to the Remount depôt and sold by public auction at Kimberley and Barkly West. The average prices were (horses) £6 16s. 6d. and (mules) £5 7s. 6d. It is quite evident that Duck was favourably impressed with the Cape horse; in fact he states so in one of his enclosures—(c) above. ([13])

In regard to oxen, 61 were government owned and 8,128 were hired. Although Gentle refers to lung-sickness among the transport cattle, the few government oxen remained free.

([13]) Rutherford, C. (*Natal Govt. Gazette*, Jan., 1886. Govt. Notice 44 of 1886) states that " 50% of the horses, from wearing nose bags every night, suffered from ' cold ', some so badly as to be unfit for work for a few days".

Duck makes the following recommendations :—

(i) The Inspecting Veterinary Surgeon to be provided with a clerk;
(ii) Veterinary Surgeons to inspect slaughter cattle;
(iii) Clerical work to be reduced; *e.g.* Duck discontinued the Record of Treatment;
(iv) Veterinary Stores to be supplied by the A.V.D.
(v) Farriers to be more under veterinary control; and
(vi) Rearrangements of field chests and small "chests of prepared medicines to be provided for detached farriers."

Fig. 5.

ALEXANDER HENDERSON GENTLE, M.R.C.V.S * (1851-1936).

Gentle joined the Cape M.R. in London and arrived at Capetown on 27th April, 1881, He left shortly afterwards and later was one of the three volunteer veterinary surgeons attached to the A.V.D. for the Bechuanaland Expedition (1884-85). (*Vet Rec.*, 30-5-36.)

It is presumed that Duck, as S.V.S. of a command abroad, received "charge pay" as the total number of animals exceeded 1,500. A Royal Warrant authorising this was issued in April, 1881 (Smith, 1927, p. 174).

No award was issued for the Expedition (letter 25-6-1930 from Major Hare, R.A.V.C.).

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* For Obituary Notice see *Vet. Rec.* 23-5-36.

1885. As Smith (1927) makes but little reference to salaries of Army Veterinary Surgeons, it is advisable to tabulate the information as extracted from a contribution by a German veterinarian, August Zündel from Strasburg, to a discussion on "Die Reform des Militär-Veterinärwesens" at the Fifth Conference of members of the German Veterinary Council at Leipzig on March 30th to 31st, 1885 (14). Zündel not only gives details concerning the salaries of the various grades of veterinary surgeons in several European armies, but also gives information concerning the establishments. With regard to the British Army the establishment is said to have been : 2 Principal Veterinary Surgeons, 7 Inspecting Veterinary Surgeons, 40 first class Veterinary Surgeons, and 88 Veterinary Surgeons.

The salaries (15) vary according to station, whether in Europe or in India, as follows :—

Rank. (16)	*Europe.*	*India.*
P.V.S.	£860	£1,400
Inspecting V.S.	£440—520	£1,200
1st Class V.S.	£280—438	£640—800
V.S.	£182—264	£440—480.

Zündel emphasises that while the status was similar to that of combatant officers, the salary was "um ⅓ höher". No information is available as to South Africa.

In 1885 there appeared in the *Cape Times* (August 19th and 20th) an article on "Horse-sickness in South Africa" by C. Rutherford, M.R.C.V.S., A.V.D., Inniskilling Dragoons. Having been stationed in Zululand in 1883–84 and taken part in the Warren Expedition, he had obtained a good insight into South African veterinary conditions. He held to the firmly established belief that anthrax and horse-sickness were one, but differentiated clinically the disease of which he had more experience, *viz.* horse-sickness. This he represents as having (a) acute, (b) "dikkop", (c) bluetongue, and (d) bilious forms. His concluding sentence is : "I have no doubt that protective inoculation with cultivated virus, specially prepared for the country and breed of horses, will be the course pursued in South Africa . . .". The paper was later "published for general information" as Government Notice 44/1886, *Natal Government Gazette,* January, 1886.

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(14) This publication was presented to the Library, S.A.V.M.A., by Otto Henning (1865—1933), formerly of the German S.W. African Veterinary Service.

(15) According to Smith (1927, p. 165) "a new entrant received £250 a year exclusive of allowances" as far back as 1878. The scale given above was probably an old one.

(16) The ranks shown here are relative, remaining so until 1891, when they became compound, e.g. Veterinary-colonel, etc. In 1903 non-compound rank was conferred.

In 1885 admission to the A.V.D. and examination of officers for promotion, hitherto left entirely to the P.V.S., became governed by regulations.

1886. The P.V.S. War Office (Fleming, G.) for the first time makes statistical reference ([17]) in his *Annual Report for* 1886 (dated March 31st, 1887) to South Africa. The returns apply to horses and mules in Natal. Regarding horse-sickness, he states that " during the South African summer of 1885 - 86 there was a very heavy mortality among the horses of the Colonists and the Government horses and mules did not escape, 71 having been admitted for treatment; of these only 10 recovered, 61 having been returned as dead. In view of the ravages made by this disease almost annually . . . sanction was given for one of the most intelligent and enthusiastic officers of the Veterinary Department, Mr. Nunn, to be sent to South Africa to investigate the nature of the malady . . . After undergoing a course of special training in the pathological laboratory of the Brown Institution, London, and in that of the Cambridge University, as well as visiting M. Pasteur's laboratory in Paris, . . . Mr. Nunn left for South Africa in December (1886) . . . so as to arrive there about the time when the horse-sickness begins to show itself . . . Mr. Nunn has also been directed to report upon other contagious diseases in South Africa, a task for which he is particularly fitted from his long experience as Inspector under the Contagious Diseases (Animals) Act in the Punjab. He is also to enquire into the question of horse supply . . . with the view of ascertaining its resources, and in what way improvement (if any is necessary) may be best effected, for the requirements of India or even of this country. Mr. Nunn, having been for some time Assistant Superintendent of horse breeding operations in Bengal, should be well qualified to give an opinion as to the kind of horses required for India."

Fleming also reports " a form of intestinal derangement caused by the horses, chiefly those of the Inniskilling Dragoons, eating sand ". Of 61 cases 7 died. This could have occurred either at Pinetown or Fort Napier, for it was not until May that the 6th Dragoons proceeded to the latter station.

In a paragraph concerning the Army Veterinary School, Aldershot, it is mentioned that specially trained farriers accompanied the expedition to Bechuanaland ([18]).

([17]) See Table IV.

([18]) Wiltshire furnished a further Report on Horse-sickness in 1886, this being published under Government Notice 280/1886 in the *Natal Government Gazette* of July 6th, 1886.

1887. The *Annual Report for* 1887 again refers to Natal, as it " is the only Colony in which we have mounted troops ". As before, horse-sickness is the chief menace, there being 174 admissions out of 228, and of the former 122 were fatal. The Remount Department

SOUTH AFRICA.

The Veterinary Returns from South Africa are limited to one Colony, Natal. The animals employed there by Government, and which are shown in these Returns, were horses and mules. These were in number 732, of which 75·95 per cent. were admitted to treatment; of these 12·43 per cent. died and 1·77 was destroyed, making a total mortality of 14·2 per cent.

The most notable diseases were anthrax and a form of intestinal derangement caused by the horses, chiefly those of the Inniskilling Dragoons, eating sand. Of the latter there were 61 cases, of which 7 died. With regard to the disease named anthrax, this is a most serious scourge in South Africa, and is usually known as the Horse-sickness (the " paard ziekte " of the Boers). Its ravages among horses have been recorded from time to time since that part of Africa was inhabited by Europeans, and in some of the most fertile portions of the Cape and Natal Colonies it is impossible to keep horses during the summer season—nearly all perishing from this plague. During the South African summer of 1885--86, there was a very heavy mortality among the horses of the Colonists, and the Government horses and mules did not escape, 71 having been admitted to treatment; of these only 10 recovered, 61 having been returned as died.

In view of the ravages made by this disease almost annually, of its sinister effects on the prosperity of our Colonists in that part of the world, and of the losses the Imperial Government sustain by the death of the horses and mules in that country, sanction was given for one of the most intelligent and enthusiastic Officers of the Veterinary Department, Mr. Nunn, to be sent to South Africa to investigate the nature of the malady, and to attempt its suppression by the adoption of the method of protective inoculation with attenuated or modified virus, introduced by Pasteur and others so successfully in the case of European anthrax. After undergoing a course of special training in the pathological laboratory of the Brown Institution, London, and in that of the Cambridge University, as well as visiting M. Pasteur's laboratory in Paris, in order to acquire a knowledge of certain technical matters, Mr Nunn left for South Africa in December last, so as to arrive there about the time when the horse-sickness begins to show itself. I am anxiously hoping that his investigations may not only fix the exact nature of the disease, but also admit of its prevention by a simple, inexpensive, and safe method of inoculation, and thus confer a great boon on the Colonists. Mr. Nunn has also been directed to report upon other contagious diseases of animals in South Africa, a task for which he is particularly fitted from his long experience as Inspector under the Contagious Diseases (Animals) Act in the Punjab. He is also to inquire into the question of horse supply in that country, with the view of ascertaining its resources, and in what way improvement (if any is necessary) may be best effected, for the requirements of India, or even of this country Mr. Nunn, having been for some time Assistant-Superintendent of Horse Breeding Operations in Bengal, should be well qualified to give an opinion as to the kind of horses required for India. (Rpt AVD for 1886)

Fig. 6.

THE *ANNUAL REPORT OF THE A.V.D. FOR* 1886 WAS THE FIRST TO REFER TO SOUTH AFRICA.

purchased 104 horses at an average price of £33 5s. 4d. Twenty-five horses were bought for the Royal Artillery at an average price of £25

12s. 2d. Forty-nine horses were cast and sold, the average price being £6 3s. 9d.

Reference is made to an Interim Report furnished by Mr. Nunn, who indicates that horse-sickness is not anthrax; also to the fact that one of the subjects discussed at a meeting of the Army Veterinary Medical Association, Aldershot, was " Anthrax in South Africa ", this no doubt actually being horse-sickness.

In 1887 Veterinary Surgeon F. F. Crawford was stationed in Pietermaritzburg and while there distinguished himself as a cricketer.

In the *Natal Government Gazette* of April 26th, 1887, under Government Notice 217/1887 Wiltshire describes an outbreak of anthrax among stock, chiefly sheep, on a farm near Greytown. He mentions that Mr. Nunn, A.V.D., prepared specimens from material furnished by Wiltshire " and also inoculated a guinea pig with some of the blood from the sheep —which died about 24 hours after, showing the characteristic lesions of Anthrax ".

In 1887 was created the Army Remount Department, cavalry regiments prior to this being responsible for the purchase of their own remounts—(letter of June 14th, 1934, from Lieut.-Col. S. L. Slocock, M.R.C.V.S., to Dr. F. Bullock). The A.V.D. later (1899) fell under the control of the Director, Army Remount Department.

In 1887 F. Duck presented before the Army Veterinary Medical Association, Aldershot (21-1-87), a paper entitled " The Working of the A.V.D. in the Field ". It was a valuable contribution based on his experiences in South Africa and later appeared in the *Vet Jl.* XXIV, p. 202.

1888. The chief event of the year 1888 was the Zulu Rebellion and its suppression, for which Imperial troops were actively employed.

(ii) *Zulu Rebellion May–November,* 1888.

The arbitrary partition of Zululand by Sir Garnet Wolseley after the Zulu War, 1879 ([19]), did not result in peace. There was soon inter-tribal strife among the thirteen chiefs and the reinstatement early in 1883 of Cetshwayo as King led to no improvement. His son Dinuzulu, who succeeded him on his death (February 8th, 1884), made matters worse by relying on the support of Dutch burghers, from the adjoining South African Republic, to defeat his chief adversary, Usibebu, at Etshaneni on June 5th, 1884. The immediate result was that the Dutch were compensated with farms which together formed what was known as the New South African Republic ([20]). The Zulus, being dissatisfied, appealed to the British Government for redress, with the ultimate result that Zululand was annexed in May, 1887! The dissatisfaction following

([19]) For a veterinary history see *Vet. Rec.*, Jan. 5th, 1935.

([20]) This became definitely incorporated in the South African Republic in 1888.

this step developed into open rebellion a year later, when the threatening attitude of Dinuzulu necessitated the employment of Imperial troops.

During 1887 detachments of the 6th Inniskilling Dragoons had been sent to Zululand, but on the outbreak of hostilities in May, 1888, the whole regiment was transferred from Pietermaritzburg.

The first occasion the 6th Dragoons were needed was at the beginning of May when, in order " to support the Magistrate's authority " in the attempt to arrest certain rebels, " a squadron . . . under Colonel (afterwards Sir Richard) Martin was advanced to a point north of the Ivuna Stream, and about seven miles from the (Nongoma) Magistracy " —(Gibson, 1911, p. 301). Later in May the rebels, with reinforcements, took up a position at Ceza Mountain and on June 2nd another attempt was made to effect the arrests. " The regular troops were commanded by Capt. Pennefather of the 6th Dragoons, and consisted of 3 officers and 81 N.C.Os. and men of that regiment and 43 and 35 N.C.Os. and men respectively of the Royal Irish Fusiliers ([21]) and the 1st North Staffordshire Regiment, as M.I. . . . The Zululand Police under Mansel numbered 66 men . . . and the whole force, exclusive of native levies, numbered about 208 " (p. 304). An engagement followed during which the Government force withdrew. " The Usutu (followers of Royal House) felt that they had been victorious and pursued the retreating troops . . . but were prevented by Capt. Pennefather from getting within effective distance " (p. 305). The whole force returned to Inkonjeni next day. Twenty days later the rebels not only defeated Usibebu, who was actively supporting the Government, but captured " the horses of the officials and police and all cattle that happened to be at the (Nongoma) fort " (p. 309).

In the meantime another focus of rebels collected at Hlopekulu Mountain under Chief Tshingana, but as this centre " was within striking distance of the military stationed at Inkonjeni " (p. 312) an attack was arranged on July 2nd. This was undertaken by about 2,000 men including Imperial troops, police and native levies and was successful, in fact was " the determining event of the disturbances " (p. 314).

Just at this time (July 25th, 1888) the border difficulty with the South African Republic was settled and the authorities were better able to concentrate against the rebels. " The conduct of operations was now in the hands of the officer commanding the regular troops ([22]), it having been found that the local police were unequal to the task of restoring order " (p. 314).

([21]) It would appear that this should be Royal Inniskilling Fusiliers.

([22]) According to Gibson (p. 315) the Imperial troops about July "numbered 86 officers and 2,163 N.C.O's. and men, 816 of whom were mounted ". In addition there were native levies.

Vigorous operations and the establishment of " military posts . . . in various parts of the territory " (p. 316) soon led to pacification. Dinuzulu, however, did not surrender until November 15th at Pietermaritzburg, after which he was exiled to St. Helena.

From a veterinary point of view the fact that the campaign had taken place during the winter was a great advantage, in that losses from horse-

Fig. 7.

P.V.S. JAMES LAMBERT (1835–1905).

Taken from *Vet. Jl.*, May, 1905.

sickness and nagana were reduced to a minimum. Of particular interest was the new system of shoeing, which " though not perfected . . . had already been tried at Suakin, and in Bechuanaland . . . and the reports upon it were highly favourable ". (*The Ann. Statist. Gen. Rpt. A.V.D.*

1888, p. 13.) According to the O.C. Inniskilling Dragoons, " The Regiment having been on Field Service in Zululand during the last six months, the new regulations as regards cold-shoeing could not be carried out in every particular; though, as far as was practicable and possible, every effort has been made to act on and carry out the instructions issued. Shoeing the horses with cold-shoes has been done most fully, and I think very successfully . . . In a standing camp there can be but little difficulty about the details of the system being carried out in their entirety. The material of the shoes is excellent, and if they are slightly heated by gentle blows from the hammer, they can be opened out or otherwise altered in shape, so as to fit the foot without injuring the shoe; though if they are opened out before being treated as above, they nearly always break at the toe. This arises from want of care on the part of the farriers, and will always be the case unless caution is exercised . . . The nails, though nearly equal to those issued some time ago, are, as a rule, of good quality, well made, and carefully finished.

" Owing to the regiment being in detachments, and constantly on the move from one place to the other, the horses were all shod with the ready-made shoes and nails supplied for the cold-shoeing, none being shod with the hand-made shoes and nails, as it was an impossibility, under the circumstances, to make them . . . With reference to paragraph 7 of the Army Order referred to, the shoes and nails are durable and last well.

" The conditions of service under which these shoes were used were exceptional, as the troops were on Field Service during the whole period. The shoes, therefore, were tried under severe conditions, and I consider that they bore the trial satisfactorily ". ([23])

It has already been seen that observations regarding shoeing were made during the Bechuanaland Expedition, but " the new organisation of the Shoeing Department " was only introduced on April 1st, 1888.

Other details from the P.V.S.'s *Annual Report for* 1888 may be quoted as follows : " In the Colony of Natal, at the end of 1887, there were 633 troop horses and mules, and 77 chargers; at the termination of 1888, there were 711 horses and mules and 79 chargers.

The admissions to treatment were 701, or 104.94% of the total average strength. In 1887 it was 94.47%. The increase was due to operations in the field, which greatly augmented the surgical diseases and injuries, there being 175 sore-backs and 14 girth-galls, as well as 57 cases of contusion and 40 contused, 11 incised, 10 punctured, 8 lacerated, and 3 gun-shot wounds, with 44 cases of sprain and 19 of laminitis. There

([23]) P.V.S. Fleming, who includes Col. Martin's report in his Annual Report, adds that the Swiss Army adopted " our system of shoeing, pattern of shoes, and appliance for enabling horses to travel on ice and slippery roads ".

were 56 cases of the fatal disease peculiar to Africa ("known as horse-sickness"), 21 cases of debility (asthenia), 10 of rheumatism, and 2 of exhaustion.

Of the 56 cases of "horse-sickness" admitted, 54 died. A peculiar feature of this disease during the year in question was its prevalence for 11 months : it is usually seen only in the South African summer; whereas in 1888 the mortality was greatest in August and September—an extraordinary occurrence, since the disease is most frequent in the early months of the year. The following table shows the prevalence of the malady among Army animals and those of the Colonial transport :—

Month.	Imperial Army. Deaths.	Colonial Transport. Deaths.
February	2	14
March	3	13
April	5	17
May	4	15
June	5	5
July	—	8
August	11	17
September	20	14
October	1	4
November	1	2
December	1	—
Total	52	109

Veterinary officers stationed at Eshowe included R. W. Raymond (1888–93), Wilkinson (1893), E. H. Kelly (1896), and J. T. Coley (1897).

With regard to horse-sickness, veterinary opinion ([24]) in the early eighties, as has been seen, inclined to the view that horse-sickness and anthrax were one and the same disease. Nunn furnished three reports ([25]), dated September 14th, 1887, June 13th, and October 16th, 1888. These, together with a letter of transmittal from P.V.S. Fleming (dated October 27th, 1888) to General Viscount Wolseley, Adjutant-General of the Forces, were published as *Bluebook* 116/*Cape*/35 in December, 1888. In the first Report Nunn showed that he had failed to substantiate bacteriologically and by experimental transmission the view that horse-sickness was anthrax. He apparently succeeded in transmitting horse-sickness from a natural case to mule 2721, but states that the mule "contracted horse-sickness naturally and that no contagion existed in the

([24]) Wiltshire (1878), Lambert (1881), and Hutcheon (1881). Hutcheon (1882) believed that heartwater was of the nature of anthrax.

([25]) In Library S.A.V.M.A. at Onderstepoort.

inoculations ". As the period of incubation is 4—10 days, it is also quite possible that the disease was set up artificially (date of inoculation March 25th—date of death April 4th). In the second Report Nunn refers to " further experiments . . . in Pietermaritzburg . . . that . . . merely confirm my previous opinion, that horse-sickness is not anthrax . . .". In the final Report he describes the four clinical forms of horse-sickness already distinguished by Rutherford.

Nunn's *Report on the Horse-supply of South Africa* is not obtainable in this country ([26]), even from the Archives at Cape Town; but thanks to Hutcheon (1901) his views are available. Nunn described the Cape

Fig. 8.

S.V.S. FRANCIS DUCK (1845–1934).

Taken from *Smith's History of the R.A.V.C.*

horse in the following terms : " small and stunted in growth, deficient in bone, pinned in at the elbows, good shoulders and forehead, narrow chest,

([26]) Another publication that is unobtainable is V.S. B. L. Glover's pamphlet " Suggestions for the General Management of Horses and Mules while on Field Service in Natal and the neighbouring countries, with notes concerning their more common ailments " (1878). Dr. Bullock has also been unsuccessful in London. See, however, *Vet. Jl.*, Nov., 1888 (XXVII, p. 335).

very badly coupled and ribbed up, with bad drooping quarters, badly developed muscles of the croup and thighs, split up behind with crooked hind legs, the hocks being very far back. In fact the South African horse is, although small, good before and bad behind the saddle". The Report was addressed to the Deputy Adjutant-General, Capetown, 1888.

"In 1888 it was decided that a tour of foreign service was to be six years instead of five . . ." (Smith, 1927, p. 185). In 1899 this was reduced to 5 years (*Veterinarian*, 1899, p. 522).

1889. In 1889 S.V.S. Rowe gave evidence before the Commission appointed in August, 1889, by the Natal Government to enquire into the best means for improving the breed of cattle and horses in Natal. The members of the commission were Messrs. T. K. Murray, C. A. S. Yonge, J. Baynes, P. R. Botha, and G. Turner. They presented a report, one of the most valuable in the history of Natal, at the end of February, 1890 ([27]). Among those who furnished information were Lieut.-Col. E. G. Pennefather, 6th Dragoons, S. Wiltshire, C.V.S., H. D. Winter ([28]), C. B. Lloyd, D. Hutcheon, and Col. R. Rowe. Among the recommendations of the Commission were the establishment of a Board of Agriculture and the increase of the Veterinary Department. It was suggested "that the Government communicate with the President R.C.V.S., London, requesting his co-operation in obtaining the services of at least four competent gentlemen to act as additional Veterinary Surgeons in this Colony . . . The salaries should not exceed £300 *p.a.* in addition to private practice at a fixed tariff of charges. Each of these gentlemen should, moreover, be microscopists . . . ." The additional Veterinary Surgeons were not appointed until Rinderpest threatened in 1896.

1890. In 1890, on the retirement of P.V.S. Fleming, a departmental scandal occurred, his successor being J. D. Lambert, the next senior, who should actually have retired a day before Fleming. Owing to official jobbery, however, a Royal Warrant was published in June (dated May 16th, 1890) authorising the extension of Lambert's service until he reached the age of 60 instead of 55 ([29])! His appointment suited the authorities; for as long as *he* was satisfied he was prepared "to live in peace with all branches and all persons in authority" (Smith, 1927, p. 190).

1891. In 1891 the designation of the P.V.S. was altered to Director General and compound rank (*i.e.* prefix veterinary) introduced, Lambert being Veterinary-Colonel.

([27]) *Report of the Commission to Enquire into the best means . . . for improving the Breed of Cattle and Horses in Natal.* Wm. Watson, Pietermaritzburg, 1890.

([28]) From 1899 to 1903 Minister of Agriculture, Natal.

([29]) Smith who gives this information (p. 190) states that Lambert's dates of birth and death were 1835 and 1902 respectively. The *Vet. Jl.* (May, 1905) gives the date of his death as 1905!

In 1892 Fleming, who was a prodigious worker even in retire-
1892. ment, " urged the trial of mallein . . . on the strength of Nocard's report " (Smith, p. 178). It would appear that it was Wiltshire who first used mallein in Natal. In February, 1894, he returned from England on furlough and in the Annual Report for 1894–1895 he refers to his having employed mallein " with most satisfactory results ". He mentions that, when early in 1894 he was ill, Major Berne attended to some of his cases ([30]).

In 1892 there visited South Africa that remarkable man, Capt. M. Horace Hayes, F.R.C.V.S., who, accompanied by his plucky wife, gave exhibitions in horsemanship throughout the country. Although a veterinary surgeon, qualifying at the New Edinburgh Veterinary College in 1883, he did not serve in the A.V.D., but owing to his literary talent, so unusual

Fig. 9.
S.V.S. FRANK F. CRAWFORD
(1850–1900).

among veterinarians, he deserves to be remembered. He commenced his career in India in the Royal Artillery and he explains in the preface to the 6th edition (1903) of his *Veterinary Notes for Horse Owners* that his experience as a horse-master impressed on him the need for veterinary study. After qualifying he retired from the Army and had a varied experience as army crammer, veterinary surgeon, horse-dealer, showman, and author. His works, in addition to the book mentioned above, include *Points of the Horse, Illustrated Horse Breaking, Riding and Hunting, Stable Management and Exercise, Horses on Board Ship, Among Horses in Russia, Among Horses in South Africa,* and *Among Men and Horses.*

([30]) In December, 1895, Col. D. Bruce published his *Preliminary Report on Nagana in Zululand.*

He also translated Friedberger and Frohner's *Veterinary Pathology* and *Breaking and Riding* by James Fillis. He contributed to current literature and once wrote on horse-sickness (*Vet. Jl.* XLII, p. 23, 1896). His death took place at Southsea in 1904 (*Vet. Rec.*, Sept. 10th, 1904).

(iii) *The Matabeleland Rebellion, March–October,* 1896.

1896. The only direct evidence that the A.V.D. was represented in the Matabaleland Campaign of 1896 is Smith's (1927, p. 258) mention of Veterinary Surgeon A. England. *The Annual Statistical and General Report of the A.V.S. for the year ended 31st March,* 1897, merely explains that the high percentage of admissions and mortality of army horses and mules in South Africa was due in a great measure to active service in Matabeleland. Of a strength of 897 troop horses and mules and 35 chargers, the admissions were 848 or 91.18% and the mortality 256 or 27.52%. Seventy-one deaths were due to exhaustion owing to lack of grazing. One case of glanders occurred on October 30th, 1896. The report is signed by Vet.-Col. F. Duck, Director-General, on July 7th, 1897, and addressed to Sir Redvers Buller.

As Veterinary Surgeon England, A.V.D., was the first Army Veterinary Surgeon to enter Southern Rhodesia, it is convenient to consider briefly other events of a veterinary nature in Rhodesia about this time.

In 1890 Mashonaland (now the eastern half of Southern Rhodesia) was occupied by a pioneer expedition under Major Frank Johnson ([31]). The column, consisting of 187 Europeans and 150 natives, finally camped at Fort Salisbury on September 12th, thereafter being disbanded. According to the Medal Roll of the Department of Defence, Southern Rhodesia Forces (Minute C.S. 518.20 of July 14th, 1934) E. O'Connell Farrell accompanied the expedition as Vety.-Lieut.; in fact he is to be seen (on the extreme left, top row) in a photograph of a group of officers of the 1890 Pioneers in the Special Pioneer Number (Sept. 12th, 1930) of the *Rhodesia Herald*. On being requested to trace Farrell in the Register of Veterinary Surgeons ([32]), Dr. Fred. Bullock replied (letter R.583 of Sept. 11th, 1934) that " no trace " could be found of the name.

In regard to the Matabele War of 1893 and the subsequent annexation of the country (now the western half of Southern Rhodesia), no veterinary surgeon appears to have been present.

In 1896 occurred the Matabeleland Rebellion and before referring to the veterinary surgeons participating a résumé will be given of the campaign.

([31]) See *Guide to Rhodesia,* 2nd Edition, Davis & Co., Buluwayo, 1924.

([32]) The *Register* refers only to members and other veterinarians registered by the R.C.V.S.

Of the several causes responsible for the rebellion, the one which may be termed the veterinary factor was probably the most important. Sykes (1897, p. 7) summarises the position as follows : " After the occupation of the country in 1893 the Chartered Company [British South Africa Company], by right of conquest, claimed all the King's [Lobengula's] cattle, which virtually meant all the cattle in the country; for Lobengula, as absolute despot, only allowed his subjects the right of keeping cattle on sufferance, hence in a way they were all royal cattle. The number at the time was estimated at a quarter of a million head ([33]). Confiscation by the authorities accordingly took place, but so dissatisfied were the natives that eventually it was agreed that the Government should retain 45% and the remainder should be branded N.C. (native cattle) and be returned to the natives (p. 8). Again, just before the outbreak of the Rebellion, Rinderpest made its appearance, being officially reported at Bulawayo on March 3rd. With the destruction of infected herds by the administration, in order to prevent extension of the disease, the desperation of the natives can be easily imagined. Matters came to a head on March 20th, at which time many of the police were away, having taken part in the Jameson Raid.

The campaign may best be divided into two phases, (a) from March to May when defensive measures were employed and (b) from June to October when the rebels were vigorously attacked and finally peace restored.

(a) On the outbreak of hostilities, the European settlers concentrated for safety at three centres, Bulawayo, Gwelo, and Belingwe. The several corps ([34]) which had been formed in Bulawayo were " organised . . . under the title of the Bulawayo Field Force", which functioned until July 4th, when sufficient reinforcements had arrived from the south. The Imperial Government, realising the sorry plight of the colonists, especially with the collapse of transport due to rinderpest, not only permitted the organisation, under Imperial officers of the Matabeleland Relief Force, but also despatched regular troops, principally a detachment of No. 10 Mountain Battery from Natal and later two squadrons of the 7th Hussars, and M.I. of the Yorks and Lancaster Regiment.

From a veterinary point of view much information is available concerning the Matabeleland Relief Force, 750 strong and commanded by Lt.-Col. Herbert Plumer. Sykes (1897) records that " recruiting was commenced at Kimberley and Mafeking on the 6th April, and with the exception of 150 men and 163 horses raised at Johannesburg, all recruiting

([33]) Sinclair (1922) suggests that the number of cattle which died from rinderpest was " upwards of half a million ". In March, 1898, approximately 25,000 remained.

([34]) The Rhodesia Horse was disbanded; serving in this was F. M. Hill, M.R.C.V.S. The various corps included Grey's Scouts, Afrikander Corps, and Dawson's Scouts.

was done at those two places " (p. 56). Mafeking, the base of operations, was then the terminus of the railway, nearly 600 miles from the objective, Bulawayo.

Sykes adds : " The horses . . . were obtained from the Free State and Kimberley districts. They were . . . veld-fed animals averaging between 14.2 and 15 hands high . . . the first horse was bought on April 10th and the last was delivered at Mafeking on April 25th, so that 1,100 horses . . . were inspected, purchased and delivered in the remarkably

Fig. 10.
S.V.S. RICHARD ROWE (1844–1930).
Photo taken during the Great War.

short space of fifteen days". The casualties during the march were " only 88 out of the 1,100 ". Capt. Beresford of the 7th Hussars ([35]) purchased the horses at an average price of £18 per head, but whether a veterinary surgeon inspected the animals is not known. The transport for the column consisted of 50 wagons and 700 mules ([36]).

([35]) Baden-Powell (1897) states that in June, 1896, Capt. F. W. E. de Moleyns, adjutant 4th Hussars, was O.C. Remount Department.

([36]) A corps of coloured men (Cape Corps), which marched from Mafeking to Bulawayo in 30 days, had donkey transport. Owing to debiltiy only 40 animals out of 90 reached their destination. (Sykes, p. 84.)

" The saddles were not all of the same pattern, some . . . being cheap inferior articles, bad both for horse and rider. By far the best sort were the old B.B.P. [Bechuanaland Border Police] saddles . . . " (Sykes, p. 63).

(b) With the arrival of the Matabeleland Relief Force in Bulawayo at the end of May (as well as another force from Salisbury), the position so improved that the Bulawayo Field Force was disbanded on July 4th. Command of the military operations from the 3rd June was assumed by General Sir Frederick Carrington, who by organising mobile columns to attack the rebels soon cleared the country, except for the Matoppo Hills. It was then that Cecil Rhodes, accompanied by three unarmed companions, proceeded into the rebel camp and by his patience and reassurance gained their confidence. Peace was formally restored on October 13th.

The 7th Hussars did not reach Gwelo from the south until September 4th. On the march north, Monogola's stronghold had been captured with slight loss, and in October Ndema's and Wedza's villages were destroyed. At the end of November the regiment marched to Bulawayo where they " went into standing camp ". In June, 1897, they proceeded to Mashonaland to deal with the latter stages of a rebellion there ([37]).

With regard to the veterinarians who took part in the Matabeleland Rebellion, the Medal Roll of the Department of Defence, Southern Rhodesia Forces, again records the participation of men who cannot be traced by Dr. F. Bullock in the *Register of Veterinary Surgeons*. These are Vety.-Lieut. T. G. Wignall (Rhodesia Horse Volunteers) and Vety.-Surgeon T. L. Knapman (Salisbury Field Force).

V.S. A. England, A.V.D., no doubt accompanied the 7th Hussars but no report of his is available.

Another veterinarian who was present was Charles Elias Gray, who became Chief Veterinary Surgeon, Southern Rhodesia, and later transferred to the service of the Transvaal Government. When rinderpest broke out he was stationed at Macloutsie as a telegraph operator (there being no state veterinarian), but his duties were changed temporarily. He later joined "H" troop, Bulawayo Field Force, "raised by Selous . . . and served with it throughout the rebellion, making himself generally useful . . ." (letter from C. E. Gray). The Medal Register duly records that Corporal C. E. Gray received the 1896 Rhodesia Medal.

For the Mashonaland Rebellion (June-November, 1896) there does not appear to have been an A.V.D. officer; in fact, Lieut.-Col. Alderson (1898, p. 21) in referring to " one of the horses taken on board at Durban" states that it developed " symptoms of farcy . . ." and as " we had no veterinary surgeon with us, and rather than run the risk of

([37]) This brief outline is based on Sykes (1897), *Guide to Rhodesia* (1924) and Howard Hensman's *History of Rhodesia* (1900).

infection in the crowded ship (*en route* to Beira), I had the horse shot at once".

Provision was actually made for a veterinary surgeon to accompany the Mounted Infantry (which Alderson later took to Mashonaland) to South Africa, as shown in Appendix A of Alderson's account of the Rebellion (p. 265). The officer is stated to have been Vet.-Maj. G. D. Whitefield, but according to a list of A.V.D. officers ([38]), it was *Whitfield,* who was in South Africa from May 2nd, 1896, to June 23rd, 1897. Smith (in a private letter) also mentioned that Whitfield did *not* proceed to Mashonaland. The Imperial troops left Mashonaland at the end of December, but owing to a recrudescence of rebellion the following winter, Imperial troops, this time the 7th Hussars, were again summoned. In July they took part in the successful operations against Mashingombi and returned to Natal via Beira in October. Although the A.V.D. was not represented in Mashonaland, the veterinary profession was, in that G. W. Lee ([39]), recently appointed under the B.S.A. Co., took part.

1897.

In June of this year Vet.-Col. Lambert completed his seven years as Director-General and was succeeded by Francis Duck, who had seen much South African war service. Duck's greatest trial, however, was still to come ([40]).

Early in the same year there was a rising in British Bechuanaland, *i.e.* the southern portion of the territory annexed as a result of the Warren Expedition and which in 1895 was transferred to the Cape Colony. The first manifestation of rebellion was in December, 1896, when the stringent regulations enforced by the Cape Administration concerning rinderpest were carried out (Williams, 1909, p. 48).

As Imperial troops were not used in the campaign and the A.V.D. was unrepresented, no more need be said about the rebellion, except that two civil veterinary surgeons, G. N. Tomlinson (Cape M.R.) and W. Robertson (Diamond Fields Horse), were present.

In August, 1897, the International Rinderpest Congress ([41]) was held at Pretoria, and of interest to the A.V.D. was the fact that Vet.-Capt. A. J. Haslam, who had been seconded as transport officer on the Uganda railway, was chosen as a representative of the Natal Government.

([38]) Kindly furnished by Major G. E. Oxspring, R.A.V.C.

([39]) His letter of Dec. 12th, 1929. According to the Medal Roll of the Dept. of Defence, S. Rhodesia, "two persons named T. W. Lee, served in the Matabeleland Relief Force in . . . . 1896 . . . ."; but there is no trace of a G. W. Lee. In the *Jl. S.A.V.M.A.* IV (1) (March, 1933) Lee is incorrectly stated as having taken part in the 1896 Mashona rebellion.

([40]) Duck was honorary associate (1928—1934) of the S.A. Vet. Med. Assn.

([41]) The Congress was held from 2nd to 13th August, 1897. The Report was published at the State Printing Works, 1898.

He had stopped at Natal on his way to East Africa in order to purchase mules, and with C. B. Lloyd, the Commissioner for Agriculture, attended the Pretoria Conference. Soon after his arrival in what is now Kenya, his services were needed inland on account of rinderpest; and while "superintending preventive treatment . . ." he entered Kikuyu territory, where he was murdered on July 17th, 1898. Several issues of the *Vet. Journal* (XLVII, pp. 76 and 195) and *Vet. Rec.* (Aug. 27th, 1898, Sept. 17th and 24th, 1898) contain references to this promising officer, whom Smith (1927, p. 200) describes as "so well equipped educationally" ([42]). Robert J. Stordy was actually the pioneer of the profession in Kenya, but Haslam followed him closely. Haslam was born in 1863 and possessed in addition to his veterinary qualification the medical degrees of M.D., C.M. He is mentioned by Paterson in *The Man-eaters of Tsavo* (MacMillan & Co., Ltd., 1930).

1898. In 1898 a serious outbreak of osteoporosis "occurred at the Military Camp at Wynberg, Capetown . . . amongst a troop of horses which came from the Argentine." Details of Capt. Lane's report concerning the disease were published by Hutcheon (1905) in the April issue of the *Cape Agricultural Journal*. See also Lane's report in the *Rept. of the A.V.D. for year ended* 31-3-05.

On June 23rd, Lieut.-Col. Iles Matthews arrived in South Africa with instructions to investigate the cause of past mortality and to suggest measures for prevention in the future. He visited each garrison in the country and submitted an *Interim Report* in November, 1898. In this he set down the causes of previous mortality as follow: (1) Exposure to tropical climate without stables, sometimes without nose-bags ([43]); (2) watering and working animals in the early morning before dispersal of mist; (3) horses breaking loose at night and wandering into unhealthy localities; (4) exhauted condition produced by the Natal climate during January to April; (5) use of freshly cut grass for bedding; (6) scarcity of water at Pietermaritzburg and impurity of supply at Ladysmith; (7) incidence of horse-sickness, the two M.I. companies (Leicester Regiment and Royal Dublin Fusiliers) at Pietermaritzburg losing heavily, one 92 out of 123 cobs and the other 83 out of 123; and (8) failure to become acclimatised, since nearly all horses had been imported the previous spring.

Matthews refers to want of proper facilities for treatment, infirmaries, and trained attendants. He was promoted P.V.O., South Africa, on October 9th, 1899 (*Veterinarian*, 1899, p. 845).

([42]) Col. Stordy writes (12-6-35) that Haslam was buried at Fort Smith. See Fig. 36.

([43]) Since Edington had shown in 1892 that horse-sickness was communicable by sub-inoculation of the blood, it is surprising that such faith was placed in nose-bags. Of the ponies of the M.I. on picket lines 53% died, while of the Cavalry horses in stables only 2% died. (*Vet. Rec.*, 16-7-98, p. 43.)

An unusual occurrence experienced in 1899 and one presumably
1899. not reflected in Table IV was the loss at sea of some horses belonging to the 9th Lancers. The regiment embarked at Bombay for Cape Town on 24th and 25th September aboard the *Nairung, Nowshera* and *Wardha*. When a fortnight later Durban was reached, stores were picked up and the voyage continued to Cape Town. *En route* a terrific gale was encountered and while the first two ships weathered the storm safely, the *Wardha* damaged her rudder and made for Durban. During the storm " one half of the horses " on the *Wardha* were washed overboard (letter from O.C., March 3rd, 1932).

At this point it is convenient to refer to the preparations which were being made for the Second Anglo-Boer War which broke out on October 10th, 1899. Smith (p. 3) states that " early in 1899 the unsettled condition of affairs in South Africa attracted attention, and regulations for the mobilisation of a Field Force for that country were drawn up . . . During the four years occupied in evolving the scheme for mobilising the army, the veterinary service would appear to have been lost sight of . . .". It must be remembered that while the A.V.S. was indeed departmentalised, no arrangements had been made, except in India, for hospitals or subordinate personnel. Smith adds : " Some so-called sick [horse] depôts (44) were framed on paper and placed under the Remount Service, who were thereby charged with all the duties of providing for the removal and care of the sick, while the veterinary officer's function was limited to treatment ". The feud between the Remount and the Army Veterinary Services actually led in 1898 (45) to the abolition from the Army in the Field of every Veterinary Lieut.-Colonel ! In fact, the only indication of the existence of an A.V.S. was a base depôt and advance depôt of stores, the latter under a farrier sergeant ! The P.V.O. indeed remained, but with no administrative staff to carry out his orders ! (Smith, 1927, p. 203.)

It must be emphasised that, in spite of the general attitude of the War Office to the A.V.S., the authorities early in 1899 decided upon the formation of a station veterinary hospital at Salisbury as an experimental measure, notwithstanding the objection of the Director-General A.V.S. ! " In consequence of the decision . . ., the Director-General (F. Duck) urged the formation of a subordinate veterinary staff for hospitals . . .", but before any decision was made " the storm of 1899 broke over our heads " (Smith, 1927, p. 202).

Reference has been made to the feud between the A.V.S. and the Remount Department; but there are many reasons for believing that the veterinary profession, military as well as civil, has had itself to blame

(44) In 1888 one sick horse hospital was provided for a force consisting of an Army Corps, a Cavalry Division and L. of C. troops. It was located in the rear of an army and was not only a hospital, but was to receive and issue remounts ! (Smith, 1927, p. 202).

(45) An edition of *War Establishments* was published in 1898 (Smith, 1927, p. 203).

for many of its difficulties. A complaisant attitude and ineffectual platitudinising will never lead to the advancement of a profession.

To turn now to South Africa before the outbreak of war, the veterinary arrangements made locally were associated with (a) the creation of the Natal Volunteer Veterinary Corps in July, 1899 (Curson, 1934);

Fig. 11.

FULL DRESS UNIFORM VETERINARY SURGEON *, A.V.D., 1883.

This was the first time the A.V.D. had its own uniform, dress previously (from 1861) having been distinctive only for the P.V.S. and Staff V.Ss. Smith (1927, p. 248) indeed adds that "it (in 1861) can hardly be called distinctive, being that of the Cavalry Depot."

Regimental V.Ss., previous to 1883, wore the uniform of their regiments.

The above uniform, taken from Smith's *History of the R.A.V.C.* (Plate I, p. 184), shows :—

Headdress. Cocked hat and red plume, as worn since 1861 by all V.Ss. (except in Household Cavalry), retained; but the red plume varied in length according to rank.

* Relative rank Lieutenant. Uniform for mounted duty.

TABLE III.

(c) LIST OF ARMY VETERINARIANS AFTER DEPARTMENTALISATION OF THE A.V.S.
From April, 1881 to Outbreak of Second Anglo-Boer War, 10-10-99.

The subjoined list (received from Major G. E. Oxspring, R.A.V.C.) is a continuation of a list of Army Veterinary Surgeons who served in South Africa (see footnote, (1), p. 41). As some of the officers previously mentioned again visited the country, their names are given as follows: F. Duck (1881–5), C. Rutherford (1884–5), and R. Moore (1896). W. Gladstone, D. C. Pallin, J. G. Rayment, and C. Rutherford took part in the Second Anglo-Boer War (1899–1902). It will be noticed that the number 37 is placed opposite V.S. Finlayson's name. This indicates that he was the 37th officer of the A.V.D. to be sent to South Africa. The dates of service regarding South Africa refer presumably to departure from and return to England. Some are apparently incorrect, but are recorded as received.

Name.	Born.	College.	Qualified.	Gazetted.	South Africa From	South Africa To	Retired.	Died.	Notice of Death. P = Photo. * = Career
Finlayson, J.		New Edinburgh	1882	1884	5.9.84	9.11.87		7.2.98 Dublin	*Vet. Rec.* 19.2.98.
Elphick, J. E.		London	1866		4.11.84	21.12.85	7.4.86	21.11.93 Singapore	*Vet. Jl.* Jan. '93.
Berne, J. C.	1843	Edinburgh	1867	7.8.67	28.11.84	28.10.85	18.1.98	19.3.07 London	*Vet. Rec.* 30.3.07.*
Cox, J. H.	1847	London	1867		20.11.84	28.10.85		8.3.91 Umballa	*Vet. Jl.* Apr. '91.*
Crawford, F. F. (h)		London	1873		14.11.84	13.11.90		16.1.00 P.M.Burg	*Vet. Rec.* 26.8.33.*
Woods, J. A.		London	1870		20.11.84	3.11.85	11.6.02	17.11.31 Reading	Dr. F. Bullock's letter of 19.8.35.
Rowe, R.	7.2.44	London	1869	14.7.69	31.3.86	13.3.92	7.2.00	21.3.30 Balham	*Vet. Rec.* 5.4.30.*
Nunn, J. A. (h)	10.5.53	London	1877		10.12.86	10.10.88	5.2.08	23.2.08 Oxford	Smith (1927) *: *Vet.* Sept. '05. P *: *Vet.* 7.3.08.*
Raymond, R. W. (h)	20.9.59	London	1883		1.12.87	16.8.93	4.10.13		
England, A. (h)		London	1891		5.6.93	9.3.03	8.10.13		
Frost, R. F.	1848	London	1870		20.9.95	3.2.99	10.2.00	28.8.09 Dublin	*Vet. Rec.* 4.9.09.
Wilson, F. W. (h)		London	1891		28.3.95	3.4.03	28.6.26		
Wilkinson, H.		New Edinburgh	1885		6.4.95	7.10.95		7.10.95 P.M.Burg	*Vet. Jl.* Nov. '95.
Hunt, F. W. (h)		London	1895		24.9.96	26.6.03	28.11.28		
Millar, W. M. (h)	12.1.67	London	1895		26.8.96	–.3.98		25.4.14 Lucknow	*Vet. Rec.* 23.5.14.*
Kelly, E. H.		London	1880		2.1.96	1.4.98	2.4.98	1914 ?	
Whitfield, G. D.		Edinburgh	1871		2.5.96	23.6.97	28.12.98	16.3.25 Guernsey	D.G./A.V.D., letter of 11.11.35.
Larnder, E. W. (h)		London	1887		1897				
Shore, F. H. (h)		New Edinburgh	1895		16.4.97	23.2.03	19.6.08	6.12.16 Saharanpur	D.G./A.V.D., letter of 11.11.35.
Newsom, A. C. (h)		London	1887		1.10.97	31.12.03	15.10.22	20.1.36 P.M.Burg	*Vet. Rec.* 15.2.36.*
Lane, A. H. (h)		London	1892		16.4.97	27.6.03	3.12.13		Name removed from Register, own request. 1921.
Coley, J. T. (h)	17.6.70	London	1895		15.5.97	9.9.01		1906 Rawalpindi	*Vet. Rec.* 30.6.06 & 21.7.06
Matthews, Iles (h)	27.3.54	London	1872		4.6.98	6.3.03	25.5.06	25.5.06 Simla	
Tatam, W. J. (h)		New Edinburgh	1888		16.7.98	11.1.99	8.7.19		
Harris, C. B. M. (h)		London	1890		7.12.98	15.11.04	13.1.21	22.7.32 Leamington	
Bartlett, E. B. (h)		London	1896		8.4.99	21.10.02			
Cochrane, R. C. (h)	11.5.71	London	1897		22.4.99	7.12.05	24.2.25	23.10.25 Droitwich	*Vet. Rec.* 7.11.25.*
Durrant, H. M. (h)	1875	London	1897		1.7.99	25.4.02	13.12.13	24.4.25 ?	*Vet. Rec.* 9.5.25.
Griffith, J. J. (h)		Edinburgh	1893		1.7.99	12.2.05	16.3.19		
Williams, G. M. (h)		New Edinburgh	1892		15.7.99	24.2.03	25.10.25		
Moore, John, Sir (h)		New Edinburgh	1885		15.7.99	5.12.00	16.6.21		See *Jl. R.A.V.C.* Nov. '32 E
Smith, W. D. (h)		Glasgow	1888		15.7.99	29.1.03	1.12.25		
Southey, J. B. (h)	1873	London	1895		25.9.99	12.9.01		6.9.09 Khartoum	*Vet. Rec.* 11.9.09.*
Todd, A. G. (h)		New Edinburgh	1892		25.9.99	3.12.04	9.1.28		
Conder, G. (h)		London	1896		28.9.99	28.3.02	24.4.24		
Day, E.	1857	Edinburgh	1879		30.9.99	28.1.01	18.5.04	28.2.30 Reading	*Vet. Rec.* 22.3.30.

(a) J. C. Berne was again in South Africa from 22-7-90 to 20-11-95.
(b) F. F. Crawford was again in South Africa during the early part of the Second Anglo-Boer War (1899—1900).
(c) J. A. Nunn was again in South Africa from 9-8-05 to 20-6-06.
(d) E. W. Larnder was again in South Africa from 1-11-99 to 4-3-01.
(e) W. M. Millar was again in South Africa from 23-12-01 to 3-1-03.
(f) J. B. Southey was again in South Africa from 24-1-02 to 1-4-03.
(g) R. W. Raymond was again in South Africa from 10-6-98 to 10-11-04.
(h) Served in Second Anglo-Boer War (1899—1902) — Smith (1927).
(i) J. Moore visited Natal in 1898 "with a shipload of horses from the Argentine . . . in the *S.S. Denton Grange* when we stuck on the bar for 4 days". (Letter of 5th Sept., 1930.)
(k) Note that Smith does not include Day. E.!

" the P.V.S. wearing an eight-inch feather, Inspecting (Staff) V.S. six-inch, other ranks four-inch ". Sir John Moore (letter of 5-9-30) as a Vet.-Lieut. in 1899 wore cocked hat and red plume; but in 1904, this " was limited to the Director-General and full Colonels", all other ranks wearing the " infantry helmet with ball " (p. 251). Moore adds that no plume was worn " in undress blue uniform in marching order ".

Tunic. Blue Dragoon, as worn by P.V.S. and Staff V.Ss. since 1874, and still in use. *Cuffs and collar* velvet, maroon, the veterinary colour since 1874.

Badges of rank (as at present) were " worn on the shoulder cords " †. Rank was also denoted " by the number of twists given to the Austrian knot on the sleeve ".

Belts and Sword Slings were of white patent leather. A black *sabretasche* " bearing the Crown and Royal Cypher was worn on mounted duties " (p. 250).

Pants ‡. Blue with scarlet stripe.

Jack-boots as shown in the figure.

(b) the raising in August of two mounted regiments for the defence of the Bechuanaland Protectorate (Protectorate Regiment) with headquarters at Mafeking, and Southern Rhodesia (Rhodesia Regiment) with headquarters at Bulawayo. The veterinary officers for these units were Lieuts. W. D. Smith and J. Moore, A.V.D., respectively; (c) the establishment of remount depôts at Durban, Pietermaritzburg and Ladysmith in Natal and at Cape Town ([46]); and (d) the " purchasing of medicines, dressings, etc., in anticipation of the raising of local forces ". (Smith, 1912–14, p. 6.) In order to expedite matters, the P.V.O., Col. Iles Matthews (and Capt. R. W. Raymond, A.V.D.) left Cape Town (although the G.O.C., South Africa, was at Cape Town) and established his working base at Pietermaritzburg.

At the beginning of October, 1899, " with the reinforcements from India came the greatly needed Field Veterinary Hospital (No. 6) . . . . under the command of Capt. Newsom ([47]) . . . It deposited at Maritzburg the anxiously awaited veterinary chests furnished by India. Major Crawford also arrived from that country and was posted to Maritzburg in charge of the base ". (Smith, p. 10.)

On the outbreak of war (10-10-1899) there were four cavalry regiments in Natal, the centre of the approaching struggle, distributed as follows : Eighteenth Hussars at Dundee, 5th Dragoon Guards (1 squadron) at Durban, and 5th Lancers and 19th Hussars (along with most of the Natal mounted units) at Ladysmith. There were in addition Mounted Infantry companies of the several infantry battalions, artillery, and the various departmental corps, all requiring veterinary attention—naturally a tremendous strain on the neglected A.V.D.

([46]) Lieut. W. G. Williams, A.V.D., accompanied Col. Stevenson, the Remount Officer in Natal, from August, 1899, to November, 1900 (Smith, p. 125).

([47]) " India . . . was compelled after the Afghan Campaign of 1879 to recognise the necessity of Veterinary Hospitals for both peace and war . . ." (Smith, p. 10).

TABLE IV.

(d) STATISTICS RELATING TO ARMY HORSES AND MULES IN SOUTH AFRICA.

	Year ending	31.12.86	31.12.87	31.12.88	31.12.89	31.3.91 [b]	31.3.92	31.3.
	Strength	732 [a]	717 [a]	790	616			
Percentage	Admitted to treatment	75.95	94.47	104.94	72.61	98.39 [c]	84.3 [d]	77.2
Percentage	Died	12.43	22.59	11.08	3.18			
Percentage	Destroyed	1.77	0.31	1.2	0.3			
Percentage	Total Mortality	14.2	22.90	12.28	3.48	5.48	8.26	5.9

TABLE IV—(*Continued*).

	Year ending	31.3.94	31.3.95	31.3.96	31.3.97	31.3.98 [k]	31.3.99
	Strength				932	2,218	2,519 [i]
Percentage	Admitted to treatment	128.01 [f]	104.34 [g]	97.53 [h]	91.18	100.33	74.19
Percentage	Died					17.54	
Percentage	Destroyed					1.10	
Percentage	Total Mortality	10.14	5.72	8.89	27.52	18.64	17.98

[a] A discrepancy exists.

[b] Instead of the year ending 31-12-90, it ended 31-3-91.

[c] The number was actually 610.

[d] The number was actually 510.

[e] The number was actually 471.

[f] The number was actually 745.

[g] The number was actually 601.

[h] The number was actually 592.

[i] Average strength.

[j] Accompanying these horses were Lieuts. E. W. Larnder and J. T. Coley, A.V.D. The former at Cape Town in 1897 assisted in saving from drowning a fireman of SS. *European*. For this he was presented with a testimonial on vellum by the Royal Humane Society.

[k] Among these were horses from America brought over by Capt. E. Day, A.V.D.

Discussion.—The above data, although incomplete (and collected during a hurried visit to London in 1926) serve to show: (i) The augmentation of army horses and mules in 1897/98 owing to the purchase of 1,403 remounts in South America, and the transfer of three batteries R.F.A. (397 horses) from England [j]. In spite of the mortality of 27.52% during 1896–97, followed by a loss of 18% during each of the two subsequent years, the average strength at the end of March, 1899, was 2,519, a clear indication that the authorities expected trouble! (ii) The lowest admission rate was 72.61% in 1889, incidentally the year with the remarkably low mortality rate of 3.48%. The highest admission was 128% for the year ending March 31st, 1894. (iii) The total mortality rate varied from 3.48% in 1889 to the high figure of 27.52% for the year ending 31-3-1897. As a result of the severe mortality in 1887 and

1897 V.C. Nunn and Lieut.-Col. Iles Matthews respectively were sent out to make investigations, the latter later taking over as P.V.O.

Except for 1888 and 1896, when military operations were in progress, the chief cause of mortality was horse-sickness.

Other maladies responsible for heavy losses were sand colic (Ladysmith); glanders (Natal) and rinderpest (Natal) in 1897–98; osteoporosis (Wynberg) in 1898–99; and anthrax (Eshowe) in 1898–99. Mallein was used in 1897 at which date the Board of Agriculture in Great Britain was still considering its value.

Fig. 12.
DEPARTMENTAL BUTTON
ADOPTED 1894.
From 1894–1900 the pattern was raised; thereafter it was indented.

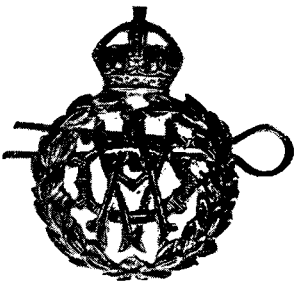

Fig. 13.
BADGE OF A.V.D.
ADOPTED 1891.
With regard to shoulder titles, Sir John Moore writing of the Second Anglo-Boer War states (5-9-30): "A.V.D. was the usual designation on the shoulder straps." C.V.S. F. J. Dunning writes (22-10-32) "no shoulder badges were worn by a C.V.S."

(e) Conclusion.

The chapter bridges a gap in the history of the A.V.S. in South Africa. Events up to March, 1881, have been described by Curson (1935) in *Matters of Veterinary Interest* 1795–1881, while Sir Frederick Smith has given a fairly complete record of the Second Anglo-Boer War of 1899–1902.

The period is concerned with the early days of departmentalisation, which, although a decided improvement on the regimental system, was lacking in efficiency. This was due chiefly to the absence of veterinary hospitals and subordinate staff.

† These were first adopted in the Army in 1857 (p. 248).
‡ These with jack-boots for mounted duties replaced booted overalls in 1872 (p. 249).

After the retirement of P.V.S. Fleming in 1890, a period of stagnation followed, his successor possessing but little initiative or driving force. The declaration of war in October, 1899, found the A.V.D. totally unprepared for a campaign.

Of particular interest are the following facts : (a) The P.V.O. was in Natal while the G.O.C. Imperial troops was at Capetown; and (b) the use of the *Natal Government Gazette* as the medium for publication of veterinary reports.

A list of Imperial Cavalry regiments, which served in South Africa from 1795 to 1914 (Table I) is included, as are tables giving the P.V.Os. and V.Os. from 1881 to 1899. The dates indicating periods of service in South Africa require confirmation.

(*To be continued.*)

PERSONAL NOTES.

Mr. W. P. Hamlyn, M.R.C.V.S., formerly G.V.O., Johannesburg, retired on pension on the 22nd February, 1937, after nearly 23 years in the Service. He has been succeeded by Mr. V. Cooper, B.V.Sc., formerly G.V.O., Estcourt.

* * *

Mr. A. R. Thiel, B.V.Sc., who was G.V.O., Port Shepstone, has resigned from the Service in order to take up private practice in Johannesburg, where he will be in partnership with Mr. J. G. Boswell, B.V.Sc.

* * *

Recent visitors to Onderstepoort are Mr. D. A. Lawrence, B.V.Sc., Director of Veterinary Research, Southern Rhodesia, and Mr. H. Carter, B.V.Sc., of Sydney, who is engaged on wool research.

* * *

The following transfers have occurred in the Field Service : Mr. F. B. Wright, B.V.Sc., from Nongoma to Estcourt, Mr. L. W. Rossiter, B.V.Sc., from Ladysmith to Nongoma, Mr. J. H. B. Viljoen, B.V.Sc., from Dundee to Ladysmith, Mr. J. M. Fourie, B.V.Sc., from Allerton to Hoopstad, and Mr. A. F. Tarr, B.V.Sc., from Umtata to Port Shepstone.

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Disinfection.

By MAX STERNE, B.V.Sc., Onderstepoort.

In the medical sense disinfectants are agents which can kill pathogenic bacteria and viruses. This definition includes antiseptics and bacteriostatics; for these are but disinfectants acting slowly. For example, corrosive sublimate in a dilution of 1 : 300,000 is a bacteriostatic and inhibits the growth of *B. anthracis* in culture. The same substance in 1% solution kills more rapidly and is then considered to be a disinfectant. The distinction, although useful, is quite arbitrary.

Bacteria can be killed, not only by chemical agents, but also by radiations, such as heat and light, by emanations from radio-active substances, by great pressures, and even by sound waves. All these might be termed disinfectants. Specific lethal factors, such as antibodies, are usually excluded from the definition, and will not be discussed in this review.

Nowadays, almost every layman knows that disease is caused by microbes and that these can be killed by certain chemicals. Unfortunately, many members of the veterinary and medical professions can claim little more knowledge of the subject of disinfection. Although the use of disinfectants requires knowledge and discrimination, a great deal of reliance is still placed on the effects of strong smells, and so substances which have repeatedly been proved ineffective are still used.

The usual procedure, firmly entrenched by custom, is well known to all of us. A contaminated floor is swabbed down with a bucketful of cresol emulsion and very little attention is given to correctness of dilution or to the length of time necessary for disinfection. It is rather instructive, therefore, to consider the killing power of some of the commonly used chemicals.

Alcohol is frequently used to sterilize surgical instruments. I have seen metal cases which are specially designed to carry syringes constantly immersed in alcohol. From this one judges that alcohol is assumed to be particularly active against organisms liable to contaminate wounds, that is against streptococci, staphylococci, and pathogenic anaerobes. But what do experiments show? *B. coli* can survive in 70% alcohol for an hour, in 80% alcohol for a day, and in absolute alcohol for longer periods. Staphylococci withstand autoclaving at 120° C. in absolute alcohol, while anthrax spores can live in it for months. In practice, alcohol is not even used at its most potent concentration (50–70%), but at 96%—as rectified

spirits : this concentration seems to be somewhat less toxic for bacteria than is distilled water. It would almost appear that those who use alcohol are more concerned with the safety and care of their tools than with the welfare of their patients.

Ether is almost equally popular for washing out syringes and it probably owes a considerable amount of its popularity to its penetrating odour. A 50% solution kills *B. coli* in a few minutes; *V. septique* is not killed in 24 hours; and anthrax spores can live in it indefinitely. It must be remembered that the times I have quoted are those obtaining under ideal experimental conditions. In the presence of serum, pus, and other organic material, the time taken to kill even delicate organisms may be greatly prolonged.

Phenol was at once time widely used as a laboratory and household disinfectant, although it has now largely been replaced by a number of cresol compounds. Under ideal conditions, in the absence of organic matter, a 1% solution kills paratyphoid organisms in an hour; a 5% solution takes more than a day to kill tetanus or anthrax spores and a day to kill the tubercle bacillus. Thus it will be seen that in practice this germicide is rarely allowed to act for a sufficient length of time. A common practice when liming out a stable is to add phenol to whitewash. This is an irrational procedure, because the sodium and calcium salts of phenol are relatively inactive, and moreover the acid neutralizes some of the alkalinity on which the germicidal activity of the lime depends. Carbolic acid has a very high dilution coefficient, so that halving the concentration may reduce its activity by as much as sixty times. Obviously, therefore, it should not be used where dilution is to be expected.

Cresols are now more generally used than phenol, because they are less irritating and have higher coefficients of disinfection. However, the latter aspect is not of great practical importance, since the cresols are correspondingly more affected by admixture with organic matter. Anthrax spores survive for days in a 2% emulsion, and botulinus spores resist a 5% mixture for a similar length of time. Neither phenol nor the cresols are very effective against Gram-positive bacteria or against viruses, but are probably the chemicals of choice against the tubercle bacillus.

Lysol, which is a cresol soap, is supposed to be particularly effective in the presence of greasy matter, although it has been stated that the soap actually protects the bacteria by forming a layer around them. The addition of common salt increases the potency of phenol and the cresols, provided that amounts of 5 to 10% are added. Like phenol, the cresols have a high dilution coefficient.

Formaldehyde is another commonly used and abused disinfectant. Its well-known action on toxins and its pungent odour have been responsible for an exaggerated idea of the rate at which it kills bacteria. Ten per cent.

formalin (an unpleasantly strong concentration) takes two hours to kill anthrax spores and ten minutes to kill typhoid bacilli in the absence of organic matter. Moreover, formalin has a high temperature coefficient and its activity is much reduced at temperatures below 10° C. The times quoted above were obtained at 20° C. Formaldehyde, as a gas, has been used extensively for the disinfection of houses and rooms, mainly because it is non-injurious to metals, fabrics, and wood. Provided that a sufficiently high concentration of the gas and water vapour are maintained for a considerable time, good results can be obtained. However, the disinfectant action of formalin is too slow where rapid sterilization is required.

Phenol, cresols, and formalin are the organic germicides in most general use. Comparatively recently certain aniline dyes came into favour, but owing to their rather selective action have not established themselves as general purpose disinfectants. Some dyes, like brilliant green and gentian violet are most active against Gram-positives, while the flavines (*e.g.* fuchsin) are most active against Gram-negative bacteria. Therefore these dyes are very usefully employed in various selective culture media. Some dyes, like brilliant green, inhibit the growth of Gam-positives in dilutions up to 1×10^7. At one time this fostered the hope that miraculous results would attend the use of such dyes in cases of infected wounds, but unfortunately it was found that serum markedly suppressed their action.

Corrosive sublimate is one of the best known of the inorganic disinfectants. At one time it was thought to be almost the perfect germicide, but more recent work has shown that this favourable opinion is not altogether justified. It used to be said that anthrax spores were killed in a matter of minutes by a 1 : 1000 solution. Actually these spores can remain alive for months in this concentration. Recent work offers two very good explanations for the great discrepancy in these findings.

Firstly, the mercury salt forms an albuminate coating on the surface of the bacterium and this, although inhibiting generation on artificial medium, does not kill, but actually protects the microbe against further disinfectant action. Such supposedly dead organisms may thus still infect when injected into an animal.

Secondly, as was mentioned earlier, mercuric chloride has a bacteriostatic effect even in very high dilution. Thus, when cultures are made from bacteria subjected to the action of the sublimate, sufficient of the latter may be carried over with the inoculum to prevent multiplication of bacteria, even if they are still alive. It is essential to neutralize the corrosive sublimate, or at least to test the inoculated medium for bacteriostasis.

Thus recent work has shown that staphylococci are resistant to a 1% solution and that anthrax spores resist a 1% solution for four months and a 5% concentration for two months.

According to some authors, the disinfectant action of mercuric chloride is increased by the addition of acid or alcohol, while others contend that it is decreased. To be on the safe side, it is probably better not to add these chemicals. In any case, mercuric chloride in absolute alcohol is practically inert. Common salt is definitely antagonistic and should not be added, although corrosive sublimate tablets often contain some of this agent.

Compared with phenol and the cresols, mercuric chloride has a low dilution coefficient, so that halving the concentration only halves its effect. The converse is of course also true, and the lethal effect is not rapidly enhanced by increasing the concentration.

Corrosive sublimate is particularly susceptible to the inhibiting action of albuminous matter such as pus and serum, and so should not be used to sterilize contaminating secretions and contaminated wounds.

The organic mercury compounds such as mercurochrome compounds have had a considerable vogue as antiseptics. However, they are much inferior to iodine as skin disinfectants and even as *in vivo* disinfectants have not fulfilled early hopes.

The halogens are active germicides and chlorine is probably the most generally employed disinfectant. This is as a rule used in the form of different hypochlorites and the disinfectant action then depends on the formation of hypochlorous acid (HOCl). This is extremely active, a 1% solution killing anthrax spores in half-a-minute. The dissociation of hypochlorites with the formation of HOCl occurs only under acid conditions and therefore such germicides are always somewhat corrosive and irritating. Non-irritating chlorine disinfectants are inferior to the more acid solutions and the claims of certain firms that their chlorine compounds are highly effective, while completely non-irritating, are exaggerated.

Organic material reduces the efficiency of chlorine disinfectants, but not to the same extent as in the case of corrosive sublimate.

Hypochlorites are the disinfectants of choice for the purification of swimming-baths and water supplies, for the relatively low concentration of organic matter in such water leaves bacteria susceptible to quite small amounts of chlorine.

Another halogen, iodine, is frequently used as an antiseptic and wound-dressing. At one time its use was almost universal, but a little while ago its efficiency was questioned and as a result iodine fell into disrepute as an antiseptic. This was most probably a reaction from the favour it once enjoyed. When people realised that its antiseptic action was not instantaneous, there was a tendency to discard iodine altogether. However, more recent investigations have shown that there is nothing better. Iodine in 1 : 100—1 : 300 solution is more effective than chlorine,

and far more effective than corrosive sublimate or mercurochrome. It is relatively little affected by serum—a great advantage when used as an antiseptic. The greatest objection to iodine is of course its irritating action on tissues. This is partly due to its use as a tincture and can be avoided by employing aqueous solutions.

Strong acids, such as sulphuric and hydrochloric acid, are rarely used, as their corrosive action is far more evident than their disinfectant power.

Strong alkalis—caustic soda and caustic potash—are powerful and useful germicides. Their action is mainly due to the hydroxylion concentration, and therefore they ought never to be combined with disinfectants having a neutralizing effect. A 1 : 500 solution of commercial lye will kill most viruses and Gram-negative bacteria in about ten minutes, but for disinfecting stables and sheds it is advisable to employ a concentration of 1 : 100 or 1 : 150. Such a concentration is not seriously affected by urine or faeces and stands a fair amount of dilution. The tubercle bacillus is almost unaffected by strong alkali.

Weak alkalis, such as lime, have some disinfectant and bacteriostatic properties and are often used to paint the walls of sheds and stables. Lime-washing should only be done with freshly-slaked lime, because commercial whitewashes are often of low alkalinity and useless as disinfectants.

I have now discussed a number of organic and inorganic disinfectants and I hope it is clear that no disinfectant acts instantaneously. It should also be clear that the commonly used substances, such as alcohol, formalin, phenol, and corrosive sublimate, act quite slowly. There is no chemical that can sterilize albuminous material in a matter of seconds, and the germicides in everyday use, such as lysol, are generally so diluted that they are quite ineffective.

The veterinarian must regard disinfection from two aspects—the safety of animals and the safety of human beings. It is not a serious matter if a veterinarian does not sterilize a syringe properly; for, after all, fatal infections from dirty syringes are infrequent. In human practice such neglect is criminal. However, at times the safety of human beings depends on the veterinarian. He may have to disinfect an abattoir, where an anthrax carcass has been opened. It is patently absurd to mop the place with some lysol, as the time of exposure to disinfection is far too short. Due regard must be given to the time factor and allowance must be made for the presence of albuminous and other organic matter.

A veterinarian is often requested to disinfect premises contaminated with pathogenic bacteria; for example, a fowl-house infected with fowl-typhoid. Now formaldehyde gas is of little use, because of the high concentration required; cresols are effective against Gram-negative bacteria, but are very much weakened by dilution and by particulate

organic matter. However, commercial lye is cheap, is effective against Gram-negatives, and is not much affected by dilution or by organic matter. Thus it permits quite free use of the hose. Lye would probably be the disinfectant to choose in such a case. On the other hand, if a patch of flooring had to be sterilized, it would be advisable to use a strong cresol emulsion. A concentrated emulsion is much more powerful than a dilute one (high dilution coefficient) and is fairly active against spore-formers : a 1% emulsion is quite useless.

It is clear then that disinfection implies the choice of a chemical which will be reasonably efficient in a particular case. This means that the chemical must be diluted properly, allowance must be made for probable further dilution, and above all enough time must be given. It may be necessary, for example, to prevent run-off by contriving a small ledge.

In dealing with the practical aspects of disinfection, it should be mentioned that the hose-pipe can make good deficiencies in the rest of the technique, but should be used after, not before, the disinfectant.

Disinfection by heat might be assumed to be a simple and fool-proof procedure. However, I have seen even professional men adopt methods of heat-sterilization bordering on the ludicrous. In human practice such methods may have distressing consequences; in veterinary practice the results may not be as serious, but may yet prove annoying and wasteful.

The facts relating to heat-disinfection are simple. Any pathogenic organism can be killed if its temperature is raised to 120° C. for 15 minutes. In practice, however, it is often surprisingly difficult to attain this. Take dry heat for instance—the heat of a flame or of a hot-air oven. Combustion in a flame will of course kill any living thing, but this should not lead us to infer that a flame has magical properties. Therefore, if we lightly flame a large cool surface with a Bunsen burner or with a blow-lamp, there is little chance of the temperature rising enough to kill vegetative bacteria, however much the flame roars. As generally applied, the flame does not raise the temperature of a surface to a warmth uncomfortable to the touch, yet bacteria are expected to shrivel up ! After all, the whole object of using commercial flame-throwers for disinfection is to destroy contamination without damaging woodwork, in other words to burn bacteria without heating them. This sounds ridiculous, and it is ridiculous. Streptococci easily resist the usual flaming practised in accordance with the recommendations of makers of apparatus.

Another quite common practice that must be condemned as stupid is to pour alcohol on instruments or dirty surfaces and ignite it. Since a finger may be dipped into alcohol and ignited without much discomfort, how can this degree of heating harm bacteria ?

Hot air ovens are efficient up to a point, and it must be borne in

mind that dry heat penetrates quite slowly. Vegetative bacteria are killed in 1–2 hours at 105° C. Tests have shown that the temperature at the centre of a bundle of tow, 2 cub. ft. in volume, which is heated to 150° C. for three hours, is only 75° C. In practice, dry heat of a degree sufficient to kill spores chars cotton-wool and most fabrics.

A rough test shows quite clearly the difference between dry heat and moist heat. The hand can be put into quite a hot oven and held there for an appreciable time, provided that the sides are avoided. The same experiment with steam at 100° C. will convince anyone of the difference between the two forms of sterilization.

Moist heat is widely used for sterilizing instruments and all manner of infected material. Boiling kills most vegetative bacteria in a few seconds, provided they are not protected by extraneous matter. Spores are much more resistant : anthrax spores—by no means the most resistant —may resist 100° C. for 40 minutes and 95° C. for 50 minutes. It should be remembered that water boils below 100° C. above sea-level and allowance should be made for this where necessary.

Boiling is therefore the best method for the emergency sterilization of instruments. However, spores of dangerous anaerobes are not destroyed and where serious contamination of this kind is suspected further precautions must be taken. For example, a very thorough washing may be necessary. The objection to boiling is its detrimental effect on the edges of knives and the points of needles. To avoid this some people sterilize instruments and needles by heating in oil. In this case much higher temperatures should be used than are customarily employed, as this is a form of dry sterilization.

In laboratories and disinfectant stations, steam under pressure is used to sterilize instruments, bedding, etc. The process is carried out in autoclaves and the basis of all such installations is that the objects to be sterilized come into intimate contact with *saturated* steam under pressure. It is safe to say that all vegetative bacteria and spores will be killed by steam at 15 lbs. pressure for 30 minutes. However, even this process is not fool-proof, and certain requirements are essential :—

(1) The air in the autoclave must be completely replaced by the steam, otherwise the sterilization is partly a dry one.

(2) The steam must be saturated, *not* superheated, for the same reason as in (1).

(3) If empty plugged bottles are to be sterilized, a little water should be left inside them, otherwise the sterilization is again a hot air one.

(4) Above all, time must be allowed for every part of the object to attain the desired temperature and to remain thereat for the correct period. For example, the temperature in the centre of a

flask containing 500 ccm. of water is still only 100° C. after 10 minutes at 120° C. in the autoclave. Large batches of media or bulky objects may take several hours to attain the correct temperature throughout.

I hope that I have cited sufficient examples to show that disinfection and sterilization are not procedures to be entrusted to unsupervised unskilled workers. Members of the public often ask us how to disinfect stables, instruments, utensils, and other objects. We should know enough to be able to guide them in the choice of method and material : it is useless to say, "Just throw some dip on it."

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The facts quoted in this review have been collected from a number of sources. Only some of the more important of these are given below. Readers are particularly referred to the paper by McCulloch (1936).

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We have recently received the latest number of "The Bloodless Phlebotomist" (Vol. VIII, No. IV). This small publication of only 24 pages contains a variety of medical and other articles which members of the profession will find of considerable interest.

Among the non-medical articles is one which is, appropriately enough, entitled "Staggering". It is the story of a native of Bombay who claims descent from one of the "Wise Men of the East" of Biblical fame, and from which one learns the amazing fact that every living person on earth has had, since the birth of Christ alone, the prodigious number of 144,115,188,075,855,870 ancestors, and the article tells how this figure is, simply enough, arrived at.

Among the medical articles are "Successful Arthritus Therapy," "Rheumatic Sciatica," and "Bronchitis." "Control of Human Death predicted by Dr. Carrel" also makes interesting reading.

"The Bloodless Phlebotomist" is published by the Antiphlogistine Laboratories in the interests of their product, but the reader is not wearied with a lot of advertising. It is sent gratis to members of the medical and allied professions throughout the world. Any of our readers who fails to receive a copy may obtain one by writing direct to The Denver Chemical Mfg. Co., 163, Varick Street, New York.

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Biliary Fever (Nuttalliosis) of the Cat: A Case in the Stellenbosch District.

By CECIL JACKSON, D.Sc., B.V.Sc., Onderstepoort, and F. J. DUNNING, F.R.C.V.S., Stellenbosch.

The question has often been discussed among veterinarians in South Africa: Is the cat susceptible to "biliary fever"? The query, as propounded in this form, reveals some confusion of thought; for it really comprises two problems which should be clearly distinguished:—

(1) Is the cat susceptible to *Piroplasma canis*?

(2) If not, is there nevertheless a disease which occurs in cats and which is comparable with canine piroplasmosis?

A negative answer to the first of these questions cannot, in the absence of any information on the second, be expected to satisfy clinicians in this country, several of whom have, in the course of their practice, observed in cats a disease which in its symptomatology closely resembles biliary fever of the dog. Indeed there are at the Onderstepoort Laboratory blood-smears from at least three such animals which have been sent in from time to time by different veterinarians, who have, however, published no records of their observations. [1])

It is the purpose of this note to supply a categorical answer to the second, in addition to the first of the questions mentioned above.

The Non-susceptibility of the Cat to *P. canis*.

Both Robertson, as long ago as 1901, and Nocard and Motas in 1902 attempted transmission of *P. canis* to the cat, with negative results. The latter authors (1909) were not even able to obtain transmission to wild Canidae (jackal, fox). Knuth and du Toit (1921) state very clearly that this parasite is pathogenic only for the dog. Thomas and Brown in 1934 again undertook transmission experiments with cats. Their work was prompted by the opinions, already mentioned, prevalent among veterinarians.

The entirely negative results which they obtained, even after splenectomy, are to be regarded as complete confirmation of the earlier

[1]) The following communication in this number of the Journal, by Mr. J. McNeil, M.R.C.V.S., was received for publication while this article was in the press. —(Editor).

observations that the cat is not susceptible to *P. canis,* and are further supported by the absence of any observation of the natural occurrence of this infection in the cat, although, as Thomas and Brown point out, these animals must frequently harbour ticks infected with this parasite.

Nuttalliosis of the Cat.

The literature regarding Piroplasmidae of carnivores other than Canidae is sparse. From a table published by Carpano (1934) it is seen that six species are known. Of these, the two affecting Felidae are *Babesiella felis,* Carpano, 1933—a parasite of the Puma (*Felis concor*)—and *Nuttallia felis* (Davis, 1929), which affects the Sudanese wild cat (*Felis ochreata*).

Experimentally, *Nuttallia felis* is transmissible to the domestic cat, in which however it causes no illness, the percentage of infection of the erythrocytes never rising above 1%. Even after splenectomy, Davis did not observe very severe symptoms : there was anaemia and haemoglobinuria, but never jaundice and scarcely any hyperthermia; in only one of his splenectomised animals was there a fatal result, but this was complicated by tuberculosis.

Case Report.

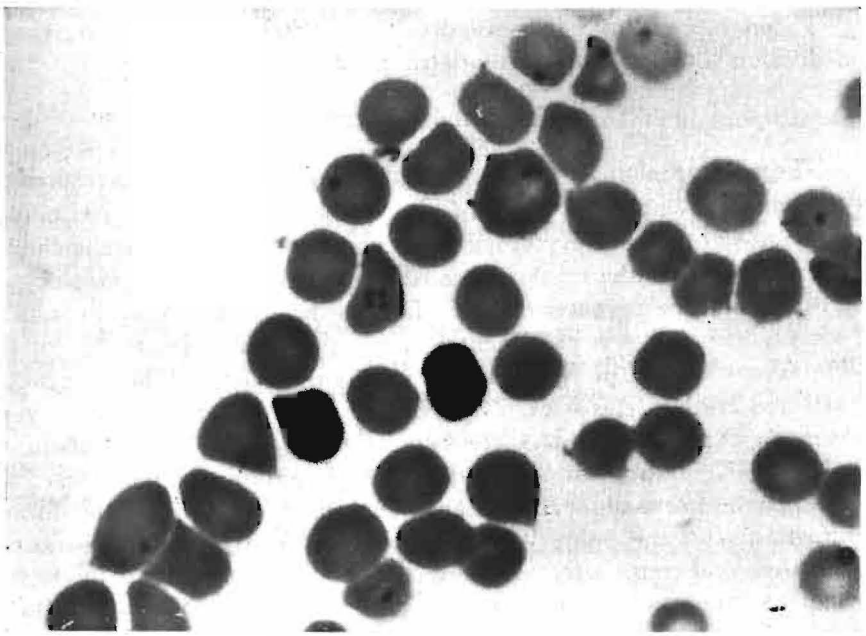
On the 24th May, 1936, a sick dog and a sick cat were presented to one of us (F.J.D.), having been brought in from a farm in the Stellenbosch district. The dog had a temperature of 105.2°; the pulse was 120; blood-smear examination revealed *P. canis* to be fairly frequent. A curative injection of Akiron was administered. The temperature of the cat was 104.8°; the conjunctiva was pale and yellowish discoloured. The blood-smear showed signs of anaemia and a heavy infection of piroplasms. Biliary fever was diagnosed and an injection of 0.4 cc. Akiron was administered. Within 48 hours, both patients were reported to be convalescing and to have regained their appetites. On the 1st June, both had a slight relapse, whereupon the Akiron injections were repeated. Uneventful recovery ensued in the case of the cat; the dog, however, had a second relapse necessitating further treatment.

Description of the Parasite.

Morphologically the parasite is indistinguishable from *Nuttallia felis* (Davis, 1929), so far as can be judged from the description published by Davis. He describes this parasite in strained blood-smears as

> a small circular body with faint blue-straining cytoplasm and dark red-straining chromatin. The disposition of the chromatin is usually peripheral; in some it is in the form of a small granule, in others it is dispersed around a varying proportion of the periphery of the parasite. Many of these are vacuolated in the centre, in which case they appear ring-shaped . . . Departures from the circular shape are common. Many of the irregular forms are doubtless produced by the act of making the blood-film, but

others appear to result from amoeboid movement of the parasite. Evidence of sub-division in the shape of four daughter individuals disposed in a cruciform manner, is occasionally encountered. In some of these, chromatin is visible in others, faint traces of blue-staining cytoplasm can be made out. The daughter individuals are pear-shaped. In some groups the individuals appear to be attached to each other at the center of the cross; in others they are disjoined. Erythrocytes were also seen containing four young parasites larger than those just described, in which the blue-staining cytoplasm is quite distinct. Forms of the adult parasite are occasionally met with, the nuclear material of which is greatly increased at the expense of the cytoplasm. Many of these, appearing to consist mainly if not entirely of chromatin, tend to be X- or H-shaped. It would seem probable that such formations are preliminary stages of subdivision. During a period of heavy infection, multiple infected red blood corpuscles are often seen; two or more parasites, each representing a different stage of development may inhabit one red cell . . . A number of parasites, measured by means of a micro-projection apparatus, were found to vary from less than 1 μ up to 2.25 μ in diameter, the majority being about 1.25 μ.



BLOOD SMEAR FROM A CAT SUFFERING FROM NUTTALLIOSIS (2290 x).

Note the cross form (to left of centre), a normoblast (to right of centre), anisocytosis, and (lower right corner of field) a Jolly body.

Little need be added to this account in order to describe the parasite of the domestic cat. The great majority of the piroplasms are circular or irregularly circular in shape and have an average diameter of ca. 1.5 μ. The larger ones commonly exceed 2 μ. The chromatoid material (staining reddish with Giemsa) is usually arranged as a crescent around part of the

circumference of the blue-staining cytoplasm, which is centrally often conspicuously vacuolated. The majority of the infected erythrocytes contain one parasite; often two are seen, in different developmental stages, as described by Davis. Few cells contain more than two. A minority of the parasites are somewhat elongated and may measure as much as 2.8 μ x 2 μ. Distinct pyriform types are rare; they may measure as much as 4 μ in long diameter.

Division forms appear to be predominantly of the cruciform type characteristic of the genus Nuttallia. Very commonly two parasites of equal size are found closely apposed to each other near the centre of the cell; there was no direct evidence, however, that this represented binary fission. The frequency of this arrangement offers a possible distinction from Davis' parasite. Rarely divergent forms were seen, two parasites lying with their narrow ends in contact and arranged at an obtuse angle to each other, as in *B. divergens*. This also may represent binary fission.

The parasite stands in marked contrast to *P. canis*, the size, shape, and division forms all constituting striking differences.

The Blood Picture.

There is pronounced anisocytosis, the erythrocytes as measured in dry smears varying from 7μ to 16 μ in diameter. Since the normal average size of the cat's erythrocytes as measured in dry smears is ca. 7μ, it will be seen that the erythrocytes of abnormal size are all macrocytes, microcytes not being encountered. Diffuse basophilia (polychromasia) of the erythrocytes is a prominent feature, affecting at least 10% of the cells. About 0.08% of the red cells are normoblasts. The percentage of affected erythrocytes is ca. 7%. The Jolly bodies mentioned by several authors in the blood of cats are seen, but apparently not in abnormal numbers. They are distinguished from the piroplasms with ease, the dense appearance and deep staining and sharp outlines of the former contrasting with the lighter staining and less clearly outlined parasites. The differential count was : L 30%, M 1.5%, N 67%, E 1.5%, which is normal for the species. The Arneth count was : I – 70, II – 24, III – 5, IV—1. This also would appear to be normal for the cat, although we know of no published statistics on this point.

Systematic Classification of the Parasite.

The parasite falls readily into the genus *Nuttallia* Laveran, 1901. The characteristics of this genus are that the parasites are oval or pear-shaped, no typical rod-forms occur, and multiplication is by cruciform quadruple division. Davis points out that in the genus Nuttallia the adult parasites tend to assume a pear-shaped form, and that this " is not

charaacteristic of the cat parasite ". Davis proposed the name *Babesia felis*. It must be admitted that the majority of the adult forms in the cat are not only not pear-shaped, but that the degree of elongation to produce oval forms in most cases is very limited, so that most of the parasites appear roughly circular. Nevertheless, both pear-shaped and distinctly oval forms are by no means uncharacteristic, so that at least the tendency to such forms is there. In any case Davis by following Wenyon (1926) in refusing to recognise Du Toit's classification of the family Babesiidae into six genera (of which Nuttallia is one), escapes from the difficulty : he has to decide only whether the parasite belongs to this family, which according to Wenyon comprises but one genus. To a follower of Du Toit's classification, Davis' parasite should have been named *Nuttallia felis*. This Carpano had no hesitation in doing. It seems desirable that a clear distinction should be made between *N. felis* and the parasite of the domestic cat, on the basis of the striking difference in pathogenicity for the cat. We therefore propose for this parasite the name *Nuttallia felis var. domestica*.

ACKNOWLEDGEMENTS.

We are indebted to Dr. P. J. du Toit, Director of Veterinary Services, for advice in the preparation of this article.

Mr. Theo. Steeg, of Messrs. Taeuber & Corssen (Pty.), Ltd., kindly took the photomicrograph, using a Leitz Panphot microscope.

SUMMARY.

The cause of biliary fever in the domestic cat in South Africa is described as a piroplasm belonging to the genus Nuttallia. This is the first record of a piroplasm pathogenic for the domestic cat.

The suspicion that there is an aetiological connection between piroplasmosis of the dog and piroplasmosis of the cat, which has persisted in spite of negative transmission experiments, is thus without foundation.

The parasite is closely related to *Nuttallia felis* (Davis, 1929), a parasite of the Sudanese wild cat, and indeed is probably morphologically indistinguishable from it.

The symptoms and blood picture of the disease as observed in a single patient are briefly described.

We propose for this parasite the name *Nuttalia felis, var. domestica*.

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Piroplasmosis of the Domestic Cat.

By JOHN McNEIL, M.R.C.V.S., Cape Town.

Piroplasmosis of the domestic cat is a disease wihch has been under my notice for several years, but so far as I am aware it has not been described by veterinarians as affecting this animal either in Cape Province or in other parts of South Africa. This disease is not a very frequent one, but isolated cases come to my notice each year. It is endemic, may occur at any period of the year, and is found in places which are in close contact with virgin coutnry fringing Table Mountain or with the raw, unbroken veld adjoining Cape Town and its hinterland.

Many cases of the disease may exist; but perhaps owing to the low value placed on cats, as well as to the instinct of this species to seek solitude when sick, many may escape notice.

Symptoms: The diseased animal is seldom presented until such time as well-marked symptoms of illness have manifested themselves. The most outstanding symptoms are those associated with anaemia. The temperature is 103 to 104.5 F.; breathing is slow and hardly perceptible; there is very marked lethargy, the animal having a tendency to remain in the same position for many hours. When forced to move, the patient shows a markedly unsteady, rolling gait, and after a short time appearances of great exhaustion, pronounced weakness, and helplessness in accomplishing simple natural movements. There is also a great loss of weight.

The normally pink mucous membranes are either very pale yellow in colour or almost completely blanched. The deeply yellow jaundiced sclerotic and mucosa is not a marked feature except in few instances, but the pale blanched conjunctiva is present in most cases.

Constipation is a most frequent symptom and when faeces are passed, they are of a bright yellow, orange, or Indian red colour. The urine, when it can be examined, will be found to be stained bright citron, yellow, orange, or red (haemoglobin), and it froths at the time of emission.

In thin animals the spleen may be palpated, and will be found much enlarged [1]).

The pulse is hard and frequent, more especially if examined after the patient has been very slightly exercised.

Ticks are rarely found on the animal. The only species I have found during the course of the disease has been *Haemophysalis leachi.*

Microscopic diagnosis is readily achieved by staining blood-smears with Giemsa or Leishman. The piroplasms will be seen as small intracellular pear-shaped, or (what is more frequent) rounded or rod-like bodies about a third of the size of *P. canis,* occupying a position about the middle third of the diameter of the red blood cell.

Post-mortem examination reveals an almost entire absence of adipose tissue, and the carcass, viscera, and peritoneum seem " dry ". Even the mesenteric blood-vessels seem almost empty. The blood most often has a pale orange appearance. The peritoneum and subserosa vary in colour from a pale ivory to a bright citron; but any vestiges of fatty tissue which remain are usually icteric. The spleen is softer than usual, varying in colour from brownish red to the deepest purple; the edges are rounded and the whole organ is about three times its normal size; the pulpa is softened but not liquified. The gastro-intestinal tract is usually empty, except near the rectum, where the mucosa is covered by a thick tenacious yellow or orange mucus. The liver is enlarged, softer than usual, friable, and usually bile-stained. The gall-bladder is distended and contains a viscid reddish-green bile. The urinary bladder contains a quantity of deep orange or dark red urine, and the mucosa is the seat of haemorrhages.

This disease of the cat is not attended by nearly so high a mortality as is canine piroplasmosis, in which in my experience 100% of untreated animals have died. A cat placed at my disposal had 3 ccm. blood extracted from the left ventricle and made a recovery where no treatment had been adopted. Recovery is in most instances effected by the hypodermic injection of Trypan Blue or Acaprin, given in doses bearing the same proportion to the body weight as are recommended for dogs.

I have performed a few experiments in connection with the disease. It may be stated that the habits of the cat cause difficulties which would be unexpected in most other species. As already stated, it is rare to find

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[1]) Examination of the spleen of the cat is much more difficult than in the case of the dog, where it lies much more externally. Splenic enlargement is a valuable accessory symptom of piroplasmosis of the dog.

a tick present on the skin of the animal, as the cat is notoriously thorough regarding its toilet and will not tolerate the irritation caused by the presence of such parasites.

As a general practitioner, one does experimental work under conditions of difficulty. Much careful laboratory work is still required in the investigation of the disease.

I. *Experiments with blood taken from a cat suffering from piroplasmosis.*

Blood taken from the left ventricle of an infected cat was subinoculated into three other cats by the intramuscular, subcutaneous, and intraperitoneal routes respectively, the dose being 1 ccm. in each case.

Daily temperatures were taken for 30 days, but no abnormal temperature or general reaction or other symptoms of the disease appeared; nor did the cats, for a further period of two months, show any symptoms of the disease.

II. *Experiments with ticks cultivated from an adult female tick* (Haemophysalis leachi) *taken from a cat suffering from piroplasmosis.*

(a) I placed a large batch of larvae hatched from eggs deposited from the above tick on a year-old cat, but no reaction or any symptoms of disease followed.

(b) I placed about 50 nymphae grown from eggs deposited by the above tick on a nine-year old cat, with equally negative results.

(c) My bottle containing the fully developed imago ticks was unfortunately broken by a fall and as this occurrence was not reported to me until too late, I was unable to recover any of its contents; but strange to say, my own household cat contracted the disease. In fact, I got a shock when I saw that the animal was ill and examination showed him to be suffering from piroplasmosis. I am confident (although I have no complete proof) that the animal contracted the disease from the experimental ticks. The diagnosis was confirmed microscopically.

CONCLUSIONS.

My conclusions are that the disease is conveyed by the imago of the *H. leachi* and that it is not conveyed by the larval or nymphal forms of this parasite. The disease was not transmitted by subcutaneous, intraperitoneal, or intramuscular injection of 1 ccm. of blood from an infected cat.

I am of opinion that the reservoir of the disease in this area may be looked for in the wild cat (*Felis caffra*) which exists here in great numbers. As already stated, the disease is not nearly so fatal as is canine piroplasmosis. In the wild cat the disease may be still more benign. The occurrence of the disease in parts adjacent to unbroken country supports this view of the source of the infection.

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Calcium and Phosphorus Metabolism in Relation to the Production of Osteodystrophic Diseases in Domestic Animals.*

By A. I. MALAN, D.Sc., Biochemist, Onderstepoort.

My object in this brief summary of the work done on the above subject here and overseas is to emphasize the known and accepted facts, and secondly it is an attempt to consider apparently contradictory results and to present certain observations and hypotheses for your consideration and criticism.

The osteodystrophic diseases that are usually associated with abnormal Ca : P metabolism are (1) rickets, which is referred to as osteomalacia in the adult, (2) osteofibrosis, and (3) bone atrophy or osteoporosis, but of course it is not claimed that the occurrence of these conditions is invariably associated with a dietary deficiency of P or Ca or Vitamin D; for indeed it is not. Marked bone atrophy may be brought about by a number of causes, such as worm-infestation and diseases which lead to poor nutrition and condition of the animal, but it is also associated with low dietary Ca and P and it will be mentioned in this connection only.

A survey of the literature in an attempt to correlate these osteodystrophic diseases individually or separately with Ca or P metabolism or both brings out very clearly that in the absence of assistance to undertake the pathological aspect of the work the majority—possibly 90 per cent.—of the investigators have had to content themselves with referring to the conditions produced as rickets or bone disease or merely abnormal bone formation, without any attempt to differentiate between the diseases. This lack of specificity has yet another cause, viz. the absence of an aetiological distinction on the part of pathologists between rickets or osteomalacia and osteofibrosis. Naturally, therefore, the production of a diseased condition was the object of the investigators rather than the investigation of and differentiation between the conditions produced.

Thus we have the interesting position in the literature that rickets, osteofibrosis, and osteoporosis are associated with abnormal Ca : P metabolism, without any aetiological distinction between these conditions. The cause for this apparent confusion is not far to seek. The occurrence of rickets in infants is invariably associated with vitamin D deficiency and hence experimental work on the production of rickets was almost

* Paper presented at a meeting of the Onderstepoort Research Staff on 3rd March, 1937.

invariably carried out under conditions of vitamin D deficiency, until Sir A. Theiler described rickets caused by P defiency in bovines receiving abundant supplies of vitamin D. Furthermore, natural cases of rickets in calves have been reported and were found to be due to a deficiency of vitamin D; this also associated the occurrence and production of rickets in domestic animals with a vitamin D deficiency. Thus these facts gave stimulus to the investigation of Ca and P metabolism under conditions of vitamin D deficiency. It must be clear that such work on Ca and P metabolism will produce results that cannot be attributed solely to a deficiency of Ca or P or both, but to the deficient mineral plus a deficiency of vitamin D—hence an indefinite result due to at least two variables.

In order to appreciate the relation between abnormal Ca : P metabolism and the development of osteodystrophic diseases it is necessary to consider briefly the factors which produce normal bone under normal conditions. Robinson and his co-workers at the Lister Institute have done much to give us an insight into the difficult problem of ossification. The factors responsible for precipitating the bone salts in the bone matrix or osteoid are first inorganic P and Ca continuously supplied by the blood and by the fluids in which the bone matrix is bathed; secondly, an enzyme called phosphatase which produces inorganic P from organic salts or esters; and in addition an unknown third factor, which is influenced by vitamin D. Vitamin D itself has no demonstrable effect on ossification *in vitro* according to Robison and Rosenheim; but lack of it affects the unknown ossifying factor and abnormal bone is produced. Actually, therefor, we have (1) an unknown factor dependent on the presence of vitamin D for proper function, (2) phosphatase for the production of inorganic P from organic compounds, and (3) inorganic Ca and P in solution which are deposited in the bone matrix as soon as the concentration of Ca and P reaches a certain point, or, in more chemical terms, when the concentration of these ions exceeds the solubility product of calcium phosphate.

Phosphatase is always present and need not be considered further as it has never been shown to be responsible for abnormal ossification. Hence the factors for consideration are P, Ca, and lastly vitamin D, inasmuch as it affects the unknown ossifying factor.

A point that should be emphasized here is the *modus operandi* of vitamin D at the site of ossification. If (as Fischman, Robison and others have shown) it affects the unknown ossifying factor, then osteodystrophic diseases produced by deficiencies of vitamin D or Ca or P respectively are distinct from an aetiological standpoint, which Theiler believed to be the primary principle in the definition of a disease.

The essential point is that it would be unwise to assume tacitly that the lack of any one of the three ossifying factors will necessarily produce one and the same bone disease merely because all three happen to be

functionally associated with ossification. Each one may and does play a specific part in this complicated process and lack of it might not affect ossification in the same way as lack of either of the other two factors would.

With the above in mind let us examine some of the recent results obtained by investigators.

Shohl and his collaborators at the Harvard Medical School, and Huffman, Duncan and their co-workers at the Agricultural Experiment Station, Michigan, have investigated from both a bio-chemical and pathological aspect the problem of Ca : P metabolism and the production of osteodystrophic diseases.

The workers at the Harvard Medical School used rats as their experimental animals and selected diets low in P or Ca or both, in other words diets containing normal and abnormal ratios of these two constituents. Rickets was produced in all animals where the diets were low in Ca or P or both, no matter whether the ratio was normal or abnormal. All these experiments were carried out under conditions of vitamin D defiecency and the result must therefore be regarded as being due partly to the mineral deficiency and partly to a vitamin deficiency—*i.e.* at least two causes, of the relative importance of which we are without information. The Michigan workers studied the pathology of rickets in dairy calves and used natural cases of rickets due to a deficiency of vitamin D. Their results throw little light on the respective rôles of Ca and P, and this also applies to the other American workers, like Harris, who published extensively on human rickets, Maxwell, Hu and Turnbull, Dodds, Cameron and others.

In Europe, pride of place must be given to Marek and his co-workers who have published extensively in this field the results of work that they have undertaken since 1912. For the most part, pigs were used in the earlier experiments; lately cattle have been employed. These workers have attempted more than any others to determine the true relation between Ca and P metabolism and bone diseases. Briefly their conclusions are as follows :—

No aetiological distinction can be made between rickets and osteofibrosis in swine and cattle. Either or both these conditions can be produced by an imbalance of Ca, P, and Mg in the diet. Marek defines narrow limits for these constituents in the diets and provides numerous experimental data to support his conclusions. Marek's work was begun and the first results obtained before the importance and rôle of vitamin D in P : Ca metabolism was known, with the result that throughout his work he hardly mentions the rôle he assigns to vitamin D in his conception of the production of osteodystrophic diseases in animals. However, from the few later experiments in which vitamin D was supplied and in which the animals remained healthy on Marek's abnormal mineral intake, one is

led to the conclusion that Marek's conception is meant to apply only when vitamin D is absent and that the diseases were produced under conditions of vitamin D deficiency, *i.e.* by a mineral deficiency on the one hand and a vitamin deficiency on the other.

However, a study of Marek's results reveals that although he produced rickets or osteofibrosis or both on practically any diet unbalanced in Marek's sense with regard to P, Ca, and Mg, not a single case of osteofibrosis was produced on a diet deficient only in phosphorus. In all cases of P deficiency only rickets was reported. Marek apparently pays no attention to this significant fact. Apart from the evidence that osteofibrosis was not produced by P deficiency, Marek's work is not of great assistance in determining the aetiological difference between rickets and osteofibrosis.

Experimental rickets is usually produced in animals kept on standardized diets low in P, high in Ca, and poor in vitamin D. In other words, it is realized that in addition to low vitamin D, low P in the diet helps to produce dickets; however, it was left to Theiler and his associates to show that rickets could be produced in bovines by phosphorous-low diets in the presence of abundant vitamin D. Subsequent work by du Toit and others has shown that the association of rickets with low P in the diet in the presence of abundant vitamin D applies also to sheep, goats, pigs and probably to horses. This relation may and probably does hold for all species of domestic animals. The general conclusion that P deficiency in the diet has led to rickets in all the species investigated makes the association of rickets and rickets only with P deficiency as definite as can be.

With regard to vitamin D deficiency and the production of bone diseases there appears to be a difference among the species of animals. Although difficult in all species when the diet is properly balanced with regard to Ca and P, it is, however, not impossible to produce low vitamin D rickets in bovines, while it cannot be produced under such conditions in rats. It may be of course—and this probably is the true explanation—that with optimum intake of Ca and P the vitamin D requirements of animals become so reduced that the traces present in the food are suffcient for normal bone-formation in some species, as for instance in rats. Vitamin D is extraordinarily stable, and compounding a ration entirely free from it is practically impossible. In any case, rickets due to vitamin D deficiency is indistiguishable from that due to phosphorous deficiency.

The remaining essential factor required for bone production is calcium, the relation of which to the production of disease will now be considered.

The investigations of Sturgess and Crawford in 1928 in Ceylon, the Japanese work of Niimi and Aoki (1927), and the extensive investigations

of Kintner and Holt (1932) in the Phillipine Islands proved conclusively that the occurrence of osteofibrosis in horses (usually referred to as bran disease, thick head, bone disease, etc.) was closely related to dietary calcium. This disease was subsequently produced at Onderstepoort on diets deficient in calcium or containing high P and comparatively low Ca. A point which Theiler stressed in his London lectures was that although both the Japanese and the Phillipine workers referred to the condition produced as osteomalacia or rickets, he had, from a careful study of their reproductions, no hesitation in substituting osteofibrosis for their osteomalacia. Thus the important point has been established that abnormal Ca metabolism in horses always produces osteofibrosis. This fact and the absence of osteofibrosis in all the cases of phosphorous deficiency produced at this Institute, *i.e.* true rickets or osteomalacia, were responsible for extensive investigations into the effects of Ca-deficiency on animals. This work, although started before Sir Arnold Theiler's recent sojourn at Onderstepoort, could not have been successfully continued without his leadership, initiative, and enthusiasm. These results which I wish to place before you now were all save one obtained during the last few months and therefore unfortunately after Sir Arnold's death.

Bovine No. 6029, 18 months old at the beginning of the experiment, developed osteofibrosis after 19 months in the experiment on a diet low in Ca, high in P, and containing abundant vitamin D. No. 5456—the experimental mate of the animal mentioned, but kept in semi-darkness—developed slight osteofibrosis after approximately the same period. The remaining experimental results may be more shortly summarised as follows :—

No. of Animal.	*Diet.*	*Duration of Expt.*	*Diagnosis*
6421	Low Ca, excess P, and kept in semi-darkness	11 months	Osteofibrosis.
6416	Low Ca, excess P, in light	11 ,,	Severe osteofibrosis.
6414	Low Ca, sufficient P, in semi-darkness	11 ,,	Moderate osteofibrosis.
6413	Low Ca, sufficient P, in light	11 ,,	Severe osteofibrosis.

In spite of the absence of direct light, no rickets, *i.e.* due to vitamin-D deficiency, developed. The feed of these animals, being grown under South African conditions, probably supplied enough vitamin D for their requirements. This conclusion is in agreement with the work of Huffman and others on vitamin-D deficiency in calves.

Bovine No. 6299, placed on the diet which produced osteofibrosis in horses and therefore low in Ca, developed the same disease in 12 months time.

Goat No. 40922, on a low Ca, high P diet, developed slight osteofibrosis after 16 months in the experiment.

Pig No. 1075, on low Ca, high P, was killed 4 months afterwards and the bones showed incipient osteofibrosis.

Other pigs on a slightly higher Ca intake did not develop osteofibrosis after 9 months in the experiment. Further work on the effect of Ca-low diets upon pigs has been started recently.

Osteofibrosis in sheep receiving low Ca diets has not yet been produced.

The last case I wish to mention is the Hereford bullock No. 5430 which received a diet low in both P and Ca and which therefore should (theoretically) develop rickets on account of the P deficiency and osteofibrosis as a result of the Ca deficiency. Judging from the determination of inorganic P and of phosphatase in the blood, this animal showed rickets shortly after the beginning of the experiment. Advanced rickets was diagnosed microscopically from a section of a rib removed 18 months after the beginning of the experiment and, at death last week, severe rickets and osteofibrosis, *i.e.* approximately 2½ years after the beginning of the experiment. Other animals kept for as long a period on a P deficient diet but sufficient Ca showed rickets only.

Both rickets and osteofibrosis are always accompanied by osteoporosis, which, as Theiler states, appears to b a transient stage in the evolution of some disease and merely the result of increased bone resorption—a condition which is actively taking place during the development of both rickets and osteofibrosis. It is a disease which accompanies bone resorption, whatever the cause for such resorption may be, and which is therefore always associated with abnormal Ca : P metabolism and hence with osteodystrophic diseases.

From the foregoing you may have gathered the impression that rickets develops rapidly when animals are placed on a P deficient diet, while osteofibrosis, due to low Ca, takes a great deal longer to appear. That is actually the case, and incidentally probably supplies the reason why Shohl and his co-workers reported rickets only in short-term experiments with rats kept on low Ca diets in the absence of vitamin D. The average time taken by Marek to produce osteofibrosis in addition to rickets was considerably longer than that to produce rickets only. The work at this Institute shows the contrast in the relative periods necessary to produce these two diseases to be a very sharp one, rickets developing almost from the start of the experiment and osteofibrosis after many months, if at all.

This contrast appears to me to find a ready explanation in the

respective functions of the responsible factors, *viz.* P and Ca in the animal body.

Ninety-nine per cent. of the Ca present in the animal body is contained in the bones and only the remaining 1 per cent. takes part in the general metabolic processes of the body. In other words, it would appear that almost all the dietary Ca is destined primarily for bone formation. Hence abnormal bone formation due to Ca deficiency, as in osteofibrosis, might very understandably not be so easily produced as is rickets, which follows a dietary deficiency of the very much more widely functioning prosphorus. Indeed, the needs for Ca, apart from skeletal requirements, are so small and those for P so great that they cannot be similarly considered; and judging from the rapidity with which the P deficiency disease, *viz.* rickets, develops in the skeleton, the conclusion appears justified that the demand for stored phosphorus probably plays as important a rôle in the development of rickets as that for skeletal phosphorus. Incidentally, the low blood phosphorus in aphosphorosis and the normal blood calcium in the case of Ca deficiency support this view, as also the finding that P deficiency is more often responsible for osteodystrophic diseases than is a calcium deficiency, at least under natural conditions of feeding.

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Jl. S.A.V.M.A.
VIII (2) : 98–100
1937.

Aegyptianellosis and Leg-Weakness of the Goose.

By J. D. W. A. COLES, B.V.Sc., Onderstepoort.

Although Brumpt (1930) says that the goose is susceptible to Aegyptianellosis, the literature on the subject is so meagre that it is pardonable to report cases as they occur.

In South Africa the disease has not infrequently been encountered in the fowl, and fatal cases have been noted in the duck.

In November, 1934, a month-old gosling was examined by the author's colleague, Mr. W. O. Neitz, B.V.Sc. It seemed paralysed in the legs; blood smears showed a moderate degree of anaemia and a heavy infection of *Aegyptianella pullorum*. It was seen again after three days, when the parasites were still frequent in the blood, and the bird was *in extremis*.

In January, 1936, the writer received from Migdol in the Transvaal a live Toulouse goose (female) about 4 months old. According to the history, 35 geese had died within two months, the cases occurring at more-or-less regular intervals. The goose was ill and lethargic. The legs were so weak that it could not stand. A blood smear showed slight anaemia, and *A. pullorum* was frequent (even a good example of a schizont was seen). The following day the goose was killed with ether. Apart from slight enlargement of the spleen, the autopsy was negative. Four ducks, 3 months old, were etherised and, together with twelve white mice, were inoculated intracerebrally with a suspension of the macerated brain and spinal cord of the goose. This was done in an unsuccessful endeavour to ascertain the cause of the leg-weakness. But 20 days later two ducks died suddenly, and autopsy revealed moderate *tumor splenis*. Blood smears showed marked anaemia; in one smear *A. pullorum* was numerous; in the other duck the parasites were plentiful only in the blood of the lungs. The other two ducks and the mice remained well. Thus the susceptibility of the duck to *A. pullorum* of the goose was proved. It was established that the premises of the owner of the second goose were badly infested with *Argas persicus*. This was no doubt the vector, as it is in the case of Aegyptianellosis of the fowl.

It is by no means certain that the mortality on the farms or the leg-weakness was due to Aegyptianellosis. From time to time farmers have complained that their geese became paralysed in the legs and died after a few days. Similar reports about ducks are fairly common. In January, 1936, a farmer sent two live Pekin ducks (3 months old) from Leslie in

the Transvaal. The one had its neck turned to the right (torticollis); the legs were extended behind the body and the bird was unable to move; drinking was not impaired. The other was very weak in the legs, but could stumble a few steps occasionally. Both were killed and showed slight *tumor splenis*. A blood smear from one showed anaemia and a few *A. pullorum;* a smear from the second showed only fairly marked anaemia. (Most probably *A. pullorum* was also present.) This is yet another case (this time in ducks) of Aegyptianellosis associated with leg-weakness. It should be mentioned, however, that Aegyptianellosis in ducks unaccompanied by leg-weakness has been seen near Onderstepoort.

At the end of January, 1936, a farmer of Pretoria North brought a sick Pekin duck (6 months old) for examination. According to the history, 25 ducks out of 100 had died during the previous month with similar symptoms. The ducks were running round the farm house and had no access to water. The duck could not use its legs in any way; the wings were very weak; the neck was weak and usually held slightly twisted; the eyes were slightly retracted; drinking was possible, but there was no inclination to guzzle in the water; a blood smear showed very slight anaemia, a fact which was not surprising when it was discovered that the duck-house was heavily infested with *A. persicus;* a few half-engorged *A. persicus* larvae were attached to the skin under the wings. During the night the duck died; the autopsy was essentially negative. Here was a disease of the nervous system, associated with the presence of *A. persicus,* but apparently not with Aegyptianellosis. Numerous parasites were collected, taken to Onderstepoort, and starved for two months.

On each of two occasions, a 4-month-old duck was placed in a glass box overnight with 35 starved adult parasites. During the night most of the ticks fed, and in the morning all the ticks were removed from the box. The two ducks were then kept in cages. Within 6½ days in one case, and 7 days in the other, the ducks became suddenly paralysed in the legs, and were able to move only by lurching forwards and flapping the wings. Only the legs appeared to be affected : the birds were still able to defaecate and to waggle their tails. One duck was killed at once for transmission experiments, which were unsuccessful. Emphasis should not be placed on these failures, because only one experiment of each description was conducted. The disease in the other duck was allowed to run a natural course in the favourable environment of the laboratory, and recovery was complete in a week. Farmers report a recovery rate of about 25%. On no occasion was *A. pullorum* detected in either duck. It should be noted, too, that the incubation period of *A. pullorum* in the fowl after tick transmission is usually 13 days, and the paralysis appeared in the ducks in 6½ and 7 days respectively.

Fowls infested with the larvae of *A. persicus* are often, but not always, somnolent and unable to move. We have seen such fowls recover

when removed from the risk of reinfestation; within 10 days the larvae have all engorged and dropped off and the bird usually returns to normal. Without a thorough investigation of *Argas* paralysis of the fowl, it is unwise to speculate whether the condition in the fowl is the same as that in the duck.

The aetiology of duck paralysis and of goose paralysis, which is probably the same, remains uncertain. If the cause is an intoxication, we have to explain the interesting fact that the leg-weakness develops as long as a week after a single night's exposure to a number of the ticks. It is even possible that a virus is responsible. Whatever the cause, there is strong evidence to suggest that only on certain farms can the *Argas* produce the condition.

Summary.

An *Aegyptianella pullorum* infection has been described in two geese. In one case the organisms were transmitted successfully to two ducks, which died.

Both geese showed pronounced leg-weakness, but evidence has been adduced to indicate that Aegyptianellosis and paralysis are two separate conditions transmitted by *Argas persicus*. It is only natural that both should often appear in one and the same victim.

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JANE ALBERTA AMOS.

We record with deep regret the death on the 25th February, 1937, of Mrs. Jane Alberta Amos, wife of Mr. S. T. Amos, F.R.C.V.S., the President of the Association.

To Mr. Amos and to his daughters, Mrs. I. L. M. v. d. Poel of Johannesburg, and Miss J. E. Amos, M.R.C.V.S., of Durban, we extend our deepest sympathy.

THE ASSOCIATION.

Minutes of Council Meeting held at Polley's Hotel, Pretoria, on 24th March, 1937.

Present: C. J. van Heerden (Chair), A. C. Kirkpatrick, P. J. du Toit, A. D. Thomas, C. Jackson, H. H. Curson, A. M. Diesel, G. v. d. Wath, and S. W. van Rensburg (Hon. Sec.-Treas.).

1. Minutes of Council Meeting held on 28th October, 1936, were read and confirmed.

2. **Arising from these minutes:**

(a) *Scale of Charges for Veterinarians.*—The Secretary read a letter dated 6th July from the N.V.M.A. of Great Britain and Ireland. Decided to refer this question to the Status Committee with power to co-opt, and to report to the next Council meeting.

(b) *Veterinary Organisation in Defence Force.*—A letter dated 3rd December, 1936, from the Adjutant-General was read. Decided to let this matter stand over pending the return to Pretoria of the Minister of Defence.

(c) *Expert Witness Fees.*—A letter dated 9th July, 1936, from the Secretary, Royal College of Veterinary Surgeons, was read. Decided to refer this matter to the Status Committee for investigation and report.

(d) *Cruelty to Animals.*—In view of the absence of the President (Mr. Amos), discussion on the Committee's report was postponed for a subsequent meeting.

3. **Courtesy Title.** There was considerable discussion as to whether voting in the referendum should be extended to all registered veterinarians or be confined to members in good standing. It was indicated that a referendum which violated any of the rules of the Constitution would not be binding. It was therefore decided to conduct the referendum strictly in accordance with the Constitution; to issue the résumé on the discussion at the General Meeting with Mr. Viljoen's memorandum and Faculty Committee's report not later than 16th April and voting papers on May 1st; and to send out stamped addressed envelopes with the voting papers.

4. **Public Servants' Association.** The Secretary reported on the proceedings of a meeting of the Protechnical Section of the P.S.A. held on 3rd February, 1937, at which Dr. Mönnig and he represented the veterinarians in the Service. He stated that they had strongly opposed the suggested application to veterinarians of a scale on which both the

commencing salary and the increments are lower than those pertaining to the present scale.

In view of subsequent events it was decided that no further action be taken.

Dr. du Toit reported that a new scale of £400 x 25—£800 *p.a.* with an efficiency barrier at £700 for all veterinarians in the Service had been approved by the Public Service Commission. Council passed a unanimous vote of thanks to Dr. du Toit for his services in this connection.

5. **New Members.** The following were proposed : J. W. A. Brooks, C. W. A. Belonje, H. P. de Boom, M. de Lange, W. J. B. de Villiers, D. A. Haig, J. L. Mainprize, M. J. N. Meeser, G. D. Sutton, S. J. v. d. Walt. Unanimously decided to recommend their acceptance to the next General Meeting.

6. **Sale of Akiron.** A letter dated 14th February, 1937, from Messrs. Bayer Pharma (Pty.), Ltd., was read in which the difficulty experienced by stock-owners in some districts in getting qualified veterinary assistance was indicated. It was decided that there would be no objection to the sale of the drug direct to stock-owners in such cases, though the latter should be warned regarding the toxicity of the drug and the importance of a correct diagnosis.

7. **General Meeting.** A committee consisting of Drs. du Toit, Jackson and Thomas, Mr. van Heerden and the Secretary was appointed to arrange the date and programme for the next General Meeting.

8. **General.** (a) *Reciprocity.*—Dr. du Toit reported that reciprocity with Great Britain had definitely been accomplished and that a notice to that effect appeared in the Government Gazette of 19th March, 1937.

(b) *Australian Veterinary Association.*—The Secretary submitted two copies of the *Australian Veterinary Journal* giving a description of the coat-of-arms adopted by the A.V.A.

(c) *Complaint, Mr. C. M. Sharpe.*—The Secretary submitted copies of correspondence which has passed between Mr. Sharpe, the Secretary of the Veterinary Board, and himself. He stated that the case in question was a good illustration of the many loopholes contained in Act 16 of 1933.

Dr. du Toit declared that the Veterinary Board hoped to get several amendments to the Act passed next year.

(d) *Dr. K. Schulz.*—A letter from Dr. Schulz dated 15th March, 1937, was read, asking Council to assist him in pressing his claim for expenses in connection with the shooting affair at Danielskuil. Resolved to ask Dr. Schulz for a copy of the claim submitted by him and of the relative correspondence.

(e) *Post-Graduate Degree.*—The Secretary submitted a letter dated 23rd February, 1937, from Mr. R. Clark suggesting that representations be made with a view to establishing a post-graduate degree obtainable on examination. Decided that this be referred to the Faculty of Veterinary Science.

(f) *Training of Natives.*—Correspondence between the Director of Veterinary Services and Dr. Curson regarding the training of natives in veterinary science in Uganda was submitted and recorded.

(g) *Mr. A. Hodder.*—Mr. Diesel produced personal papers belonging to Mr. A. Hodder which he found in a Maritzburg office. These were handed to the Secretary for transmission to Mr. Hodder.

The meeting concluded at 11 p.m.

FACULTY OF VETERINARY SCIENCE.

Number of Graduates.

The following data relating to the number of students who have qualified as veterinarians at Onderstepoort during the period 1924—1936 are published for general information.

1924	8	1931	5
1925	10	1932	8
1926	6	1933	2
1927	8	1934	4
1928	5	1935	7
1929	6	1936	13
1930	0	**Total**	**82.**

JOHN MARSHALL ROBINSON.

We regret to record the death on the 11th April, 1937, of Mr. J. M. Robinson at the age of 59 years. The late Mr. Robinson had served in the Boer War and was a Registered Veterinary Surgeon in practice in Pretoria.

BOOK REVIEW.

In the 3rd Edition of Milk's Practical Veterinary Pharmocology Materia Medica and Therapeutics * many more drugs and compounds are discussed than in the 2nd Edition. Among the newly included drugs are allantoin, amytal, artificial maggots, avertin, benzedrin, carotene, coramine, evipal, liver extracts, pentobarbital sodium, pine oil, disinfectants, pyrethrin, rotenone, several iodine preparations and salicylates. There is also a chapter on biological therapeutics, which contains very valuable information.

The text has been revised and where necessary the information has been brought up to date.

In the latest edition Milks again prescribes 5.0 – 10.0 c.m. carbon tetrachloride for sheep and 10.0 – 30.0 c.m. for cattle. These are highly dangerous doses. We know the quantities of carbon tetrachloride as small as 2.0 c.m. for sheep and 5.0 c.m. for 2-year old bovines have caused poisoning and even death.

Milk's Pharmacology has previously been reviewed in this Journal, hence it is unnecessary to go into the details of the book. It is a most useful book both for qualified practitioners and veterinary students. It is a well-bound and well-printed work, put up in a most concise form and embodying a large volume of very useful and practical information. The arrangement of the different groups of drugs is according to the system on which they primarily exert their effects. This is the most suitable of all arrangements, especially as far as the veterinary student is concerned, as the actions of the different drugs and compounds can be more easily memorised.

D. G. S.

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* *Practical Veterinary Pharmocology, Materia Medica and Therapeutics.* By H. J. Milks, D.V.M. 3rd Edition, 1937. Pp. xvi + 582, Illustr. 32. Baillière, Tindall & Cox, London. Price 30/-.

Jl. S.A.V.M.A.
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1937.

Pasteur on Anthrax.

Abstracted by J. H. MASON.

Scientific workers are deep in the debt of Dr. Pasteur Vallery-Radot for the trouble he has taken and the patience he has shown in assembling all Pasteur's publications *. Six volumes have already been issued : Vol. 1, " Molecular Dissymmetry" (pp. 480), Vol. 2, " Fermentation and Spontaneous Generation " (pp. 664), Vol. 3, " Studies on Vinegar and Wine " (pp. 519), Vol. 4, " Studies on Silk-worm Disease " (pp. 761), Vol. 5, " Studies on Beer " (pp. 361), and Vol. 6 (in 2 parts), " Virulent Diseases, Virus-vaccines and the Prophylaxis of Rabies " (pp. 906). A seventh volume, "Scientific and Literary Miscellany," has yet to appear. The compiler's task of searching the literature, correcting typographical and other errors, arranging the articles, letters, and addresses in chronological order, and correcting proofs must have been enormous, but has been well worth while. It is no longer necessary to delve into musty volumes of the *Comptes rendus de l'Academie des Sciences* : in a matter of minutes the article and information sought can be found.

Most South African veterinarians will agree that anthrax is one of the most serious diseases affecting livestock in this country, both from the administrative and the economic aspect. That nearly 8 million doses of preventive vaccine were issued in the year 1936 proves that the Union Department of Agriculture dreads it and is willing to go to no little trouble and expense to prevent its appearance in the herds and flocks of the country. Like that of most veterinary surgeons, my acquaintance with Pasteur's contribution to the solution of the anthrax problem had been derived from lectures at college on bacteriology, from text books and from biographies of Pasteur. I had formed a hazy picture of the man and had gained the impression that he was a genius, but a lucky genius. On reading his anthrax work carefully, and skimming over some of his research on fowl cholera, rabies, and variola, I was left with a feeling of awe for the man—a dynamic personality, a tireless worker both with hand and brain, a fearless being with penetrative insight, a genius, lucky perhaps, but if lucky, then no luckier than he deserved to be. I knew Sir Arnold Theiler only during the later and mellower days of his life, and therefore rely on the statements of others for information regarding his character, conduct, and directive and research ability of earlier days. But on almost every page of Pasteur's work on anthrax I could see Sir

* *Oeuvres de Pasteur* réunies par Pasteur Vallery-Radot, 1922–1933. Paris : Masson et Cie.

Arnold—eager, working all the time, not sparing his assistants but sparing himself less, fearless in face of apparently insurmountable odds and—always planning for the future. As Dr. A. Pijper *, in his excellent appreciation of Theiler, says : ". . . I think his mentality was more French than anything else."

The short abstracts that follow are arranged in the order chosen by Dr. Vallery-Radot. Some repetition occurs and no attempt has been made to correct this, because what Pasteur wished to emphasise during his many addresses to the various academies or societies will still bear repetition. It must be kept in mind that he had something revolutionary to present to the world, that he lectured at many gatherings and in different places : to condense all his statements and arguments so that they could be presented in a 1 – 2 – 3 fashion would detract from the reader's appreciation of the struggle he had in making the world accept his work.

The titles of the articles are given in English as close to the French as I am capable, with the year of presentation, the page of volume 6 in which they appear, the name of any collaborator, and the society at which they were given. Some communications have not been abstracted either because I considered them of insufficient interest or because they were almost reiterations of former articles.

Study on Anthrax.

(1877, p. 164, with Joubert, *Acad. Sciences.*)

In 1850 Rayer and Davaine (two Frenchmen) noted the presence of minute rods in the blood of anthrax-infected animals and showed that blood from the spleens of sheep dead of the disease set up anthrax after inoculation into other sheep. This was the first occasion on which the anthrax bacillus was seen.

Pasteur himself first noted sporulation in the microbe causing silk-worm disease and in 1876 Koch described the spores of the *B. anthracis.*

Pasteur discusses the existence of micro-organisms and the possibility of their causing disease. He says : A drop of anthrax-infected blood, containing red and white cells and bacilli is injected into an animal, which dies in from one to four days and its blood also contains these bacillary elements. Is it the bacilli which reproduce the disease or other blood elements which accompany these germs ? To check the point, Pasteur sub-cultured anthrax bacilli many times and, with his final culture, was able to set up anthrax in an animal, whereas filtered culture was innocuous. His conclusions are that *B. anthracis* can grow indefinitely in artificial media without losing its action on the animal body; in such

* Pijper, A. (1936). S.A. Med. J. Vol. 10, p. 627.

circumstances it is impossible to admit of its being accompanied by a soluble substance or virus, sharing in the causation of anthrax.

Anthrax and Septicaemia.

(1877, p. 172, with Joubert, *Acad. Sciences.*)

Pasteur notes that Paul Bert (the noted French physiologist) doubts the rôle attributed to the anthrax bacillus in the causation of the disease. He recalls the experiment in which he subcultured the germ many times and was able to set up the malady with the final culture, whereas the filtrate was unable to do so. However, Paul Bert actually inoculated a guinea-pig with a culture supplied by Pasteur and killed the animal in 30 hours; the blood of this cavy, extremely virulent as proved by inoculation, lost all virulence after one week in an atmosphere of compressed oxygen or after the action of concentrated alcohol. Pasteur gives the reason for this. The spores, much more resistant to desiccation and to heat than the vegetative elements, are not found in the animal body immediately after death, whereas a culture in urine contains a superabundance of them after a few days' incubation. By experiment he shows that spores withstand 10 to 12 atmospheres of oxygen for at least 21 days whilst the vegetative bacillus is easily destroyed by such treatment.

The dark colour of the blood of anthrax carcases is attributed to the usage of oxygen by the germ, because being an aerobe it requires much more of this gas for its metabolism than do those microbes which flourish in the absence of air (anaerobes). The agglutinative character of the red cells is caused by a diastase formed by the bacilli.

It is remarkable that one may inject large volumes of anthrax culture into the circulation of hens without producing disease, whereas, *in vitro,* the blood (or better the serum) will support growth. Again, one is not always successful in infecting guinea-pigs by intravenous inoculation, although their blood can form a culture medium. Pasteur notes that life is antagonistic to life and bacteria in the circulation have a struggle to obtain oxygen in competition with the red cells. Further, the sowing of the anthrax bacillus along with a common aerobe in the same tube of culture medium prevents an exuberant growth of the former germ and the inoculation of such a mixture into a susceptible animal may fail to set up the disease.

Pasteur answers the objections of some workers, who send to knackeries for anthrax blood and, having inoculated it into an animal, kill it in a very short time without *B. anthracis* appearing in the blood. They hold that the presence of the germs in the circulation is merely an epiphenomenon. Pasteur's reply is that blood taken immediately after death contains only anthrax bacilli, whereas if the cadaver is 2 to 3 days old, putrefactive germs have invaded it and, being anaerobes, in an anaerobic situation will outgrow and destroy the anthrax germ. The

inoculated animal dies, not of anthrax, but of septicaemia (gas gangrene). This hypothesis was put to the test using blood from a six hours dead animal and from animals dead for from 24 to 72 hours. The results proved that Pasteur was correct.

It is interesting to note that, in this communication, the *Vibrion septique* (a cause of gas gangrene and the cause of braxy of sheep) is described and, among other things, its ability to form filaments on the peritoneal surface of animals into which it is inoculated is noted.

Discussion on the Aetiology of Anthrax.

(1877, p. 189, *Acad. Méd.*)

Toussaint, Professor of Physiology and Anatomy at the Toulouse Veterinary College, had given a reading at the Académie des Sciences. He inserted the point of a vacuum tube into a vein of an anthrax-infected animal, broke the point off in the vein, allowed blood to run in and, on withdrawing, sealed it off. Blood obtained in this fashion was free from air, and in such conditions anthrax bacilli die off. Bouley (the chairman at this meeting) wrote to Pasteur saying that this proved there is no virus agent in addition to the bacteria, but reminded Pasteur that Colin (a professor at the Alfort Veterinary College) had noted the virulence of blood in the absence of microscopically demonstrable bacilli. Bouley is apparently swayed by Colin in spite of what Pasteur has previously said and proved.

Pasteur replies in a letter. He accuses Bouley of little faith and says that Colin has not taken cognisance of the results obtained with successive cultures in urine. If one subcultures 100 times, the final dilution (assuming each sowing dilutes 10 times) is 10^{100}. It is ridiculous to contend that the virulence of the final culture is due to an agent carried over from the original inoculum (a drop of blood). Colin says that filtration (of blood) through plaster can alter ferments, diastases, and soluble virulent agents and so render the filtrate innocuous. Pasteur asks why he does not mix virulent material with plaster and inject it into an animal—he would have his answer in 24 hours. Apparently Colin wishes to believe in his virulent soluble agent and refuses to consider the experiments which prove definitely the absence of such an agent. He notes that if an animal is infected with anthrax, its blood will prove to be infective after some hours but microscopically no bacilli are demonstrable. Pasteur has to point out that, even although no organisms may be *seen* by microscopical examination, culture will prove their presence.

Discussion on the Aetiology of Anthrax.

(1878, p. 194, *Acad. Méd.*)

Once again Pasteur points out to Colin that the inability to *see* the anthrax bacillus in blood is no proof of its absence. He repeats his request

for culture work and asks that a commission be set up to prove or disprove Colin's hypothesis of " anthrax without bacilli."

Discoveries relative to Anthrax.

(1878, p. 197, *Bull. Soc. cent. agric. de France.*)

This is more or less a repetition of the article " Study on Anthrax ". The bacilli must be the cause because the hundredth subculture in artificial medium can reproduce the disease. That an ultramicroscopic reproductive agent co-existing with the bacteria is not the cause is proved by the innocuity of a filtrate of the hundredth subculture.

Pasteur notes that fowls cannot be successfully inoculated with anthrax and attributes this to the higher temperature of birds. However, in an addendum to this address delivered a week later, he points out that they become susceptible if their temperature is artificially lowered. He suggests that the raising of the temperautre of an infected person to 104° – 107° F. might have a beneficial effect.

Discussion on the Aetiology of Anthrax.

(1878, p. 202, *Acad. Méd.*)

This séance of the *Académie de Médecine* is in the nature of a dog-fight between Pasteur and Colin. Once again, the ability or inability to produce anthrax without germs and the insusceptibility of the fowl are thrashed out. Eventually a commission is named to investigate the matter.

Discussion on the Aetiology of Anthrax.

hens successfully inoculated.

(1878, p. 210, *Acad. Méd.*)

Pasteur exhibits three hens, one dead of anthrax and two alive and healthy. The first had received culture and was then partially immersed in cold water and died of the disease. The second had received culture but its temperature was not lowered and the temperature of the third had been lowered but no culture was given—both lived.

The second part of the meeting is taken up by a severe chastisement of Colin by Pasteur, Colin refusing to reply.

Anthrax in the Hen.

(1878, p. 215, *Acad. Sciences.*)

(With the collaboration of Joubert and Chamberland.)

Ordinarily the hen is refractory to anthrax, but after reducing the temperature by immersion in cold water it becomes susceptible. If removed from the cold water, some time (actual time not given) after inoculation it does not develop the disease, but cure is not effected by this means when the blood is already invaded by bacilli, in the last hours of the bird's life.

Observations on a note presented by M. Colin.

(anthrax of the hen.)

(1878, p. 216, *Acad. Méd.*)

Colin had inaccurately repeated Pasteur's experiment of infecting birds with anthrax when their temperature was lowered by partially immersing them in water at 25° C. Colin used water at 39° C. instead of at 25° C. and in the single experiment mentioned, he failed to set up the disease. Colin says that he feared the bird would die of cold — Pasteur's reply was : " The first thing to do was to let it die and then examine it." In Pasteur's opinion Colin was really afraid of light and truth.

Aetiology of Anthrax.

(1878, p. 218, *Acad. Méd.*)

Colin had written a note saying that he would have been happy to have seen the anthrax germs in the bird which Pasteur showed to the Academy. Pasteur did not take the bird out of the cage and merely reported the findings of the autopsy and the microscopical examination.

Pasteur is righteously indignant and a commission is set up. Pasteur is to present a fowl dead from anthrax and Colin in the presence of Pasteur and other members of the Academy is to carry out an examination and report thereon. The commission already appointed to investigate Colin's former statements is appointed for this purpose also.

Aetiology of Anthrax.

[(report of the commission) hens inoculated with anthrax.]

(1878, p. 219, *Acad. Méd.*)

Four birds were delivered by Pasteur, three dead after inoculation and cold immersion and one alive after inoculation and cold immersion and subsequent warming up to 42° C. in an incubator.

Bacilli were found at the point of inoculation, in the comb, in the blood of an external vein and in the heart blood. All members of the commission, including Colin, signed a declaration to this effect.

Discussion on the Causes of Death in Anthrax and Septicaemic Conditions.

(1878, p. 222, *Acad. Méd.*)

Once again Pasteur is at Colin, this time because Colin does not invoke, in a new publication, an " anthrax virus " as the cause of death in anthrax. " Has M. Colin renounced the idea of an anthrax virus " ? asks Pasteur. The reply is unsatisfactory; Colin equivocates and one can picture him wriggling uncomfortably in his chair.

What appears to be a very amusing incident took place at this meeting. Colin rises after Pasteur and says : " I am very much at a loss

on the decision I must take on the subject of the questions that M. Pasteur has just asked; I am bound by the promise that I made of not arguing with him, but if the Academy will be good enough to free me from the bond, at least for to-day, I will reply." The Chairman replies : "You bound yourself quite on your own M. Colin, unbind yourself."

RESEARCH ON THE AETIOLOGY AND PROPHYLAXIS OF ANTHRAX IN "LE DÉPARTEMENT D'EURE-ET-LOIR." REPORT TO M. TEISSERENC DE BORT, MINISTER OF AGRICULTURE AND COMMERCE.

(1878, p. 225.)

Pasteur has been asked to investigate anthrax in the Eure-et-Loir district where the losses in cattle are very high. With him he has Chamberland and Vinsot, the latter a pupil of the Alfort Veterinary College, and he often seeks advice of a M. Boutet, a county councillor and his son, a veterinarian. He records that it is not easy to set up the disease by feeding lucerne water with culture, even if material such as barley awns is added.

He notes that in naturally-infected animals the lesions are found chiefly in the first portion of the digestive tract and it is here that they are most advanced.

For prevention, he advises the elimination of foods likely to injure the mouth or intestinal mucous membranes, and in addition to see to the proper disposal of anthrax carcases.

[*Note* : A footnote of interest to veterinarians is added to this report in connection with Dr. Davaine (a medical man) who first saw anthrax bacilli. It runs : M. Moisant, veterinarian, a member of the Eure-et-Loir county council, assured me recently that M. Delafond, professor at the Alfort College, taught from the year 1838 that microscopic rods were present in the blood of anthrax-infected animals. As far as I know, this is not mentioned in the works of this professor prior to 1850, the date of Davaine's observations. M. Delafond must have attached very little importance to this fact, although mentioning it every year to his students.]

THE AETIOLOGY OF ANTHRAX.

(1879, p. 230. With Chamberland and Roux, *Acad. Méd.*)

Pasteur mentions his report to the Minister of Agriculture, in which he suggests that anthrax bacilli are spread in or on the ground by anthrax-infected beasts, either before or after death. To prove the point, anthrax blood was added to soil and this was alternately watered and allowed to dry. Spores were formed and these could be demonstrated months later. In a second experiment, a sheep dead of anthrax was buried and, ten months later, the soil from the grave produced the disease when it was injected into guinea pigs. Pasteur concludes by saying that the reason for the high incidence of the disease in the Eure-et-Loir district is the heavy soil infection.

AETIOLOGY OF ANTHRAX.

(1879, p. 232. *Acad. Méd.*)

Again Pasteur chastises Colin, but on this occasion he appears tired of it all. Colin *will* contradict him although he cannot bring sufficient *bona fide* evidence forward.

M. Bouillaud, a medical man, praises Pasteur's work, but his compliments have a sting : Yes, Pasteur has exploded the spontaneous generation of disease hypothesis by actual experiment; but still, for a long time, it has been known that disease did not arise spontaneously. Induction and reason arrive at the truth sometimes just as well as experimentation. He himself, in 1860, said that glanders was an infectious disease, and that no ordinary cause could produce the disease; to become infected with it, the disease must be present. At this point a M. Pidoux interrupts to say that overcrowding of stables can bring about the disease without contagion being demonstrated. This gives Pasteur his opening to say that there do exist medical men who are partisans of the hypothesis that transmissible diseases arise spontaneously. It is these people and their doctrine that he attacks.

AETIOLOGY OF ANTHRAX.

(1879, p. 238. *Acad. Méd.*)

Pasteur has shown that anthrax spores will live for a considerable time in soil. Colin denies this. The meeting is taken up with a denunciation of Colin and his methods.

AETIOLOGY OF ANTHRAX.

(1879, p. 241. *Acad. Méd.*)

This somewhat longer article is taken up with an attack on Colin, his methods and his ill-founded criticisms of Pasteur and with an attack on the partisans of spontaneous generation. He records an interview with an Englishman, a Dr. Bastian, who wrote to the Academy saying he had found the physio-chemical conditions necessary to allow germs to appear in neutral urine held at 50° C. Pasteur was able to point out the error of Bastian's ways when he advised him to sterilize his flasks. etc., before use. Bastian admitted that he never did this. (It must have been very tiring for Pasteur to have to point out such elementary principles, known to most intelligent modern schoolboys. As it would appear he had to do this weekly, one can sympathise with his somewhat caustic remarks.)

RESISTANCE OF THE ANTHRAX BACILLUS TO COLD.

(1879, p. 253. *Acad. Sciences.*)

Notes that both the anthrax bacillus and that of fowl cholera can resist a temperature of 40° below zero.

The Aetiology of Anthrax.

(1880, p. 254. With Chamberland and Roux, *Acad. Sciences.*)

The first part of the meeting is taken up by a discussion on the spontaneous generation of disease and with Davaine's observation, in 1850, of "thread-like" corpuscles in anthrax blood.

Animals become infected by ingesting germs, but artificially it is not always easy to demonstrate this. Sheep fed with lucerne charged with enormous numbers of anthrax spores did not all die; many escaped death, although visibly ill. Others, but a smaller percentage, died with all the symptoms of anthrax after an incubation period of 8 to 10 days. The mortality may be increased if one mixes sharp-pointed material, such as barley awns, with the contaminated food.

Experiments are quoted proving that anthrax is spread from soil contaminated from the burial of infected carcases. Although, in the unopened buried cadaver, the bacillary form of the anthrax germ dies off, spilled blood is usually buried along with the animal and thus sporulation is permitted. In addition, it is recorded that earthworms can carry spores to the surface of the ground.

The Aetiology of Anthrax infections.

(1880, p. 264. Letter from Pasteur to M. Dumas, *Acad. Sciences.*)

Pasteur points out that the burying of anthrax carcases spreads the disease and quotes an experiment in which four sheep were enclosed in a small area around a two-year-old anthrax trench and four others in a space 3 – 4 metres away from this spot. In seven days, one of the first lot of sheep died of anthrax, whilst all the controls were healthy.

[*Note* : This letter was written on 27.8.1880 and the sheep died on 25.8.1880; it is obvious that sufficient time had not elapsed to allow of conclusions being drawn.]

New observations on the Aetiology and the Prophylaxis of Anthrax.

(1880, p. 266. Read in name of Pasteur at *Acad. Sciences.*)

A letter dated 1865 from Baron Seebach to the Minister of Agriculture and Commerce was read. In it, it was pointed out that Baron Seebach had observed that outbreaks of anthrax were associated with soil in which anthrax carcases had been buried. Both the earth from such ground or food grown on it could set up the disease and this observation was so often confirmed that the Baron had all dead animals buried in a separate field, surrounded by a ditch and a fence. This was begun in 1854 and it is interesting to note the death-rate from the disease before and after this innovation.

1849–1854	—	15–20% per year,
1854–1858	—	7% ,, ,,
1860–1864	—	5% ,, ,,
In 1863	—	3% ,, ,,

Pasteur enlarges upon these results and mentions another experiment in which four sheep were quartered over an anthrax trench and to their food barley awns were added. Two died of anthrax, whereas four other sheep, kept in an area some distance from the trench, remained healthy.

The Longevity of Anthrax Spores and their Persistence in Cultivated Soil.

(1881, p. 271. With Chamberland and Roux, *Acad. Sciences.*)

Pasteur was able to kill guinea-pigs with anthrax by inoculating them with soil taken from the surface of a trench used twelve years previously for burying animals. Anthrax was endemic on the farm. In addition two of seven sheep which were allowed access to this old trench (bare of herbage) died of anthrax.

Anthrax Germs in the Soil.

(1881, p. 275. *Acad. Méd.*)

Once again Pasteur has to rebuke Colin, who cannot demonstrate anthrax in soil. Pasteur says : "If I take a sod of earth and demonstrate anthrax in it, it is there, and if I place this same sod in M. Colin's hands and he does not find it, it is evident that he is making a mistake. One road leads to truth, a thousand to error. It is always one of these latter that M. Colin chooses."

The remainder of this meeting and one which took place a week later were devoted to argument between Colin and Pasteur. Colin is at pains to point out that Baron Seebach had said that a cow and a goat had died from anthrax the day after eating food from an "anthrax field". Pasteur quotes from the Baron's letter and shows that Colin is wrong. It is difficult to see Colin's object in all these puerile contradictions and interruptions. One is forced to think that they were due to personal enmity and jealousy.

The demonstration of Anthrax Spores in the Soil from the surface of Trenches in which Anthrax-infected Animals have been Buried.

(1881, p. 282. With Chamberland and Roux, *Acad. Méd.*)

Owing to the presence of other germs in soil it is difficult to demonstrate anthrax bacilli in it. This difficulty can be overcome as follows. The soil is washed several times in alkaline water and the finest deposits heated at 90° C. for 20 minutes. A constriction is made in the lower third of a tube and well-washed pieces of marble are put in it so that they fill the upper two-thirds of the tube. One or two holes made in the tube just above the constriction allow the circulation of air among the pieces of marble. The washed heated soil deposit is diluted with a little sterile alkaline water, the marble fragments moistened with this and the tube

incubated at 30° – 35° C. After a few hours, the pieces of marble are washed with a little water and this is inoculated into guinea-pigs or rabbits. The use of the marble fragments offers to the anthrax spores a large culture surface with plenty of air. Anaerobes will not grow under such conditions so that the anthrax spores have every opportunity of germinating. Another and perhaps better method is to incubate the washed heated soil deposit in yeast-water at 42° – 43° C. At this temperature, in yeast-water, the spores of anthrax do not develop, whereas a rich culture of many soil germs occurs. The culture is heated to 75° C. in order to destroy vegetative elements, and thereafter inoculated into guinea-pigs and rabbits.

Soil from anthrax-free farms, treated as above, does not set up anthrax, but very frequently septicaemia (gas gangrene).

Experiments Tending to show that Hens Vaccinated against Fowl Cholera are Refractory to Anthrax.

(1880, p. 315, letter to M. Dumas.)

The bacillus of fowl cholera does not grow in filtered broth in which it has previously been cultivated. The body of an immune bird Pasteur likens to such exhausted broth. Further, the anthrax bacillus grows very poorly in broth, filtered after supporting the growth of the fowl cholera bacillus. This observation suggested that cholera-immune birds might be immune to anthrax and in a small number of experiments the hypothesis was confirmed. However, Pasteur admits that he has only a few experiments and is more enthusiastic than dogmatic about it.

The Non-recurrence of Anthrax.

(1880, p. 316. With Chamberland, *Acad. Sciences.*)

Pasteur proves by experiment that bovines, recovered from an artificial attack of anthrax, are immune to the reinoculation of culture.

Chauveau of the Lyons Veterinary College has noted that Algerian sheep are much less susceptible to anthrax than are French sheep and that this immunity is improved by an inoculation of culture. Chauveau believes that the immunity is due to substances harmful to the proliferation of the anthrax bacillus. Pasteur says that the immunity of Algerian sheep is due to an effect of constitution, to a vital resistance. Pasteur quotes the insusceptibility of the hen—one cannot invoke the presence of harmful substances because cooling of the body renders the bird susceptible.

The Attenuation of Viruses and their Return to Virulence.

(1881, p. 332. With Chamberland and Roux. *Acad. Sciences.*)

Pasteur notes that the method of attenuation applied to the fowl cholera bacillus cannot be applied to the anthrax bacillus because the latter forms spores. One can prevent the formation of spores by culturing at 16° C. when involution forms are obtained or at 42° – 43° C. when

cultivation is easy with abundant growth. After 8 days and up to 1 month at 42° – 43° C. the culture is inoffensive for the guinea-pig, rabbit and sheep and at the end of a month it is dead. Thus, at will, one can furnish oneself with cultures of different degrees of virulence and as with fowl cholera a method of preventive inoculation is provided. Although such an attenuated culture will not kill a full-grown or even a six-day-old guinea-pig, it is lethal for a one-day-old cavy. By passage through older and still older animals a culture, returned to full virulence, may be obtained.

Pasteur then discourses on the origin of epidemics, suggesting that germs are widespread and that man, for example, carries them in his intestinal canal without any trouble arising. However, the microbes are ready to become dangerous when, through conditions such as overcrowding, lowering of body vitality, famine, etc., they have the opportunity of regaining virulence.

The Possibility of Making Sheep Refractory to Anthrax by the Method of Preventive Inoculation.

(1881, p. 339. With Chamberland and Roux, *Acad. Sciences.*)

M. Toussaint, a Professor at the Toulouse Veterinary College, had published a report in which he had stated that sheep could be rendered immune to anthrax by inoculating them with anthrax blood filtered through several layers of paper, or with blood heated at 55° C. for 10 minutes. Pasteur does not question the truth of this observation but attacks Toussaint's explanation. This is that the anthrax bacillus, multiplying in the animal body, produces a material which can become its own appropriate vaccine. By filtration or by heat, one removes the germ, and the inoculation of blood, so heated, introduces into the animal body the vaccinating substance deprived of bacteria. Pasteur notes that his attenuated fowl cholera culture, deprived of bacteria, does not immunize. (It is interesting to note that Pasteur received notice of Toussaint's work whilst on holiday and wrote immediately to Chamberland and Roux, also on holiday, informing them that they must give up all idea of a country vacation. This they accepted with their usual self-denial.) Experimentation by Pasteur and his "young collaborators," Chamberland and Roux, showed that heating at 55° C. might or might not kill anthrax cultures; if they were killed they had no immunizing value; if they were not killed, two results could be obtained. Firstly, the sheep which received them might be killed, secondly the sheep could survive and therefore be immune. (M. Toussaint abandoned his hypothesis when he heard this result.) Pasteur rightly points out that Toussaint's method is unsatisfactory—one could not be certain of preparing his heated blood vaccine so that it was, at one and the same time, safe and capable of immunizing and secondly there was the great difficulty of obtaining anthrax blood in quantity.

The Vaccine of Anthrax.

(1881, p. 343. With Chamberland and Roux. *Acad. Sciences.*)

Pasteur repeats that by cultivating the anthrax bacillus at 42 – 43° C. in chicken broth one can reduce its virulence. Beginning with a very virulent strain, he maintained it alive for 6 weeks at 42° – 43° C. After eleven days, it did not kill adult guinea-pigs, and on the 31st day it was still virulent for very young mice but not for guinea-pigs, rabbits or sheep. On the 43rd day it was avirulent for mice and even for cavies a few hours old. In a culture of such a very attenuated strain the bacterial filaments are short and a uniform deposit is formed in the flask, whereas in a culture of a virulent strain long filaments and fluffy flocculi are seen.

Summarised Account of the Experiments made at Pouilly-Le-Fort, near Melun, on Anthrax Vaccination.

(1881, p. 346. With Chamberland and Roux. *Acad. Sciences.*)

Pasteur summarises the results already noted by M. Rossignol. He enumerates many of the people present and names 15 of the veterinarians who attended. These, from being sceptics and scoffers, became ardent apostles and one even went so far as to wish to have himself vaccinated.

In the discussion which followed one member doubts that the death of the fœtus in the one vaccinated ewe which died could have been the cause of death. Pasteur notes that there were no lesions of anthrax and that blood smears revealed only a very occasional bacillus.

Colin claims that he was the first to speak on preventive inoculation against anthrax and the first to have established that small quantities of anthrax virus conferred immunity. Pasteur asks him to produce his work and accuses him of a scientific and an historical error. Further Colin asks why Pasteur has not used anthrax blood to test immunity and Pasteur offers to allow him to use such blood on the Pouilly-Le-Fort animals. The offer is not accepted. Colin maintains that cultures of the anthrax bacillus are not of value in testing immunity because one he received from Pasteur is no longer pathogenic. Pasteur accuses him of having contaminated it and of having lost the strain. Colin doggedly maintains that he places reliance on immunity tests with virulent blood.

The Virus-vaccines of Fowl Cholera and of Anthrax.

(1881, p. 358. *Congrès International des Directeurs des Stations Agronomiques, Versailles.*)

Pasteur discusses the attenuation of the fowl cholera germ and then goes on to describe the formation of spores by the anthrax bacillus. The virulence of the spore is the same as that of the vegetative element : *i.e.*, if one cultivates bacilli from virulent blood and allows spore formation to take place, the virulence of such a culture is the same as that of the blood. He then explains the attenuation of anthrax cultures at 42° – 43° C. and

the immunization of sheep with such material. The spore originating from attenuated cultures preserves the same degree of virulence as its parent bacillus, so that a strain may be held for a long time in a definite stage of attenuation. The epoch-making experiment at Pouilly-Le-Fort is then summarised briefly, and Pasteur records that since then requests for vaccine have been received daily. The previous week, orders for vaccine for 1,200 sheep and more than 100 cattle had been received and M. Rossignol, the veterinary surgeon on whose farm the big experiment was conducted, had had requests for material for 10,000 sheep.

Immunity in sheep had been shown to last for at least 6 months.

Vaccination in relation to Chicken-Cholera and Splenic Fever.

(1881, p. 370. *International Congress of Medicine*, London.)

Pasteur gives a résumé of the attenuation of the fowl cholera germ, noting that this is brought about by sub-culture at somewhat long intervals. Rapid sub-culturing does not lead to this attenuation. He then describes spore formation in the anthrax bacillus, the production of a vaccine, and the immunization of sheep with such a preventive.

Address to the Agricultural Society of Melun.

(1882, p. 382.)

It will be recalled that the epoch-making vaccination experiment of Pouilly-Le-Fort was carried out at a farm near Melun. Pasteur had written to the president of the Melun Agricultural Society suggesting that an experiment should be carried out to determine the duration of immunity in the vaccinated animals. The results of this experiment are given out at this meeting, together with the results of routine immunization in the field.

Before Pasteur speaks the president presents him with three medals, one in gold, one in silver and one in bronze to commemorate the Pouilly-Le-Fort experiments. A medal was also given to M. Rossignol for the part he played in conducting the experiments.

As an example of the results of preventive inoculation the following may be quoted :—

		MORTALITY.	
		During Vaccination.	*After Vaccination.*
Vaccinated sheep	3,663	58	0
Control sheep	2,867	60	141

In the duration-of-immunity experiment, 23 sheep were used—some had received vaccine and virulent culture, some vaccine only and the remainder no treatment (the number of sheep in each group is not given). All were inoculated with virulent cultures—the controls only died.

On certain Accidents resulting from Anthrax Vaccination. Reply to M. Weber.

(1882, p. 386. *Soc. cent. méd. vét.*)

Reports show that, in a number of instances, vaccination has failed to confer adequate protection against the naturally-occurring disease and that anthrax was produced in some inoculated animals. Pasteur advises re-vaccination when the death-rate among the inoculated proves that protection is insufficient. Deaths among the inoculated animals he ascribes to the first vaccine being too weak and the second too strong. He admits that the anthrax bacillus is not, as was previously supposed, something fixed and immutable, but, on the contrary, is variable, becoming modified with the action of time, temperature, etc. Pasteur is very frank and admits that the vaccine recently issued has not conferred the same high degree of immunity as that sent out a year previously. However, he cannot resist quoting the case of a farmer who lost six bovines in the course of a few days although no deaths from anthrax had occurred for two years. Vaccination was resorted to. He points out that if the vaccination had been carried out a month previously, the deaths would have been ascribed to the inoculation. It is encouraging (perhaps to some discouraging) to know that this much-used excuse (or explanation) employed by those unfortunates who have to prepare and issue anthrax vaccine is at least 55 years old.

The Attenuation of Viruses.*

(1882, p. 391. With Chamberland, Roux, and Thuillier. Communication made at *4th International Congress of Hygiene and Demography held at Geneva.*)

The attenuated anthrax bacillus has not the same vitality as that of the germ taken from the animal body; in a few months the attenuated strain can die off, whereas the virulent organism can survive for years (5 years is the longest period quoted).

The action of the oxygen of the air plays the chief rôle in the attenuation of the anthrax germ. If two series of broth cultures are set up, one in sealed and the other in open tubes, incubated at 42° – 43° C., and sub-cultured every 5 days under the same conditions, the cultures in open tubes will lose virulence, whereas those in sealed tubes will retain much of their original pathogenicity.

In addition to the anthrax germ, Pasteur quotes the attenuation of the fowl cholera bacillus, of a germ isolated from the saliva of rabid individuals, and of a germ obtained from the nasal discharge of horses infected with "typhoid fever of the horse."

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* "Viruses" means living germs and not necessarily ultra-microscopic or filterable viruses as known to-day.

He then refutes statements of Koch and his pupils. Among other things, Pasteur is accused of not knowing how to grow germs in a state of purity and of being unable to recognise the *Vibrion septique* (although he discovered it). In addition Koch says that the production of anthrax in "cooled" fowls is not remarkable: he asks whether they could not have been infected without first having been chilled, because a German worker has been able to infect 11 of 31 without this procedure. Pasteur rightly replies that Koch should have carried out such an experiment before indulging in criticism. Loeffler (of Koch's school) says that Pasteur introduces contaminating but non-pathogenic germs with his needle when inoculating media; these outgrow his germ and he finishes up with a culture of a non-pathogenic germ—the "attenuated culture." To overcome the difficulty of fowls, vaccinated with an attenuated fowl cholera culture, resisting the inoculation of virulent culture, Loeffler says that Pasteur has simply taken birds refractory to the disease. Finally, Loeffler does not believe that Pasteur used 80 birds in certain experiments because the expense would be too great. Pasteur's reply, obviously facetious, is that to establish the truth of attenuation, the State permitted him to carry on without worrying about expense. It is interesting to hazard a guess at what Loeffler's reaction would have been at that time (1882), could he have been confronted with the present weekly usage of small and large animals at Onderstepoort.

Pasteur answers the various criticisms satisfactorily and concludes by saying that they have only brought to light a host of errors and the lack of experience of their authors.

It seems a pity that brilliant men such as Pasteur, Koch, and Loeffler did not get together, instead of slating one another in public. On the other hand, the criticism may have spurred them on to greater effort.

Statistics of the Preventive Vaccination of 90,000 animals against Anthrax.

(1882, p. 414. *Acad. Sciences.*)

These results were collected by the Veterinary and Agricultural Society of Chartres and were read at one of its meetings by M. E. Boutet, a veterinarian. Pasteur asks the permission of the Academy to place the conclusions before its members.

(1) Sheep vaccinated during year 79,392
Annual loss before vaccination (average for 10 years) .... 9.01 per cent.
Loss since vaccination .... 0.65 ,, ,,
(During the year of vaccination the loss in the Eure-et-Loir district in the non-vaccinated was only 3 per cent. This is attributed to the great humidity experienced. Apparently big losses are to be expected during hot dry weather.)

(2) Controlled experiment (sheep)—

Loss in non-vaccinated	60 of 1,659	(3.9 per cent.)
Loss in vaccinated	8 of 2,308	(0.4 per cent.)

(3) Bovines vaccinated during year 4,562

Average annual loss before vaccination ..... 7.03 per cent.

Loss since vaccination ..... 0.24 ,, ,,

Anthrax Vaccination.

(1883, p. 418. Reply to a memoir of Dr. Koch.)

At the meeting held at Geneva Pasteur answered some of the criticisms levelled at him by Koch and his school; Koch refused to discuss the matter but said he would reply through the press. Three months later, Koch published a brochure on "Anthrax Vaccination; Reply to Pasteur's Lecture at Geneva". What follows is an abstract of Pasteur's reply.

Pasteur did not, as Koch implied, connect the germ found in the saliva of rabid people with the causation of rabies; he merely claimed to have isolated a new germ. Further, no claim was made that the germ isolated from the nasal discharge of horses suffering from "equine typhoid" was the causative agent; it was quoted as one of the four germs capable of being attenuated by oxygen.

Whilst giving Koch due credit for the observation of spore-formation in the anthrax bacillus, Pasteur points out that 7 to 8 years previously he had noted this phenomenon in the microbe causing silk-worm disease and in consequence he claims priority for the discovery of this process.

Pasteur was able to show that fermentation was the result of bacterial action, giving the death-blow to Liebig's ferment hypothesis. These and other studies occupied the years 1856 to 1876, a period at which Koch had not attained his extensive experience and pre-eminence in science. Pasteur's principal occupation was the isolation of germs and their cultivation in a state of purity, yet Koch accuses him of not knowing how to purify his cultures. This statement hurt Pasteur's feelings terribly.

Pasteur proves, almost mathematically, that Koch's belittlement of his investigations is without solid foundation—rather was his work the genesis of modern medical research. As early as 1850, Rayer and Davaine in France noted the presence of filiform rods in the blood of anthrax-infected bovines, but did nothing other than record the observation. In 1863, Davaine considered the significance of these rods in view of a lecture given by Pasteur in 1861 on butyric fermentation. In Germany, Franke wrote (1864) that following on Pasteur's studies on fermentation, the mucus of the bladder could not be regarded as the cause of decomposition of the urine and from Britain Lister wrote (1874) praising Pasteur for his work on the germ theory of putrefaction, which fully justified the adoption of antiseptic surgery. Pasteur concludes his argument by telling Koch that, willy nilly, he is a debtor to French science.

Pasteur tackled anthrax in 1877 when Paul Bert, the noted French physiologist, claimed that the disease was caused by a virus, because one could destroy the bacillus in blood with compressed oxygen and still reproduce the infection with blood so treated (see p. 107). Because Pasteur had been attacked by hemiplegia in 1868 and since then had lost the use of his left hand, he chose as collaborator M. Joubert, Professor of Physics at Rollin College. To prove that Davaine's filiform rod was the cause of anthrax, he applied the methods learned in the study of fermentation, that is, he cultivated the germ from the blood in the pure state in an artificial medium, and reproduced the disease with such a culture.

Koch had said that anthrax preventive inoculation with attenuated vaccine has not proved a success, but Pasteur reproduces his results to refute this. One experiment, carried out at the Turin Veterinary College, was apparently a failure, but the test material was the blood of a sheep dead for 24 hours, so that germs other than the anthrax bacillus could have been inoculated. A second immunity experiment, carried out at the same place gave good, but not excellent, results. Pasteur queries the amount of virulent blood used at the test; if this is too big, immunity can be broken down. It would appear that Koch was not *au fait* with Pasteur's experiments, was wrongly informed about them or omitted a number of most important details. For example, he says that vaccination was not a success because so many immunized animals died, and so many controls, but forbears to record that many more were vaccinated than were left untreated. To say that 10 animals of each group have died does not speak well of the vaccine; but if 1,000 have been inoculated and 100 left as controls the result takes on a different aspect.

Finally, Koch says that the natural disease is a greater danger to sheep than the inoculation of culture, and this despite Pasteur's experiments in which he was able to kill only 33 per cent. by feeding contaminated food and 100 per cent. by inoculating virulent culture.

Anthrax Vaccination.

(*Apropos* the failure of vaccination carried out at Turin, 1883, p. 442. *Acad. Sciences.*)

The commission of the Turin veterinary college (*apropos* anthrax vaccination. 1883, p. 452. *Acad. Sciences.*)

An experiment had been conducted at the Turin Veterinary College, and had failed, the vaccinated sheep dying after the test inoculation. Pasteur learned that the test virus had been the blood of an anthrax-infected sheep dead for more than 24 hours. In a letter he pointed out that such blood would contain pathogenic germs (the germs of septicaemia) in addition to *B. anthracis*. The college authorities asked how

he could make such a statement, not having seen the sheep. Pasteur offered to go to Turin and give a practical demonstration, but this was not accepted.

THE FIRST VOLUME OF THE *Annales de L'Institut Pasteur* AND IN PARTICULAR A MEMOIR BY ROUX AND CHAMBERLAND CALLED : " IMMUNITY AGAINST SEPTICAEMIA CONFERRED BY SOLUBLE SUBSTANCES ".

(1888, p. 462. *Acad. Sciences.*)

Pasteur's collaborators, Roux and Chamberland, had shown that the soluble products (what we now call "toxin") of *Vibrion septique* can immunize the guinea-pig against the harmful effect of the germ itself. Pasteur says that this opens up a big field in prophylaxis and expresses his joy that he was a witness of the new process carried out in his laboratory. It is a matter of history that this discovery has led to big things in the prevention of disease as proved by the success of preventive inoculation against braxy (toxin-antitoxin mixture and anaculture), black quarter (culture filtrate, formolized filtrate, and anaculture), bloedpens or lamb dysentry (toxin-antitoxin mixture and anaculture), and black disease of Australia (anaculture).

REPORT ON THE LONGEVITY OF ANTHRAX GERMS AND THEIR SURVIVAL IN CULTIVATED GROUND.

(1881, p. 693. *Acad. Méd.*)

Pasteur, Chamberland, and Roux have said that the ground above trenches containing anthrax carcasses can reproduce the disease on inoculation into susceptible animals and blame earthworms for carrying the infection to the surface. This is questioned and a commission of investigation, with Pasteur at its disposal, is set up.

Earth was taken from a 12-year-old and a 3-year-old anthrax trench, both from the surface and from a depth of 0.5 cm. – 0.8 cm.; a third sample (control) was got from a spot 20 metres from the older grave. Later, excrement from worms from the 12-year-old trench and worms themselves from the 3-year-old trench were examined.

The earths were washed several times with sterile water, heated at 90° C. for 20 minutes, and injected into guinea-pigs.

Results :

(5 guinea-pigs per series.)

12-year-old trench earth.—All dead in from 2 to 5 days, 4 from septicaemia and 1 from anthrax; bacilli observed in the blood and in the spleen of this one.

3-years-old trench earth.—Results as above.

Virgin earth.—All lived.

This experiment was repeated with the contaminated earths, using 3 guinea-pigs per series. All died, 5 from septicaemia and 1 from anthrax (12-year-old earth).

As a continuation, two guinea-pigs were inoculated with the blood of the animals dying of anthrax : both died and cultures of blood revealed anthrax bacilli.

Earth-worms :

From 3-year-old trench.—Washed excrement used — 3 guinea-pigs inoculated — 2 died from septicaemia and 1 from anthrax — culture positive.

From 12-year-old trench.—Excrement set up anthrax in guinea pigs.

Experiment of Pouilly-Le-Fort.

The possibility of rendering animals refractory to anthrax by the method of preventive inoculation.

(Report by H. Rossignol, veterinary surgeon, to the Agricultural Society of Melun on the experiments at Pouilly-Le-Fort, carried out under the direction of M. Pasteur, with the collaboration of Messrs. Chamberland and Roux.)

(1881, p. 697.)

This was what might be termed a test case which was to prove or disprove Pasteur's hypothesis. There, before a gathering of distinguished people, both scientific and lay, he had the courage to conduct an experiment which would prove either his making or his undoing. He emerged triumphant. The report is a long one, and goes into detail of the marking and arrangement of the animals, the method of inoculation, the reactions produced and so forth. The gist of the experiment and the results may be obtained at a glance by reference to the notes on the injections and to the tables.

Animals in the experiment : 48 sheep, 2 goats, 8 cows, 1 ox and 1 bull.

5.5.81 : 1st inoculation — 24 sheep, 1 goat, 5 cows, and 1 ox.
All stood the vaccination well.

17.5.81 : 2nd inoculation — As above. All stood the vaccination well.

28.5.81 : Test of virulence of virulent culture to be used for the test inoculation :
Twice vaccinated sheep — lived.
Control sheep — died 2 days.

29.5.81 : As 28.5.81.
Twice vaccinated sheep — lived.
Control sheep — died 2 days.

31.5.81 : *Final test :*
Twice vaccinated animals — 22 sheep, 1 goat, 5 cows and 1 ox.
Control animals — 22 sheep, 1 goat, 3 cows and 1 bull.
(See tables.)

TABLE I.

RESULT OF TEST INOCULATION OF VIRULENT CULTURE IN THE TWICE VACCINATED AND IN THE CONTROL SHEEP AND GOATS.

Days after inoculation.	*Vaccinated Sheep.*	*Control sheep.*
1	All well, some with slight oedema and temperature.	Sick animals numerous. Not feeding, oedema and temperature 104° F. 3 dead at 7 p.m.
2	All well.	2 p.m. In all 14 sheep and 1 goat dead. In evening all but 1 sheep dead.
3	One pregnant ewe died.*	Remaining sheep dead.
4	Indurated areas at point of inoculation.	
5	Indurated areas less in size.	
6	All well.	

* Did not die of anthrax.

TABLE II.

RESULT OF TEST INOCULATION OF VIRULENT CULTURE IN THE TWICE VACCINATED AND IN THE CONTROL BOVINES.

Days after inoculation.	*Vaccinated bovines.*	*Control bovines.*
1	All well.	1 with oedema and T. 104° F.
2	All well.	Oedema in all and one with T. 104.3° F.
3	All well.	Enormous oedema in all.
4	Indurated areas at point of inoculation.	" " " "
5	Indurated areas less.	" " " "
6	All well.	3 very ill. No appetite, quickened pulse and difficulty in standing.
7		2 with enormous oedema.
		1 with lessened "
		1 with very little oedema.
8		All improving.
9 – 14		All improving. Sold on 29.6.81.

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1937.

Rabies in South Africa.

By P. S. SNYMAN, B.V.Sc., Bloemfontein.

Descriptions of rabies and its control, as found in textbooks, are usually confined to the rabid dog of the more thickly populated areas of Europe and America.

When wild carnivora and blood-sucking animals play the main rôle in the dissemination of the disease, the control problems assume a totally different aspect. As an example of this the following may be quoted :—

(1) In Trinidad and parts of South America, the vampire bat, *Desmodus rufus,* is the chief agent. Here the outbreaks were marked by a disproportion between the small number of rabid dogs and the large number of infected cattle. In Trinidad alone, during 1929, 1930 and 1931, the death rate averaged 1,000 for each year, and 90 per cent. of the animals affected were bovines. Only two cases were seen in dogs.

(2) In the *Veterinary Record* (1935), under the heading of "Rabies Reminiscence of a Boundary Marker," is described an outbreak of rabies in Seistan, on the border between Afghanistan and Persia, first among jackals, which abounded there. From the jackals it spread to the dogs and eventually to wolves. During a single night, in a wild blizzard, two mad wolves which had entered the camel lines bit 78 camels, of which 46 went mad and had to be destroyed.

(3) In South Africa, on the other hand, we have quite a different problem to face, in that rabies is prevalent amongst the small wild carnivora, and the problem of its control therefore assumes a somewhat different aspect from that which the textbooks describe.

Origin of the Infection in South Africa.

One of the first questions asked in connection with rabies is : What is the origin of the infection ? The best reply is to recount what is known of the history of the disease in this country and leave the enquirer to form his own opinion.

Marais and Neitz (1932) give a short account of the known outbreaks. The first occurred in September, 1892, at Port Elizabeth. In January of the following year a further case occurred and was followed by numerous others. The disease spread to Uitenhage, Jansenville,

Willowmore, and Albany. It was probably eradicated the same year, as there were no further reports of outbreaks. Du Toit (1930) quotes a significant statement by the then Colonial Veterinary Surgeon, Dr. Hutcheon : "I was in great dread at one time when the disease was reported at centres a considerable distance from Port Elizabeth, lest it should be communicated to our wild animals, such as jackals, etc. But with the exception of cattle, which developed the symptoms of the disease near Stadens, we have not heard of any other animals, except dogs and cats, that had become affected with the disease."

Rabies in Southern Rhodesia.

In August, 1902, the disease was first diagnosed in a dog, and spread rapidly. Preventive measures included the destruction of 40,000 dogs in that year and 60,000 in the following year. Thereafter the incidence rate fluctuated from year to year. In 1911 another severe outbreak occurred, but in 1913 the position was greatly improved and since 1914 no further cases have been reported.

The Chief Veterinary Surgeon also feared that the disease might spread to the wild animals and so lessen the possibility of its total eradication. A few cases were reported in wild carnivora, but the disease was nevertheless brought under control.

Suspicious Outbreaks.

Cluver (1927) described all the suspicious cases that had occurred in human beings and found that in the majority of cases the persons had been bitten by a yellow mongoose or genet cat. None of these cases were confirmed by histological examination or biological test. It is interesting to recall that certain natives of the Barolong tribes believe that the bite of the yellow mongoose is venomous—like that of a snake, except that the venom takes longer to act.

The first diagnosis of rabies confirmed by laboratory examination was in a human being in 1929, and in the same year the diagnosis of rabies was confirmed in a yellow mongoose. Since then many cases have been detected and reported upon.

Areas Infected with Rabies in South Africa.

The known infected areas are as follows :—

(1) *Central Area.*—This comprises the area bounded on the north by and including the districts of Vryburg, Mafeking, Lichtenburg and Ventersdorp; on the west by Hoopstad, Boshof, Fauresmith and Philippolis; on the south by Trompsburg, Edenburg and Reddersburg; on the east by Bloemfontein, Brandfort, Winburg, Senekal, Lindley, Heilbron, Frankfort, and that portion of the Transvaal included by the above and a line from Frankfort to Mafeking. The adjoining districts are to be considered as being under suspicion.

(2) *North Eastern Area.*—This comprises the districts of Middelburg, Carolina, and Bethal. This area is joined to the central area by the suspected district of Standerton.

(3) *Southern Area.*—Here the infection is scattered in the districts of Cradock, Middelburg (C.P.), De Aar, Carnarvon, Hanover, and Britstown.

(4) *Cape Peninsular Area.*—This is the most recent infected area.

The Carriers of Rabies in South Africa.

The following list gives the number and species of animals in which rabies has been diagnosed since 1929 :—

Man	23
Dog	14
Cat (Domestic)	12
Ox	33
Sheep	3
Pig	3
Yellow Mongoose (*Cynictus penicillata*)	42
Stockstert (*Suricata suricatta*)	2
Genet Cat (*Genetta felina*)	6
Squirrel (*Geosciurus capensis*)	2
Myonax pulverulentus	1
Wild Cat (*Felina cafra*)	4

Total : 145 cases.

Taking into account that in many outbreaks of rabies in cattle there is a history of a mad mongoose, and the large number found to be infected, it is evident that this species of animal is by far the most important carrier. That the disease is more prevalent in the yellow mongoose than in the other species of wild carnivora is probably on account of the mongoose being very abundant, living in small colonies and having diurnal habits, in contrast with other species of wild carnivora like the genet cat, etc., with nocturnal habits and solitary mode of living.

In addition to the species of animals named above, several species of wild carnivora, like the jackal, etc., are suspected, and in all probability investigations will prove that more species of wild animals are associated with the spread of the disease.

The distribution of the species of animals in which rabies has been diagnosed is as follows :—

1. *Genetta felina* : throughout South Africa.
2. *Cynictus penicillata* : all over South Africa, except the coastal belts, and very abundant in sand veld.
3. *Suricata suricatta* : more or less the same as *Cynictus*, but more restricted in the Cape, where it is essentially a Karroo species.
4. *Myonax pulverulentus* : practically restricted to south of the Orange River.

5. *Geosciurus capensis* : the same as *Cynictus* but more prevalent in Western O.F.S. and Transvaal.
6. *Felis caffra* : in all mountainous parts of South Africa.

The Spread of the Disease.

Before dealing with the methods of control of the disease, there is one point which should receive some consideration, *viz.*, whether the disease is spreading in the country or whether we are only establishing the existence of the disease in the various parts. To my mind both alternatives are correct. In support of the latter alternative I wish to point out that the isolated outbreaks in various districts, with no connecting links, tend to show that the disease is prevalent there, but is left undiscovered. As proof of this, I have only to mention the recent outbreaks near Senekal and Frankfort. In both instances the nearest known infection is a matter of 60 to 70 miles away; yet in both cases there is a history of rabies having been present within a radius of six miles, about two years ago. Therefore it is more likely that the new outbreaks are connected with the previous ones, than that the disease has spread to these centres. That the disease is also spreading is evident from the fact that many outbreaks occur that have no connection with any previous ones.

However, one will not be in a position to supply the true answer until more is known of the migratory habits and the life-history of the different species of wild carnivora.

Neitz and Thomas (1934) speculate on the effects which the big drought of 1933 and the general soaking rains which terminated it would have on the incidence of rabies. A few figures may perhaps supply this information. Those given below represent the number of outbreaks for the period 1929 to 1936 in the O.F.S. and the Cape. The figures in brackets indicate suspected outbreaks.

	O.F.S.	Cape.
1929	12	2
1930	2 (2)	2
1931	4	0
1932	8	5
1933	22	3 (2)
1934	4	1
1935	9	0
1936	22 (12)	3

The answer would therefore be in the affirmative. The severest part of the drought was from September to November in 1933; the rain started in November and stopped in April, 1934. Immediately prior to this rainy period there were 18 outbreaks, while there were 8 during the period, and only one during the next six months of the year.

Control Measures.

As it is possible to adopt proper control measures only where we have sufficient knowledge of a disease, our control measures in respect of rabies are therefore still very inadequate.

Propaganda.—The first and most important step to be taken in connection with a disease of which the existence has only been known for a short time, and of which the public is very ignorant, is the education of the community. This can be done by public lectures, by addresses to farmers' associations at farmers' days, by giving prominence to outbreaks in the press, etc.

It is very essential that school children should be warned, since a percentage of the human cases occur in children, as a result of catching what appears to them to be "tame" meercats, but which animals are actually rabid.

As the word quarantine reacts adversely on all farmers, it is essential to stress that the quarantine conditions, although they appear irksome, are not so in practice.

Diagnosis.—Of the utmost importance in the control of any disease is a speedy and accurate diagnosis. It stands to reason that if one is able to make a diagnosis on the spot, one is in a far better position to decide on the control measures to adopt. Unfortunately, in the case of rabies we are handicapped in this respect. The quickest diagnosis is by a histological examination of certain portions of the brain, an examination which can only be done in a well-equipped laboratory. It takes up to ten days before the result of the examination can be expected, in cases where Negri bodies are found; or it may be as long as three months where no Negri bodies are found, or where the material was unsuitable for histological examination. In these cases one has to await the result of the biological test.

In many instances no material for examination is available owing to decomposition of the carcase, or owing to laceration of the brain in cases where the suspected animal has been destroyed by a shot through the head. In such cases one has to rely solely on the history, which is sometimes based on imagination. I have to point out here, that one of the first precautions to be taken is to treat with anti-rabic vaccine any human being who has been bitten or has come into contact with infection by dosing infected cattle for some other disease. The District Surgeon, who performs this, only undertakes it on our recommendation, and as this treatment is very urgent it is immediately realised what responsibility the veterinarian shoulders. Previously, when outbreaks were very rare, it was quite easy to recommend it in all suspected cases; but as the treatment is expensive and the cases that now occur are so numerous, one cannot recommend is so freely as in the past, and discrimination becomes essential.

Every possible means should be employed to instruct the public in the procedure to be adopted in suspicious cases. As a safeguard to them and to assist us in our difficult task, it should be advertised on all railway premises and other public places, as in the case of plague, that on no account must a dog that has bitten any person be destroyed, but that the animal should be secured and the matter reported to the authorities. If this is done, a better opinion may be formed by keeping the suspected animal under observation for a few days. If in ten days time the suspected animal is still normal, no action need be taken.

In connection with the foregoing remarks the following figures may be of interest. During the past 12 months in the O.F.S., out of the total number of 34 outbreaks dealt with, no material for laboratory examination was available in 10 instances, and out of 20 cases where a clinical diagnosis of rabies was made, in two cases only was the diagnosis not confirmed.

PRECAUTIONS TO BE TAKEN ON THE OUTBREAK OF RABIES.

Whenever an outbreak of rabies occurs, consideration is given to the following points :—

(1) Destruction and disposal of infected animals.
(2) The area to be placed in quarantine.
(3) The quarantine measures, *i.e.* as regards isolation, preventing contact between different animals, etc.
(4) Anti-rabic treatment.
(5) Measures adopted to control the source of the infection.

1. *Destruction and Disposal of Infected Animals.*—I need not elaborate on the manner of destruction of the infective carcases, as it is performed by taking the usual precautionary measures in disposing of infective material.

2 and 3. *The Area to be Quarantined and the Quarantine Measures.*—The area to be quarantined depends mainly on two factors—the kind of animal involved and the locality.

It is self-evident that when an animal like a dog, which can roam long distances, has been the cause of the outbreak, a larger area must be considered than in the case of a bovine, which is usually confined to a comparatively small space in an enclosed camp or farm.

It is usual in the case of dogs to spread the area out as far as possible, even up to a radius of six miles, if there is the least justification for doing so. In the case of cats and other domestic animals excepting dogs, the area placed under restriction is confined to the farm only, if there are no neighbouring homesteads.

On commonages and in towns one has to be a little more careful, as control measures are somewhat more difficult to carry out, and it is therefore not uncommon to have two areas defined, *viz.* the immediate

vicinity of the outbreak, where drastic measures are taken; and farther afield, where less drastic measures are enforced. In the former case, all dogs are to be securely chained and are not allowed off the premises. In the second case, the dogs must be confined to their premises, and if taken for exercise must be leashed and muzzled. In the case of cats, another aspect has to be considered, *viz.* the rôle they play in the prevention of bubonic plague, and some latitude must be allowed. At night, however, some control has to be exercised, and quarantine conditions therefore imposed to shut the cats up at night in the house or barn. Incidentally their place of confinement offers them an opportunity to be useful on the right spot.

4. *Anti-rabic Treatment.*—Apart from treating human beings that have actually been in contact with infective material, wholesale treatment is very seldom adopted.

Anti-rabic treatment in man need not be considered here except to remark that in every outbreak which has so far occurred in bovines, the owner or his servants have dosed the animal for gallsickness. The method of dosing by pulling out the tongue or inserting the hand in the mouth is, in view of small abrasions likely to be present on the skin, tantamount to a bite. The practice of natives in this country of skinning and eating all carcases often necessitates the treatment of large numbers of natives.

Anti-rabic treatment of animals is very seldom resorted to on account of practical difficulties. Dog-owners in the U.S.A. and elsewhere often demand preventive inoculation, but owing to the following practical difficulties this is never resorted to here: (i) insufficient control over the canine population; (ii) owners neglecting to have dogs treated; (iii) stray animals. Resort to such preventive measures would therefore encourage a feeling of false security on the part of the public.

5. *The Control Measures adopted at the Source of the Infection.*—There is no doubt that rabies in this country is a disease of the small wild carnivora, and for the effective control of the disease these animals must be eradicated.

At the present moment there is no legislation enforcing the eradication of these animals, so that very little is being done in this regard.*

In the Hoopstad and Boshof districts, which are very heavily infected, attempts have been made through farmers' associations to organise clubs on the same lines as jackal clubs for the systematic eradication of these animals. In this way we have achieved very little. The public as a whole is very enthusiastic about the idea, but the argument always raised is that

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* After this article had been written a Government Notice appeared, authorising that a Veterinary Officer may order the destruction of wild carnivora on an infected farm. (G.N. 1910 of 18th Dec., 1936.)

it will be futile for about 90 per cent. of farmers to undertake it at great expense and trouble, if the other 10 per cent. continue to breed the animals on their farms.

Even on infected farms, it is very difficult to persuade the owner to undertake the eradication of the yellow mongoose. On farms where this is not undertaken, the nett result is that in consequence of the restricted movements of the dogs and to a lesser extent of the cats also, the rabies and plague carriers are allowed to increase in numbers. In practice this amounts to encouraging the propagation of the disease.

It will be noticed that mention has here been made only of the yellow mongoose. This is because it is the most important carrier, as has been pointed out before. This does not mean that the other wild carnivora are of no importance; but it is abundantly clear that, as they are mostly of solitary habits, there is much less chance for them to spread the disease.

In conclusion I only wish to remark that in view of the large number of outbreaks recently reported, in the absence of the immediate adoption of effective measures for its eradication, rabies will in the near future be a disease of equal importance to plague.

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Trichomonas Infection in a Heifer and a Brief Review of Abortion in Bovines due to Trichomonas Infection.

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The purpose of this article is to record the occurrence of trichomonas infection as a cause of abortion in cattle in South Africa. Although this infection has frequently been encountered in European countries and in the United States of America, there is no previous record of its having been met with in this country. From time to time outbreaks of abortion, apparently of an infectious type, have been reported, in which brucella infection could be excluded and vibrionic infection could not be traced. It is possible that trichomonas infection has been responsible for some of these outbreaks.

The case to be recorded occurred in a heifer which was in an experiment devised to ascertain whether the condition known as porphyrinuria (pink tooth) could be transmitted by a bull some of whose progeny had previously developed the condition. The bull himself had not shown symptoms of the disease.

The heifer aborted on March 23rd, 1936. The fœtus was about eight inches long and came away with the fœtal membranes. The fœtal cotyledons were greyish brown in colour and there was a small amount of a brownish exudate on them and between the villi. The internal organs of the fœtus showed no pathological changes.

In cases of abortion occurring in cows at Onderstepoort, or where fœtuses or afterbirths are sent in for examination, it is a routine measure to examine material by means of moist unstained preparations before proceeding to examine stained material. In this case a little of the material from a cotyledon was examined on a slide with a coverslip. Numerous trichomonads could be seen swimming rapidly about in the fluid and a few of them were found in the stomach contents of the fœtus as well. They could be easily seen with a magnification of 400 diameters, and with the oil-immersion lens the lashing movements of the flagella and the undulating membrane could be easily observed. Preparations stained with Giemsa without special fixation were rather disappointing. The parasites appeared contracted and their morphology could not be made out. With Giemsa stain after fixation with May-Grünwald, some very well-stained parasites were seen in which the morphology was clear. In the photograph of one of these trichomonads (fig. 1), one can see quite distinctly the

three anterior flagella, the undulating membrane, and the nucleus.

As there was a rather heavy bacterial contamination of the afterbirth and stomach contents of the fœtus, the cultures which were attempted rapidly became overgrown and trichomonads could not be found after twenty-four hours incubation. When material which had been kept in a refrigerator at about 4° C. was brought out and warmed in an incubator, the parasites could be found still actively moving after ten days, but by the fourteenth day they were all dead. No transmission experiments were attempted.

The day after the heifer had aborted, the mucus in the vagina was examined for trichomonads, but none could be found and the *os uteri* was completely closed. The herd bull also was examined, but no parasites could be found in the secretion of the sheath.

In view of the large amount of work which has been done on trichomonas abortion in recent years it may be of interest to give a brief review of the subject. Most of the information in the present article has been obtained from the excellent review of Stableforth and Scorgie (1936).

Trichomonas Infection in Cattle.

For the last thirty years it has been recognised in Europe that a condition occurs in cattle characterised by vaginitis, endometritis, pyometra, and early abortion. To the flagellated protozoon associated with it the name *Trichomonas fœtus* was given. Various investigators have reported that they have found the organism in the fœtal membranes in cases of abortion in cattle, and in fœtal stomachs as well. In cases of vaginitis and in uterine discharges it has frequently been reported. In the last few years the organism has been successfully cultivated on artificial media and the disease has been transmitted experimentally.

Trichomonas fœtus is almost entirely confined to cattle, but a report has appeared in which mention is made of the finding of a trichomonas in cases of genital disease in equines and swine.

Trichomonas fœtus infection has been reported from most European countries, the United States of America, and from Japan. Recently it has been reported from Great Britain.

Opinions vary as to the economic importance of the disease, but there is no question that the losses in some outbreaks have been very heavy. Kust (1934) found that it was responsible for 38% of the cases of sterility in cattle investigated at the clinic in Giessen, Germany. Considerable losses have occurred in districts in Germany where communal bulls have been used. A high percentage of pyometras appear to be due to this infection. In individual herds the losses may be very high, but frequently only odd cases of infection occur.

Morphology.

Trichomonas fœtus (see figs. 1 and 2) is a flagellate protozoon with an average length of about 12μ and a width of about 7μ. The shape varies somewhat in different environments, such as vaginal or uterine discharge, or culture media. The organism is usually pear-shaped and slightly pointed at the posterior end. It may be egg-shaped or spherical. At the anterior end there are three flagella which are about as long as the body. A fourth one starts at the anterior end and runs along near the body to a little distance beyond the posterior end. It is joined to the body by an undulating membrane which can be seen to be in constant movement in the living organism. The trichomonas moves about very rapidly in fresh material, although in very mucoid discharges the movements are greatly retarded. The main morphological features can easily be seen

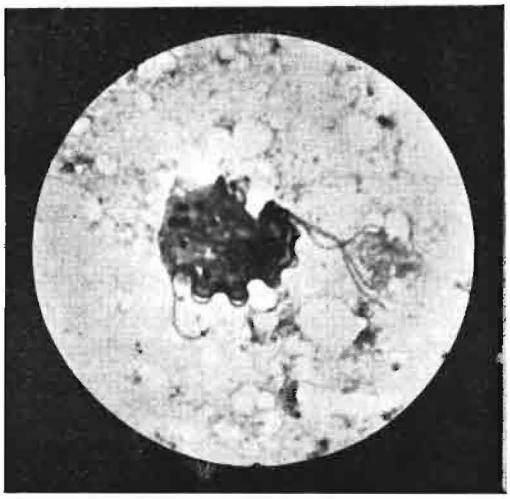

Fig. 1.
Trichomonas fœtus. (1600 ×.) Smear from fœtal membranes stained with Giemsa.

with a magnification of 400 diameters, but valuable aid in diagnosis is obtained from the use of vital dyes, such as neutral red, which slow up the movements and stain the granules in the cytoplasm.

It is not easy to prepare well-stained specimens of the parasite and one should use moist fixation in sublimate alcohol followed by Giemsa stain for 12 hours. Moist fixation with May-Grűnwald followed by Giemsa stain will give a small proportion of well-stained parasites.

In a well-stained preparation one should be able to see, in addition to the structures previously described, an oval nucleus, an axostyle and

several blepharoplasts from which the flagella arise. The nucleus is about a third of the length of the body and may contain deeply staining bodies. The axostyle extends through the body in its long axis and has a spicule which projects at the posterior end. There is a cytostome or mouth and there may be deeply staining granules in the cytoplasm.

In examining for trichomonads fresh preparations are much the best and are to be preferred to stained ones. Usually one can find the parasites easily, but sometimes a prolonged search is necessary. Cold interferes with the mobility of the parasites. Bacterial contamination rapidly overgrows and suppresses the organism. One should not be satisfied with one examination of vaginal mucus or uterine discharge, as the parasites may not be present constantly; the same applies to examination of the bull.

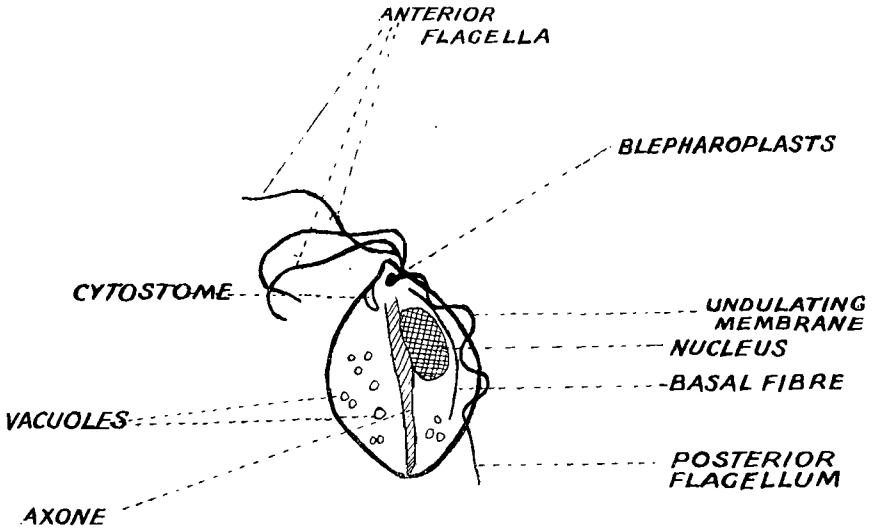

Fig. 2.

The early part of the period of œstrum is stated to be a good time to make an examination.

History and Symptoms.

Although the presence of the disease may not be suspected until several cases of abortion have occurred or pyometra is observed, it is usually noticed that animals thought to be in calf are returning to the bull and one usually finds one or more small fœtuses in the cow byre, or some cows may be noticed to have a vaginal discharge. A particular bull is usually found to be associated with the presence of the disease. Cases of pyometra do not occur early in the outbreak of the disease. Kust (1934) describes swelling and inflammation of the vulva soon after service, followed by a vaginitis with a mucous discharge containing small white flakes and in which trichomonads are very frequent. This vaginitis dis-

appears after a few days. Subsequently a chronic endometritis may develop or conception may take place followed by early abortion. The chronic endometritis cases usually come into œstrus every three weeks and conception does not take place. The fœtus may be retained and pyometra develop. Abortion usually occurs before the fourth month and the fœtus and its membranes come away together. Usually little change is noted in the fœtus and fœtal membranes, but there is sometimes a stale sweetish smell. As a rule trichomonads are frequent in the fœtal membranes and may be found in the fœtal stomach. After abortion recovery may take place rapidly and no further abortions occur, but a second abortion is not uncommon. Sometimes the fœtus dies and the cow still appears pregnant but does not calve at the expected time. The uterus will be found to be large, as if pregnant; but no fœtus will be felt. The uterus contains a large quantity of pus, usually of a whitish colour and glutinous, and one may find portions of a macerated fœtus in it. The vaginal discharge is variable in character : it may be mucoid, may contain whitish clots, or in some cases may resemble pea-soup.

Mode of Infection and Spread.

The disease appears to be a purely venereal one, though Kust (1936) has observed the parasites in the blood-stream. The disease does not appear to spread from one cow to another simply by contact. All observers agree that the bull is the transmitter of the disease. Bulls usually show no symptoms and develop a chronic infection, but a discharge may be seen soon after infection. Kust (1934) describes swelling of the prepuce with some discharge and pain on urination or at service. Small nodules may be seen on the penis, which is reddened. Trichomonads can usually be found. However, great difficulty has been found by most workers in determining whether or not a bull is infected.

Control and Treatment.

In the control of the disease a careful examination must be made of all cows which are not breeding normally. Treatment of all affected cows and bulls should be undertaken. Great care should be taken to prevent the introduction of the disease by an infected bull.

There is no specific treatment. Curative measures must therefore be on the lines of those usually undertaken in cases of vaginitis, endometritis, and pyometra. It is difficult to determine whether a bull has really recovered from the infection and a bull once infected is always suspect.

In conclusion a brief reference will be made to the artificial cultivation of the *Trichomonas fœtus*, the serological diagnosis of the disease, and artificial transmission.

Artificial Cultivation.

The cultivation of the organism has been successful in the hands of numerous workers. Broth with the addition of 5 to 10% of blood, or egg media with a drop of blood in the condensation water will allow good growths to develop. Up to 60 passages have been made and the organisms kept alive for seven months. The optimum temperature for growth is about 38° C. and the optimum pH is 7. Artificial cultivation may be useful where only a few parasites are present in the suspected material and an enrichment method is required. Bacterial contamination is difficult to avoid and, if at all heavy, usually kills out the trichomonads in cultures. A method has been devised by Glaser and Coria (1935) where by the use of a V tube one can inoculate one arm and the trichomonads will wander into the other one and can usually be found there in a few hours, without bacterial contamination.

Serology.

The complement fixation test has been applied to the diagnosis of the disease, using extracts of trichomonads from cultures as antigen. Positive results have been obtained in most cases, but the reactions are often very weak and the serological diagnosis has no real advantage over the microscopical examination of discharges.

Artificial Transmission.

Only a few transmission experiments in this disease are recorded in the literature, done with a limited number of animals. Characteristic symptoms of the disease have been set up in cows with infective material injected into the vagina and actual abortions have been produced. Typical cases have also been produced in bulls by injection of infected material into the sheath, and such bulls have transmitted the infection to cows.

Successful infection has been produced by the injection of trichomonads in artificial culture into both bulls and cows.

Small animals, such as rabbits, guinea-pigs and mice, have been infected with the organism both by intravaginal and intraperitoneal inoculation. By the latter method a peritonitis frequently results in guinea-pigs, the fluid containing numerous trichomonads.

Discussion.

The most important question arising out of the work carried out on *Trichomonas fœtus* infection is whether the organism is really the cause of the disease or not. Kust (1936) has reported the finding of an organism indistinguishable from *T. fœtus* in the blood of infected cattle and also in some cases in cows which had not been served, but most workers have not been able to find the parasite in normal cattle. However, there

is no doubt of the existence of a disease characterised by disturbances of the genital tract, associated with early abortion and pyometra, in which the trichomonas is constantly present, and no other aetiological factor can be found. The typical disease can be set up by artificial infection with natural infective material or cultures of the organism. A trichomonad, *T. ruminantium,* has been described from the rumen of cattle and may be related to, or identical with *T. fœtus,* but there is no proof of this. If the two organisms were identical, then it would be possible for the disease to arise spontaneously.

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Jl. S.A.V.M.A.
VIII (3) : 141 – 146.
1937.

Dik-oor or Geel-dikkop on Grass-Veld Pastures.

By C. RIMINGTON, M.A., Ph.D., B.Sc., A.I.C., and J. I. QUIN, D.V.Sc., Onderstepoort.

During the present season, 1936–7, many reports have reached us of outbreaks of dik-oor among sheep grazing on grass-veld pastures. In many instances, farmers stated that the animals had been turned on to oat-lands which had been recently cut and where there was a good stand of fresh, green "sweet-grass." After some days of grazing upon this grass, sheep began to fall ill with the symptoms typical of dik-oor or geel-dikkop, namely, swollen ears and face, constipation, and yellow discolouration of the conjunctivae. The ears and lips soon became hard and necrotic and the carcasses of animals which died were found, on *post mortem*, to be deeply bile-stained. Fig. 3 illustrates the condition.

Fig. 3.
Sheep suffering from "Dik-oor" after grazing on recently cut sweet-grass.

The occurrence of this disease, which presents a marked similarity to that usually termed geel-dikkop and occurring in the Karroo when sheep graze upon poisonous *Tribulus* plants, is not unknown in the Highveld and northern Free State, although outbreaks have been more severe this year than formerly.

We were able to investigate one such outbreak in detail owing to the kindness of Mr. T. du Toit of "Schaapplaats," Wolwehoek, O.F.S.,

in placing facilities at our disposal. The history of the disease on this farm is as follows : A small land of oats had been cut some 14 days before a flock of 200 Merino sheep was turned in to crop the fresh " sweet-grass " which had sprung up. In the meantime, good rains had

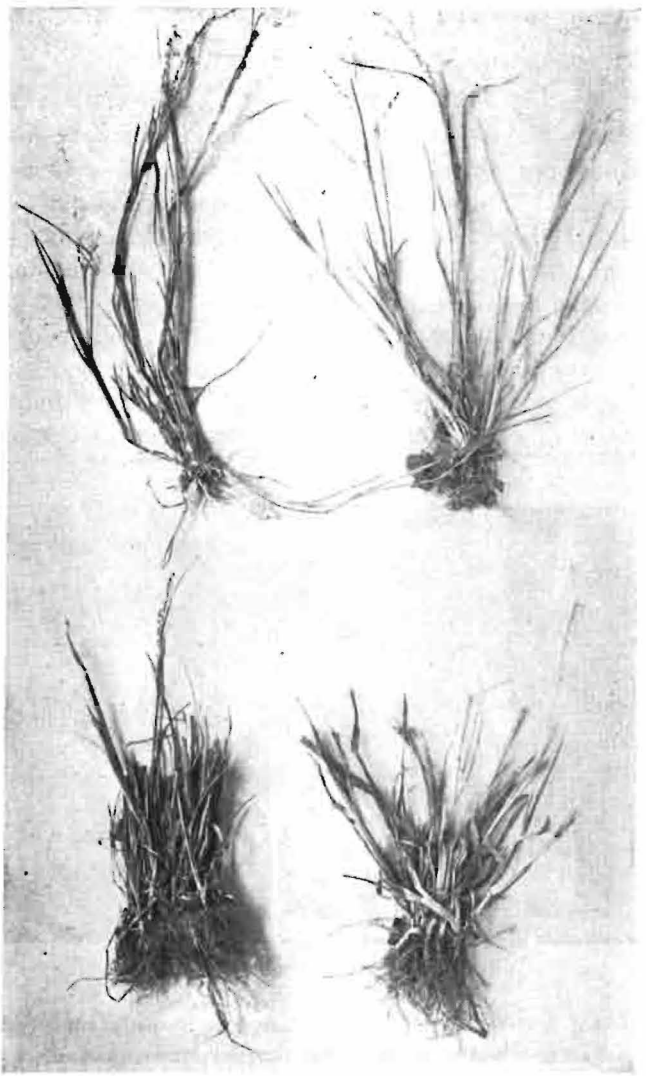

Fig. 4.
Tufts of *Pannicum laevifolium* after cutting and grazing, showing growth of new shoots. (About natural size.)

fallen. Within 7 days, cases began to occur and continued until our arrival, one more fresh case being found when we inspected the flock. A careful examination of the camp showed that, apart from the oat stubble

and a small sedge which was apparently not grazed, the vegetation was limited, in the main, to two species of grass. Specimens were taken and have been kindly identified for us by Mr. G. C. S. Roets, B.Sc., as *Pannicum laevifolium* (Blou-saad) and *Pannicum coloratum* (Os-gras).

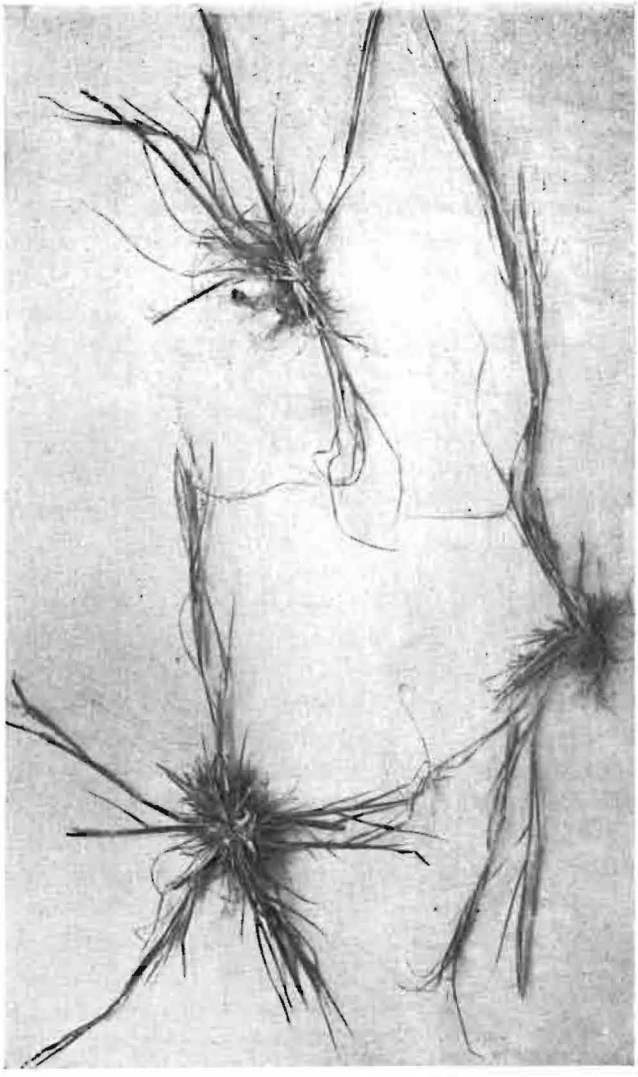

Fig. 5.

Pannicum coloratum after cutting and grazing. (About natural size.)

Over the greater part of the camp these grasses grew in tufts approximately 9 inches in height and showed signs of having been cropped, but toward the southern boundary, where the oat stand had been too poor to

cut, they stood to a height of some 2 to 3 feet. The accompanying photographs (figs. 4 to 6) show the camp and pressed specimens of the two grass species. A *post-mortem* examination of an affected animal confirmed

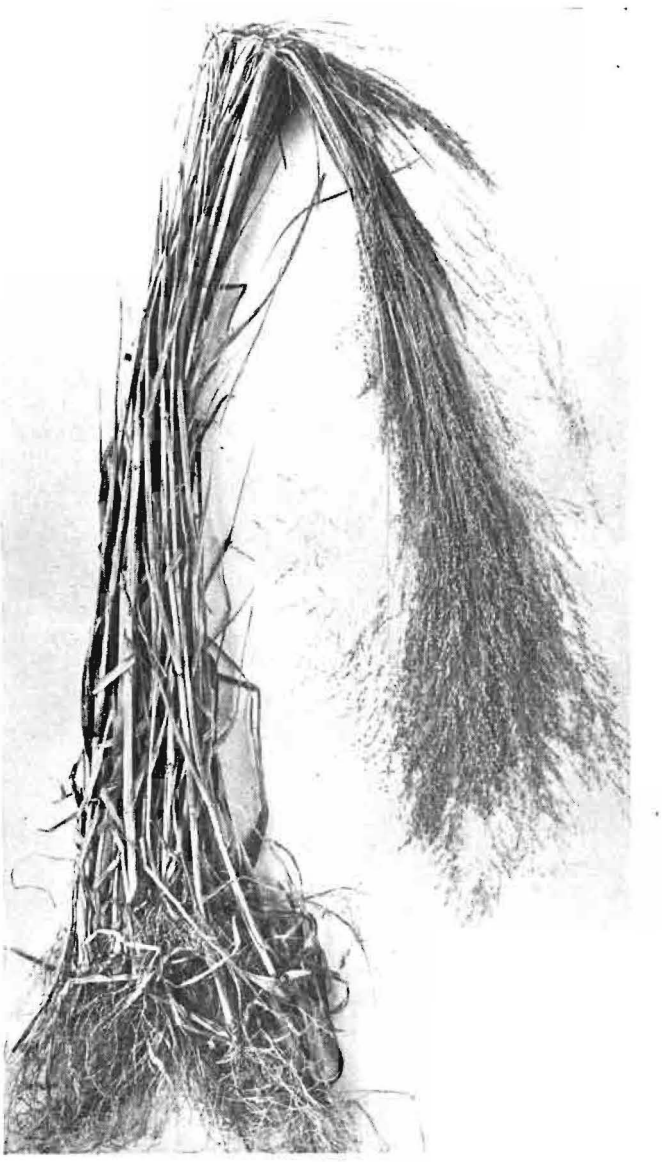

Fig. 6.
Uncut *Pannicum laevifolium*. (About natural size.)

the diagnosis of dik-oor or geel-dikkop. We now know definitely of three distinct plants, belonging to different families, which can cause this

condition, namely, *Tribulus spp.* (Zygophyllaceæ), *Lippia rehmanni* (Verbenaceæ), and these two *Pannicum* species (Gramineæ).

For some time past we have been conducting a chemical investigation of the plant *Lippia rehmanni* and have isolated from it a pure substance capable of causing geel-dikkop when dosed to sheep (Rimington and Quin, 1935). The mechanism of the photosensitisation has also been elucidated and recorded by Rimington and Quin (1934), and Quin, Rimington, and Roets (1935). Roets, in an article shortly to be published in the *Onderstepoort Journal,* will give an account of further work done upon *Lippia rehmanni* with particular reference to variations in toxicity during the year and the effect of pruning or cutting the plants back. From this article it will be seen that the new growth of leaves following pruning contains a relatively very large amount of the toxic principle " Icterogenin."

The substance isolated from *Lippia* is a resinic acid, probably closely related to the terpenes. Our knowledge is limited concerning the functions of these materials in the plant economy, but as a class they are of fairly wide distribution. It is quite conceivable that the same or very similar substances may occur in all the three plants mentioned and even in others which, so far, have not been incriminated as causing geel-dikkop. Toxicity is a matter of degree. The quantity of active principle present in *Lippia* is comparatively high—roughly 0.1 gm. per 100 gm. dry weight in the leaves—and this plant, were it eaten by stock, would most certainly prove to be a very serious cause of geel-dikkop. *Tribulus* is only at times sufficiently toxic to cause illness and the same may be said to apply to the *Pannicum* species. Only under a certain set of conditions does the amount of active principle present rise to such a height that it becomes dangerous to the animal. Cutting or trampling may be one of these conditions and we are constantly reminded in this connection of the sudden development of fatal amounts of prussic acid in certain normally excellent fodder grasses under the influence of factors as yet not perfectly understood. For a fuller discussion of the question we would again refer to the article by Mr. G. C. S. Roets.

Consignments of grass from camps where outbreaks have occurred are being investigated chemically, but the small quantities of toxin present render the work slow and laborious. We hope ultimately to gain sufficient knowledge to enable us to assign the active principle to its proper class and to show its relationship to Icterogenin from *Lippia* and the principle present in *Tribulus*. Questions of both plant and animall physiology must be taken into consideration, as well as the more purely chemical aspects of the problem.

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Mare's Death by Electricity.

By A. M. HOWIE, M.R.C.V.S., Cape Town.

On the 2nd April, 1937, I visited the Police Post at Kraaifontein in response to a message I had received from the District Commandant, Cape Town. The following history of what transpired at Kraaifontein during the day in question is, I think, worth recording :—

A police horse which was being ridden out of the yard suddenly dropped to the ground when at a particular spot between the stable and garden of the Post Commandant's house. The animal was unable to move, and was eventually pulled clear of the place where it had fallen, with apparently no ill effects.

In order to return to the stable, the animal had again to traverse the area between the garden and stable and a repetition of what had previously occurred was experienced. I was informed by the Post Commandant that the persons who handled the horse when getting it away from the area did not feel anything unusual. The horse was apparently none the worse of the shock when I saw him a few hours later.

There was a similar occurrence soon after the horse episode, when two donkeys walked over the area, and like the horse they soon recovered and suffered no ill effects.

Shortly afterwards a poor-conditioned, aged mare walked on to the area, fell down, whinnied once or twice, and died.

The Post Commander then suspected a leakage in the electrical current between his house and the stable and when the matter was investigated by the Cape Town Municipal electrical staff, his suspicions proved to be justified. The pipe containing the power wire for the stable lights was about eighteen inches beneath the surface of the ground and its course was from the garden of the house to the stable. It was here that a leakage was discovered, and the ground in the vicinity was charged with electricity, the strength being accentuated by an overnight rain—following a prolonged dry spell—which had rendered the soil damp and thus increased its conductivity.

It is well known that horses are very sensitive to electricity, and this case proves that a voltage which was sufficient to kill the mare and render other equines momentarily helpless was not even felt by human beings when handling the electrically charged animals.

Tuberculosis—An Occasional Cause of Abortion in Cattle.

By A. S. CANHAM, F.R.C.V.S., Onderstepoort.

Abortion among cattle in the Union has assumed rather alarming proportions during the last ten years. Contagious abortion caused by Bang's organism is the common cause. Many samples of blood are tested annually for this disease at the Laboratory and it often occurs that in spite of a history indicating contagious abortion negative results are given by the agglutination test.

During the last few months cases of abortion in cattle caused by Vibrios and by Trichomonads have been diagnosed at Onderstepoort. This article is written with the object of pointing out that tuberculosis may be an occasional cause of abortion in cattle and also to record a case of tuberculosis in a fœtus of about 6 months.

History of the Herd.

The cattle comprising the herd in question are pedigree animals. They are housed in modern stables and carefully looked after. Additions to the herd are made only through well-known breeders overseas and the animals are tested before being brought into this country. Abortions commenced about two years ago and took place at irregular intervals. Up to the present, 11 calves varying in age from about 3 to 6 months, have been born prematurely. The abortions have not been accompanied by retention of the placenta. Four of the cows that aborted have since had normal calves and are now in calf again. Vibrio or Trichomonas infection has not been diagnosed in this herd and in every case the test for contagious abortion has been negative.

Case 1.

On 10.10.36 a fœtus of about 3 months of age intact in its membranes was brought to the Laboratory. A sample of allantoic fluid was withdrawn and the liver was removed and placed in a tube of sterile saline. Smears made from the fœtal stomach were negative. Two cc. of the placental liquid were injected intraperitoneally into a guinea-pig and 2 cc. of a saline suspension of the fœtal liver were introduced by the same route into a second guinea-pig.

The first guinea-pig died on 17.12.36 and *post-mortem* examination revealed lesions of generalised tuberculosis. The second guinea-pig was killed and the *post-mortem* findings were similar. Acid-fast organisms were detected in smears made from the lesions of both these animals.

The farm was visited in February, 1937, and the cow from which this fœtus was obtained was examined. This animal was in fair condition, ate well, and appeared quite normal. No enlarged superficial lymph-glands could be found.

CASE 2.

On 10.3.37 a 6-month-old fœtus was brought to the Laboratory from the same farm.

The abdominal cavity was distended with a large amount of blood-stained liquid, the surface of the spleen showed two whitish raised nodules about the size of split peas (fig. 7). The liver showed a number of similar whitish nodules scattered over its surface and the periportal lymph-glands were enlarged and hard.

On opening the pleural cavity the bronchial and mediastinal lymph-glands were found to be enlarged and hard.

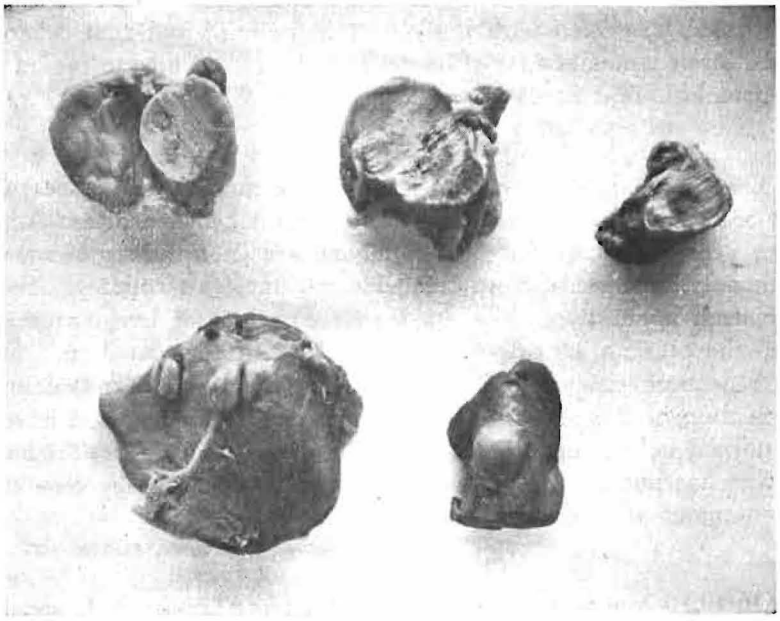

Fig. 7.

Below : Portion of fœtal liver (left) showing two incised tubercles. Portion of spleen (right) showing a tubercle.

Above : Three lymph glands showing tubercular lesions.

The three groups of glands on section (fig. 7) showed similar alterations, *viz.* irregular opaque areas surrounded by a reddish zone. In most cases the opaque areas occupied practically the whole gland. One small periportal gland showed definite evidence of calcification.

Hutyra and Marek have described similar changes in tuberculosis of the fœtus of cattle.

Smears were made from material obtained from the opaque zones of the affected glands and very numerous acid-fast bacilli indistinguishable

from the tubercle bacillus were seen. Smears from the stomach contents of the fœtus revealed many acid-fast organisms.

Guinea-pigs were inoculated as follows :—

(a) With mashed material from the affected glands.
(b) With stomach contents diluted with saline.
(c) With blood-stained abdominal liquid.

Fourteen days later the guinea-pigs in groups (a) and (b) died showing lesions of generalised tuberculosis. Guinea-pigs of group (c) were killed after 6 weeks and showed generalized tuberculosis.

Ante-mortem Examination of the Cow from which Case 2 Originated.

This cow was sent to the Laboratory for destruction.

She was in good condition, ate well, and apart from a slight muco-purulent discharge from the vagina appeared normal in every respect. Smears made from this discharge failed to reveal any acid-fast organisms. A single intradermal tuberculin test was performed on this cow and a marked positive reaction resulted. The skin-fold at the site and time of injection measured 7 mm., while 48 hours later it had increased to 38 mm. The swelling was slightly tender, and warm and œdematous. The lymphatics in the region of the site of injection were corded.

Post-mortem Examination.

Generalized tuberculosis was revealed after the carcass had been opened. The uterus felt doughy and lumpy and contained a glairy, fœtid, yellowish, flocculent discharge. The mucous membrane was covered with miliary nodules.

My thanks are due to Dr. Thomas for his histo-pathological report on this cow.

Uterus—the wall is greatly thickened with innumerable nodules at all depths and practically to the exclusion of the uterine glands. The centre of these nodules is caseous, but there is no calcification. The granulation tissue forming the nodule wall is thick : epithelioids are frequent, but Langhanse giant cells are rare and only small. There are rare acid-fast organisms.

Udder—appears to be tuberculosis free.

Mesenteric gland—large necrotic caseous centre with calcification; rather old lesions, encapsulated, with hyperaemic rind.

Ovary—growth on capsule, partly broken down, appears granulomatous in nature.

Supra-mammary lymphatic gland—nothing unusual. The lesions in the mesenteric glands appeared to be old standing ones and these were

possibly the primary foci of infection. Generalization had possibly taken place within the previous 6 to 8 weeks.

It is suggested that at the time of conception lesions of tuberculosis in the uterus were either very slight or non-existent; but that as a result of extension of the tubercular lesions in the uterine wall interfering with the nutrition of the fœtus, premature expulsion of the calf took place.

Therefore it should be borne in mind that, in some cases, tuberculosis may be a cause of abortion in cattle.

ABSTRACT.

A case of transplacental transmission of the tubercle bacillus to a bovine fœtus. (Un cas de transmission transplacentaire du bacille de Koch chez un fœtus de vache.) Spartz, L. (1937), *Bull. Acad. Vét.*, Vol. 10, p. 106.

The dam, a four-year-old cow in good condition, showed lesions of tuberculosis in the lungs, liver, retro-pharyngeal lymph glands, lymph glands of the small intestine and the right mammary and left prescapular lymph glands. The pulmonary, hepatic, retropharyngeal and intestinal lymph gland lesions were old, caseous and caseo-calcareous and those of the mammary and prescapular glands were of more recent origin and of fibrous consistency. The six-month-old fœtus presented the following lesions : a bean-sized radiating infiltration of the posterior mediastinal lymph gland, organ tuberculosis of the liver, which contained four tubercular follicles each the size of a grain of sand and lesions in the periportal lymph glands. The other organs of the fœtus, the uterus of the cow and the umbilical cord were free from macroscopic lesions. Tubercle bacilli were seen in small numbers in stained smears, taken from the mediastinal gland of the fœtus.

J. H. M.

THE ARMY VETERINARY SERVICE IN SOUTH AFRICA, 1881—1914 (Continued).

By H. H. CURSON.

CHAPTER II.

The A.V.D. and the Second Anglo-Boer War, 1899—1902.

(a) Introduction. (b) Names of Regular Officers, A.V.D. (Table V). (c) Civil Veterinary Surgeons attached A.V.D. (Table VI). (d) Some Features of Veterinary Importance: (i) Veterinary Officers in South Africa on Outbreak of War; (ii) Lack of Organisation; (iii) Veterinary Hospitals, Mobile Sections and the Ideal Remount Depot; (iv) A Few Statistics; (v) Developments in London. (e) Veterinary Remarks Concerning some Cavalry Regiments. (f) Remount Depot, Mooi, River, Natal. (g) Veterinary Surgeons who have died in South Africa during Military Service. (h) Conclusion.

(a) Introduction.

It has already been shown that although war was expected, ineffectual preparations had been made for the struggle. It was no doubt believed that the sturdy Republicans would climb down at the last minute ([48]), or if war came, that Great Britain would manage somehow or other. Great Britain, indeed, did muddle through, but at a cost of thousands of lives and millions of pounds. Those responsible for the A.V.S. were probably confident that on the outbreak of hostilities an efficient organisation could be speedily created from private practitioners, *viz.* men who knew little or nothing of military methods! ([49])

Thanks to Sir Frederick Smith, whose *Veterinary History of the War in South Africa* is a classic in professional literature, the veterinary features of the campaign have been well described. It is proposed to give as far as possible the names of A.V.D. officers and Civil Veterinary Surgeons serving in the campaign, and then to refer to some of the salient features of the struggle as recorded by Smith (1912–14).

Smith's record is divided into three sections, Part I, which covers the period from 10th October, 1899, to December, 1900 ([50]); Part II which deals with the subsequent guerilla operations terminating with the Peace of Vereeniging (31.5.1902); and Part III, a miscellaneous section including horsemastership, transport, disease, and other special features.

([48]) The lesson of the First Anglo-Boer Campaign (1880–81) had been forgotten!

([49]) It is an extraordinary fact, especially after the lessons of the past, that the authorities in South Africa in time of peace consider an A.V.S. unnecessary, *e.g.* prior to 1914 and at the present time.

([50]) An Introduction describes the preparations for war.

At the outset it should be emphasised that there was not " a single veterinary service under a central administration," but several which worked independently with disastrous results. There were the following :

(a) The A.V.D., which later employed civil veterinary surgeons;
(b) The Imperial Yeomanry, which operated on a regimental basis (see Curson, 1933);
(c) The Natal Volunteer Veterinary Corps (see *Idem*, 1934);
(d) The Canadian (see *Idem*, 1933);
(e) The Australian (see *Idem*, 1933); and
(f) The New Zealand (see *Idem*, 1933); and

All three, *i.e.* (d), (e) and (f), worked regimentally (51).

Fig. 14.

P.V.O. ILES MATTHEWS (1854-1906).

The photograph is taken from a group showing Sir George White and his staff during the Siege of Ladysmith. Notice the gorget patches on the upright collar. Died at Simla, 15.5.06.

(b) Names of Regular Officers A.V.D.

The A.V.D., on the outbreak of war, was five short of *peace* strength, possessed no reserve of officers, and, owing to neglect by the authorities, was in a dissatisfied state. " By keeping the meagre veterinary establishment of India short of ten officers, a total of 61 was obtained for the campaign, and this left no one in England but the War Office and Educational Staffs " (p. 306). Smith (1927, p. 259) gives, (i) a list of

(51) There were veterinary surgeons from the Cape Colony and the Transvaal also serving in a regimental capacity (see Curson, 1933). See footnote on p. 305 (Smith) regarding the names of the officers of the volunteer veterinary services of the Empire.

57 *regular* officers A.V.D. who participated in the Second Anglo-Boer War. Elsewhere (Smith, 1927, p. 204) he gives the number as 40 – 50; and (ii) another list of 45 *Civil Veterinary Surgeons* subsequently commissioned, the total being 102.

According to information kindly supplied by Major G. E. Oxspring, R.A.V.C., there appear to have been later 84 *regular* veterinary officers engaged, their names being shown in Tables III (29), and V (55).

In view of the discrepancies above, it is advisable to give one of the lists mentioned, and then to discuss the points of difference. Oxspring's list is as in the attached Table V.

Discussion.—The names of *regular* A.V.D. given by both number 55 and are as follows :—

Names given by both *Smith* (1927) *and Oxspring.*

Bartlett, E. B.	*Gladstone, W.	Raymond, R. W.
*Blenkinsop, L. J.	*Gillespie, S. D.	*Richardson, A. E.
*Brown, E.	Hunt, F. W.	*Rutherford, C.
*Bostock, A.	Harris, C. B. M.	*Russell, W. E.
Crawford, F. F.	*Houston, R. F.	*Rose, C.
*Crowe, W. A.	Lane, A. H.	*Sawyer, H. T.
*Cooper, J.	*Larnder, E. W.	*Short, F. J.
*Carr, F. U.	Matthews, I.	*Sharp, F. W.
Coley, J. T.	*Mann, H. T. W.	*Sullivan, H. A.
Cochrane, R. C.	Moore, J.	Shore, F. H.
Conder, G.	*Martin, E. E.	*Southey, J. B.
*Drage, F. B.	*Millar, W. M.	Smith, W. D.
Durrant, H. M.	Newsom, A. C.	*Smith, F.
*Eassie, F.	*Olver, A.	Tatam, W. J.
England, A.	*Phillips, J. J.	Todd, A. G.
*Edwards, W. B.	*Pallin, D. C.	Williams, G. M.
*Forsdyke, F. W.	*Pringle, R.	Wilson, F. W.
*Flintoff, T.	*Rayment, J. G. R.	*Williams, A. J.
Griffith, J. J.		

TOTAL 55.

Names given by Smith (1927) *but not by Oxspring.*

Lawson, E. J. Taylor, E.

Lawson is not mentioned at all by Oxspring; but Taylor, E., is given as being in South Africa between 1909 and 1914. (See Table VIII.)

Names given by Oxspring but not by Smith (1927).

Day, E. Loughlin, J. (died Wynberg 2.11.1900).
Welch, H. C. O'Donel, J. G. (died Dublin 17.12.1901).

---

* Left England *after* outbreak of War (10.10.99). See Table V.

Day, E., is given in Table III and the others in Table V. Curiously. Smith (1927) does not mention Capt. O'Donel in his *History of the R.A.V.C.*, although the name is given in his record of the Boer War (p. 27). Vet. Lieut. John Loughlin accompanied the 1st Life Guards and died at Wynberg, C.P., on 2nd November, 1900 (*Vet. Rec.*, 10.11.1900). Smith does not even mention Welch as a C.V.S. (*Vet. Rec.*, 16.12.1899).

There still remain 25 names given by Oxspring under *regular officers A.V.D.*, which are accounted for by Smith thus : (a) 23 were civil veterinary surgeons subsequently commissioned to A.V.D.; and (b) 2 were civil veterinary surgeons who were *not* subsequently commissioned.

(a) *The Names of C.V.Ss. Subsequently Commissioned.*

The 23 names are denoted by an asterisk (*) in Table V.

(b) *The Names of C.V.Ss. not Commissioned (Smith, 1927).*

Collings, R. J. Sharpe, C. M.

TOTAL 2.

The total 84, shown by Oxspring as being the number of regular officers A.V.D. who took part in the Campaign, may be summarised thus :—

55 [which figure agrees with Smith (1927)];
4 [omitted by Smith (1927)];
25 [all C.V.Ss. who were subsequently commissioned in A.V.S. during the War].

84.

Smith (1927) in the same list (S. African War, 1899–1902) containing the 23 names just enumerated under (a) gives : (i) 17 C.V.Ss. who do not appear in Oxspring's list (Table V), but are indicated in a subsequent table with an asterisk (Table VIII) as having been stationed in South Africa as A.V.C. officers *after* the Boer War.

Of these, two, Gibbs, H. E., and Rainey, J. W., qualified after the Peace of Vereeniging (31.5.1902), and therefore did not participate as C.V.Ss. in the campaign itself; and (ii) 5 C.V.Ss. who are not mentioned *at all* by Oxspring as having served as regular officers *in the A.V.D.* in South Africa. The names are :—

Argyle, E. P. (died 26.7.35. See *Jl. R.A.V.C.* VI (4)). Barnard, L.
Lawrie, A. S. Matthews, R. C.
Roberts, N. de E.

Smith (p. 108) for example, records that Barnard as a *C.V.S.* accompanied the Rhodesian Field Force in 1900.

In a nutshell, the differences above discussed are as follows :—

Smith (1927) *gives* :—	*Oxspring gives* :—
57 regular officers + 45 C.V.Ss. subsequently commissioned = total of 102. He omitted at least Loughlin, J., and O'Donel, J. G., and did not include C.V.Ss. Collings, R. J., and Sharpe, C .M., which would total 106.	84 regular officers including 25 C.V.Ss. commissioned *during the war*. He does not give in Table V the additional 22 C.V.Ss. recorded by Smith (1927) as also being subsequently commissioned. Of these, as shown above, two qualified after Peace.

If the dates of commission were known, perhaps the discrepancy of about 20 could be explained.

Probably the most reliable guide would be the Medal Roll at the War Office (plus deaths); but the perusal of such a register can only be undertaken in person.

Much space has been devoted to a consideration of the names of the A.V.D. regular officers who served in the Boer War but without entire success. Nevertheless this is felt to be justifiable for this aspect is generally neglected in the recording of veterinary events ([52]). While it is true that the profession often makes the man, it is more true that the man makes the profession !

(c) Civil Veterinary Surgeons attached A.V.D.

If there is a little uncertainty regarding the names of *regular* officers A.V.D., there is chaos concerning the C.V.Ss., especially those employed on Sea Transport !

All that is proposed is to amplify a list of C.V.Ss. *who served in South Africa* as given by Smith (1927).

TABLE VI.

LIST OF C.V.Ss. ATTACHED A.V.D.WHO SERVED IN SOUTH AFRICA.

The original list is given by Smith (1927). As in several cases the name and initial(s) given by Smith cannot be traced in the *Register*, the names in such cases believed to be the correct names (and which can be traced in the *Register*) are given in the Table. The names which cannot be traced in the *Register* are indicated accordingly. An endeavour was made (*Vet Rec.*, 17.8.35) to trace certain individuals, Dr. F. Bullock being most helpful in this regard (his letter of 19.8.35).

([52]) This feature has received scant attention in the *Official History of the Great War* 1914–18, an exception being the Veterinary Report of the Campaign in German South West Africa by Col. J. Irvine-Smith, M.R.C.V.S. See p. 406.

C.V.Ss. who remained in South Africa after the campaign receive special consideration in Table VII, but in Table VI their names are indicated by an asterisk. The names of those who died during 1899–1902 in South Africa are italicised. Messrs. Harvey, Hill, and Jarvis served in the Cape C.V.D. during the Rinderpest epizootic of 1897.

Anderson, R.	Glasgow	1889
Anderson, R. G.	Glasgow	1897
Ashworth, E.	New Edin.	1900
Aitchison, D. A. D.*	———	
Armstrong, R.	———	
Anthony, H. L.	London	1901
Aggio, C.	New Edin.	1886
Brownrigg, P.	London	1894
Bell, J. H.*	———	
Brownless, J. W.	Edinburgh	1897
Buck, J.*	———	
Bell, J. P.	New Edin.	1887
Balfour, G. W.	New Edin.	1893
Butcher, G. H.	London	1896
Byrne ?	Cannot trace.	
Beattie, R. S.	Cannot trace.	
Brand, J.*	———	
Bowhill, T.*	———	
Barber, G. H.	London	1901
Brown, J. B.	Cannot trace.	
Barber, K.	Cannot trace.	
Beilby, R. E.	Edinburgh	1894
Cockburn, W. L.	New Edin.	1896
Conacher, P.*	———	
Crawford, J.*	———	
Christy, J. M.*	———	
Cowx, J. B.	New Edin.	1896
Campbell, C.	New Edin.	1885
Crone, J. R.	Edinburgh	1891
Chellew, S. G.	London	1899
Collings, R. J.	London	1900
Crossley, F.	London	1900
Cockcroft, J. E.	Edinburgh	1900
Carpenter, J.	New Edin.	1892
Cunningham, J. A.	Glasgow	1885
Chalmers, J.*	———	
Carless, F. J.*	———	
Conisbee, A.	New Edin.	1884
Cameron, G. T.	Cannot trace.	
Clark, C. C.*	———	
Cordy, C. H.*	———	
Douglas, J.	Cannot trace.	
Dyson, C.	New Edin.	1884
Donaldson, J.	Glasgow	1897
Dunphy, J. P.*	———	
Dyson, E. M.	Cannot trace.	
Duff, J. A.	Edinburgh	1898
Davies, H. E. H.	———	
Dale, T. H.*	———	
Dunning, F. J.*	———	
Drabble, J. S.	New Edin.	1900
Ensor, E. T. C.	———	
Evershed, P. M.	London	1897
Edwards, E. R.	London	1889
Edgar, J. I.*	———	
Evans, W. G.*	———	
Ellis, G.	New Edin.	–
Fyrth, W.*	———	
Farrar, J. R.	Edinburgh	1888
Fernandes, H. W.	London	1899
Forrest, J.*	———	
Fountain, F. C.	New Edin.	1898
Farrant, A. L.	London	1895
Fowler, A. B.	New Edin.	1895
Fairclough, J.*	———	
Frood, J. L.	London	1899
Fern, E.*	———	
Gavin, F. C.*	———	
Grist, A. G.*	———	
Golden, F. G.	New Edin.	1885
Goundry, C.*	———	
Green, W. G.	London	1898
Goulé, A.*	———	
Garden, G.	Edinburgh	1900
Gridley, W. B.	London	1889
Gresham, F. B.	Edinburgh	1897
Hodder, A.*	———	
Hoggan, T. R.	Edinburgh	1892
Hawes, A.	London	1893
Holland, A. B.	London	1893
Hines, A. J.	London	1897
Henderson, R.	Edinburgh	1896
Hill, W.*	———	
Hulseburg, J. H.	London	1897
Harvey, G. J.	London	1891
Hepburn, W.	Edinburgh	1900
Hingston, L. C.	Glasgow	1878
Hearn, C. G.	London	1900
Hamilton, J. R.*	———	
Hart, A.	Glasgow	1894
Hazell, H. A.	Glasgow	1892
Hogg, T.*	———	
Hirst, W. H.	———	
Jackson, A. F.*	———	

Jowett, W.*	———	
Joyce, J. F.*	———	
Jarvis, E. M.*	———	
Kellett, E.*	———	
Knight, L. P.	Cannot trace.	
Kenny, H. B.	Edinburgh	1890
Kidd, W.	———	
Kelland, P. J.	London	1901
Kendall, W. A.	Cannot trace.	
Kilpatrick, T. T.	Glasgow	1901
Lane, A. J.	London	1892
Lund, J. W.	New Edin.	1892
Lalor, A. D.	London	1890
Leyshaw, H.	Edinburgh	1898
Lindsay, F.*	———	
Malone, W.	New Edin.	1894
Magill, J. M.	Glasgow	1882
Miller, R.	Glasgow	1896
Montgomery, G.	Edinburgh	1892
Masheter, J. W. H.	Edinburgh	1893
McVean, H.	Glasgow	1896
Main, A.	Glasgow	1895
Mason, H. E. T.	London	1894
Mitchell, A.	New York	
MacDonald, R.*	———	
McNae, A.*	———	
May, G.*	———	
McGrath, J. J.	Cannot trace.	
McKie, W.*	———	
Neill, J.*	———	
Newcombe, H. H.	Cannot trace.	
Neale, C. R.*	———	
Norgate, C. E.	Edinburgh	1892
Oliver, H. O.*	———	
O'Neill, O. A.*	———	
O'Neill, H. J.	London	1897
O'Brien, J.	Edinburgh	1900
Pollard, J.*	———	
Phelan, H. W.	New Edin.	1896
Peddie, J.*	———	
Plunkett, R. A.	London	1899
Pakeman, W. G.*	———	
Pilkington, J. K.*	———	
Pye, W. M.*	———	
Parker, T.	London	1900
Parker, J. M.	———	
Parker, C. E.	London	1895
Pawlett, F. W.	Edinburgh	1899
Pringle, J. N.	Edinburgh	1892
Pickwell, G. H.	London	1885
Rennie, T.	Glasgow	1897
Rees, W. D.	Edinburgh	1897
Rowston, W. N.	London	1900
Robinson, J. M.*	———	
Runciman, B.*	———	
Reece, R. S.	Cannot trace.	
Stockman, S.*	———	
Smith, C. E.	London	1865
Smith, J. I.*	———	
Stephens, S. L.	London	1893
Symes, T. J.	London	1898
Scott, W.	New Edin.	1883
Stewart, C. D.	London	1897
Sturge, H.*	———	
Small, J. P.	Glasgow	1900
Skues, F. M.*	———	
Sharpe, C. M.*	———	
Stranaghan, D.*	———	
Smart, A. C.	Edinburgh	1896
Sellers, A. J.	Edinburgh	1895
Scarlett, H.	London	1888
Stableford, J. P.	New Edin.	1901
Sowerby, M. H.	New Edin.	1901
Somers, H. L.	London	1885
Sykes, G.	———	
Stokoe, R.*	———	
Souter, E. S.	Glasgow	1900
Stevens, W. S.*	———	
Stevenson, G. F.	New Edin.	1889
Taylor, W. E.	London	1875
Tate, J. M.*	———	
Tasker, H. K.*	———	
Twist, C. R.	New Edin.	1900
Trydell, F. W.	London	1897
Thompson, J.	Edinburgh	1890
Tamblyn, D. S.*	———	
Thomas, R. P.*	———	
Tufts, S. R.	Edinburgh	1893
Tranter, T.	Edinburgh	1898
Vans-Agnew, E.	Cannot trace	
Webb, H. M.*	———	
Wadsworth, W.	London	1891
Wells, C. E.	London	1896
Wardrope, J. R.	Edinburgh	1897
Waugh, G. N.	Glasgow	1897
White, F. F. G.*	———	
White, W. T.	Cannot trace.	
Watson, R. F.	Edinburgh	1896
Wilshaw, E. A.	London	1900
Walker, J.*	———	
Wall, F. A.	Edinburgh	1865
Wall, R. F.	Edinburgh	1896
Wilks, P. A.	London	1892
Wilson, E. A.	Cannot trace.	
Young, J. C.	Cannot trace.	

TOTAL 199.

If the figure 199 be added to 45, *i.e.* the number of C.V.Ss. stated by Smith (1927) to have been subsequently commissioned, we arrive at a total of 244 C.V.Ss. who saw service in South Africa.

Since Smith (p. 309) states "The total number of C.V.Ss. engaged for the War was 283," the difference between 283 and 244 represents the number employed on Sea Transport Service, *i.e.* 39!

The following are the names of C.V.Ss. (mentioned in the *Veterinary Record* of 1899–1902) who, not being included in the preceding list, are naturally assumed to have done Sea Transport duty; but judging from the *Veterinary Record* several saw service in South Africa.

C.V.S. ATTACHED A.V.D. NOT MENTIONED BY SMITH (1927).

The following names can be traced in the *Register* :—

Arkcoll, W. J.	London	1872	O'Donoghue, J.	Edinburgh	1888
Barrass, G.	New Edin.	1899	Paterson, J. H. G.	Glasgow	1897
Cannon, G. T.	London	1889	Prime, T. F.	London	1892
Carrick, C. J.	New Edin.	1891	Prime, W. H.	London	1894
Clarke, J. S.	Edinburgh	1891	Richmond, F. J.	New Edin.	1880
Clode, E. E.	London	1893	Ryan, H. T.	London	1898
Doyle, T. G.	Edinburgh	1898	Sugden, A. H.	London	1889
Flanagan, W. G.	London	1901	Stokes, E. E.	Edinburgh	1899
Hayes, M. H.	New Edin.	1883	Towers, J. A. R.	New Edin.	1890
Hewlett, K.	London	1900	Whipp, H.	New Edin.	1898
MacGregor, C.	Edinburgh	1886	Wilson, C. W.	London	1900
McGregor, John	Edinburgh	1888	Wilson-Barker, J.	London	1889
Munro, A. M.	New Edin.	1897			

There are in addition many names which cannot be found in the *Register*.

J. O'Donoghue, who had been a member of the Cape Veterinary Division during 1897–8, joined the South African Light Horse and presumably transferred as C.V.S., A.V.D. After the war he left South Africa and took up medicine, later graduating as M.D. (Boston). C. MacGregor later joined the Scottish Horse, and the name appears on the monument to the Scottish Horse erected at Kensington, Johannesburg. He is stated to have been a sergeant with regimental number 25,868, and is referred to as "Veterinary." He died of enteric fever after receiving wounds at Witpoort (*Vet. Rec.*, 1.3.02). Fig. 19 shows the tablet erected early in 1904 on the walls of the College to "the memory of the veterinary surgeons who died . . . in the South African War" (*Vet. Rec.*, 26.8.1933.) Cannon and Stokes remained in South Africa.

It should be noted that not all C.V.Ss. were M.R.C.V.S., nor were they all recruited in Great Britain. Parker, J. M., and Tamblyn, D.S., were graduates of McGill University. Mitchell, A., graduated at the New York State Veterinary School, and MacDonald, R., qualified at Ontario Veterinary College. Robinson, J. M., was from Australia but did

not possess a diploma. Several in South Africa joined the A.V.D. as C.V.Ss., *e.g.* Fern, E., Thomas, R. P., and Walker, J. Neale, C. R., Brand, J., and Towers, J. A. R., accompanied troops from New Zealand and then apparently transferred to the A.V.D. as C.V.Ss.

Smith (p. 309) in referring to the Sea Transport Service, reports that " the work of the British Veterinary Surgeons gave satisfactory results, but the foreigners engaged by the Remount Department . . . brought great discredit on the British Service." Not all transports were provided with veterinary assistance (*Vet. Rec.*, 14.4.1900).

Fig. 15.

C.V.S. J. IRVINE-SMITH, O.C., and the N.C.O's of A 2 Indian Field Veterinary Hospital, De Aar, 1900.

(d) Some Features of Veterinary Importance.

(i) *Veterinary Officers in South Africa on Outbreak of War.*

On October 10th, 1899, there were apparently 19 regular veterinary officers in South Africa (see Table III) distributed as follows :—

Natal—Col. Iles Matthews, P.V.O., Capt. R. W. Raymond, Capt. A. C. Newsom*, Lieuts. J. T. Coley, E. B. Bartlett, W. J. Tatam*, F. W. Wilson*, C. B. M. Harris*, Major F. F. Crawford*, and Lieuts. G. M.

Williams, F. H. Shore and R. C. Cochrane. Those marked with an asterisk had recently arrived from India with reinforcements, and the first eight names were out of action from 2nd November, 1899, to 28th February, 1900, for they were besieged in Ladysmith ([53]).

Cape Colony—Lieuts. A. England (at De Aar—his letter of 6th February, 1927) and A. H. Lane, and probably H. M. Durrant, F. W. Hunt, and J. J. Griffith. Lieut. W. D. Smith was at Mafeking, but owing to the siege of 6½ months duration, his movements were naturally restricted.

With Col. Plumer's force on the Transvaal-Southern Rhodesia border was Lieut. J. Moore.

Of the above 19 A.V.D. officers one-third were soon besieged in Ladysmith and Mafeking, leaving a dozen for remount and veterinary duties until reinforcements arrived.

Since the P.V.O. was in Ladysmith, no one with sufficient authority was available until November 19th, 1899, when Lieut.-Col. Rayment reached Cape Town. After some delay he was appointed Acting P.V.O.

(ii) *Lack of Organisation.*

The pre-war position has already been discussed, but even during the campaign anomalies existed. Not only was the Director of Transport and Supplies made responsible for the Remount and Veterinary Services (Smith 1927, p. 203), the Veterinary Department being subordinate to the Remount ([54]); but the activities of the Veterinary Service itself were largely controlled during the last six months of the campaign by a combatant officer. Col. Long, who by Army Order of December 1st, 1901, was appointed Inspector of Veterinary Hospitals (Smith, p. 194), seems however, to have succeeded in getting things done, for Smith (p. 198) records that " The more they asked for, and the more pressing the construction [*i.e.* of veterinary hospitals] was represented to be the better the Inspector of Hospitals seems pleased. He harrassed the Engineers . . . and galvanised life into the construction of stabling . . .". In 1902, as can be expected, Col. Iles Matthews (the P.V.O.) and Col. Long often came into conflict, the former, for example, attempting to provide column commanders with veterinary aid, whereas the latter " wished the veterinary staff to be withdrawn from columns and placed on the line " (p. 223).

([53]) Other veterinary officers in Ladysmith during the Siege were Col. H. Watkins Pitchford and Lieuts. W. M. Power, S. T. Amos, and P. Byrne of the Natal Volunteer Veterinary Corps, and Messrs. W. Pye and E. A. Hollingham serving in the Imperial Light Horse.

([54]) It was not until 1913 that the D.G., A.V.S. became independent of Remount control.

Other examples of lack of organisation were :—

(a) When the Veterinary Field Hospitals arrived from India (the Home Government not having any), it appeared to the Remount Service that these should be placed under Remount control. Accordingly Army Order 2 of January 30th, 1900, instructed that " when Veterinary Field Hospitals are located in the same place as Remount Depôts, the Veterinary Field Hospitals will be attached to the Remount Depôt . . . Horses which it is desired to return to Remount Depôt for rest will be inspected by a Remount or Veterinary Officer, whose opinion will be taken." Fortunately, on February 2nd, 1900, a fresh order was published which put right the status of the Veterinary Service.

On several occasions (Smith, p. 200) Remount Officers were most aggressive and ignored veterinary officers, especially the unfortunate C.V.Ss. who were without rank and therefore without authority. As Smith (1927, p. 204) comments, " when they died they were given a lieutenant's funeral " !

(b) Starvation of horses in Cape Colony.—Soon after his arrival the Acting P.V.O. agitated for a more liberal diet for animals in the Field. " The authorised scale laid down was 12 lbs. grain and grazing" (Smith, p. 14). Since there was no grazing, he pointed out the need for a regular hay ration, but the authorities refused. The result was " the breakdown of the Cavalry Division and its annihilation for the remainder of the war as an effective arm " (Smith, p. 16).

Smith (p. 28) emphasises " that neither foresight nor judgment were absent from the veterinary service at the War Office. Most of the difficulties were predicted and might have been avoided had the Department been permitted to work out its own organisation."

(iii) *Veterinary Hospitals, Mobile Sections, and the Ideal Remount Depôt.*

(a) Smith (p. 145) gives a good idea of the chaos existing at the end of 1900. He states : " The Army was riddled with glanders, while mange cases existed by thousands."

We have seen that the first of the Indian Veterinary Field Hospitals had arrived before the outbreak of war. By March, 1900, three, each " capable of subdivision into two complete self-contained establishments " (p. 29), had been landed. Altogether ten arrived from India.

During the last month of 1900 " one thoroughly equipped Veterinary (Base) Hospital " was erected, this at Elandsfontein (now Germiston). Here hospital accommodation was provided for 500 sick and general accommodation for over 2,000 horses. By the end of May, 1902, fifty similar hospitals had been established, and at these were nearly 20,000 horses and mules. The remainder of the sick, about 8,000 – 10,000, were on debility farms under Remount control.

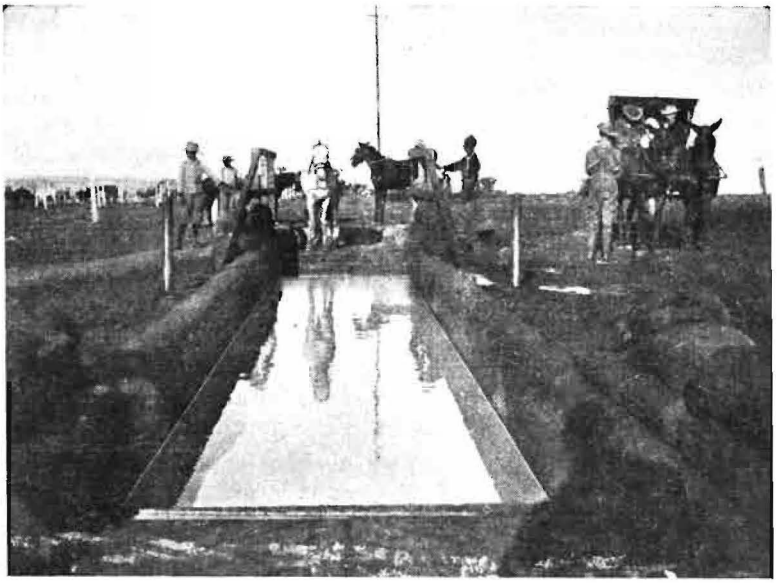

Fig. 16 a.

Fig. 16 b.

Dipping horses for mange in a tank built at Kimberley in 1901 by C.V.S. J. Buck.

On the day peace was proclaimed the Veterinary Service consisted of : Veterinary officers, 63; Civil Veterinary Surgeons, 113; European dressers, 79; Indian dressers, 528; Civil farriers, conductors, and clerks, 217; and Natives, 3,547.

The majority of the personnel of the hospitals (excepting Indian) was borrowed from other units. Only in 1903 was provision made for a permanent subordinate staff.

(b) That veterinary officers possessed initiative may be judged from the following examples :—

When General French left Bloemfontein on April 22nd, 1900, " to clear General de Wet out of the South-east of the Free State," his losses in horses were considerable. He was followed from Edenburg by the 8th Division, which was commanded by General Rundle. Lieut. A. H. Lane, S.V.O. 8th Division, seeing the abandoned horses, "was permitted by General Rundle to arrange measures for sweeping up the animal wastage . . . and a Mobile Veterinary Detachment resulted " which destroyed the unfit, but collected the fit (Smith, p. 57). This was the origin of the Mobile Veterinary Section officially established in 1913.

Another example of initiative and of the benefit of mobility may be seen in Major Rutherford's scheme in Natal in May, 1900, when as S.V.O. Natal Army he was allowed " to render both sections of No. 6 Field Veterinary Hospital mobile, and it was arranged that a section should be attached to each Infantry Division. For the first time in the history of the British Army it was accompanied in the field by some organised machinery for the treatment of the sick and injured " (Smith, p. 85).

Cases of "conspicuous ability and power of organisation" were also to be seen among the C.V.Ss. Smith (p. 200) mentions particularly J. Irvine-Smith, J. Buck, and J. M. Parker, who did excellent work in hospital management.

(c) It was left to a veterinary officer, Capt. F. Eassie, to devise the ideal Remount Depôt. At Fischer's Farm near Bloemfontein he constructed "a large circular track . . . so arranged that each horse enclosure communicated with it by means of a large gate. On the gate being opened the . . . occupants could be driven into the track and in one or two lessons readily learned to start trotting steadily . . . In half an hour or so 500 horses or so had been exercised." Arrangements also existed for feeding, watering, and catching individuals for shoeing or other veterinary attention. Further. Eassie conserved water and "grew more material than the horses could consume." Needless to say, Eassie's system has been frequently employed since 1900, particularly during the Great War.

Smith (p. 74) mentions that "difficulty was at first experienced in obtaining veterinary officers for hospitals. They preferred to serve under

a general who would probably recognise their worth, rather than be left in solitude and forgotten on the line of communication" (p. 74).

(iv) *A Few Statistics.*

Smith records that "the report written by the Remount Department after the war, has, unfortunately, never been published. In it are statistical tables of income and expenditure of horses, but all these labour under the great disadvantage of unaccountable deficiencies, amounting to as much as 30,000 animals in a year!" (p. 142).

The total losses for the war are estimated by Smith (p. 226) as follows :—

	Horses.	*Mules.*
1899–1900	110,028	29,113
1901	142,603	14,433
1902	73,442	7,853
	326,073	51,399

He calls the above dead loss, *i.e.* the total number of animals which actually disappeared out of the total paid for, and exclusive of those picked up in South Africa ([55]). These losses are 66.88% for horses and 35.4% for mules.

(v) *Developments in London.*

During the war two of the most important events from the veterinary aspect were (a) the placing of the A.V.S. under the Remount Department. In November, 1901, the supreme head was changed from Adjutant-General to Quartermaster-General (*) and (b) the assistance of Council R.C.V.S. in the struggle for improved conditions. "Thanks to repeated representations of the Council R.C.V.S. to the War Office, events took a somewhat unexpected turn in April, 1902, when a new veterinary warrant was issued" (Smith, 1927, p. 207). However, the main point, *viz.* compound rank (prefix "veterinary" to rank), had not been settled; but the opportunity was taken to draw the attention of the authorities to this and other disabilities. The letter in which this was done was dated June 7th, 1902, and strictly falls under Chapter III of this work, but it may be stated that it resulted in the appointment of a Committee—the Hardwicke Committee—in September to consider "what alterations were necessary in the terms under which officers now served in the A.V.D." (Smith 1927, p. 208). The results of this investigation will be shown in the succeeding Chapter.

([55]) Smith (footnote, p. 226) estimates the total captures at over 125,000 animals.

(*) See Smith (1912–14), p. 192, and Smith (1927), p. 216 for footnotes concerning status of Director-General A.V.S.

(e) VETERINARY REMARKS CONCERNING SOME CAVALRY REGIMENTS.

(See Table I.)

1. *Composite Regiment, Household Cavalry.*—Each regiment of the Household Cavalry supplied a squadron for the Composite Regiment. Vet. Capt. Drage and Vet. Lieut. J. Loughlin served in the campaign, the latter dying at Wynberg Hospital on 2nd November, 1900. After arrival at Bloemfontein in March, 1900, the regiment had half of its "strength (516) ineffective" (Smith, p. 54). [Letter 101/32 of April, 1932, from Col. D. C. Boles.]

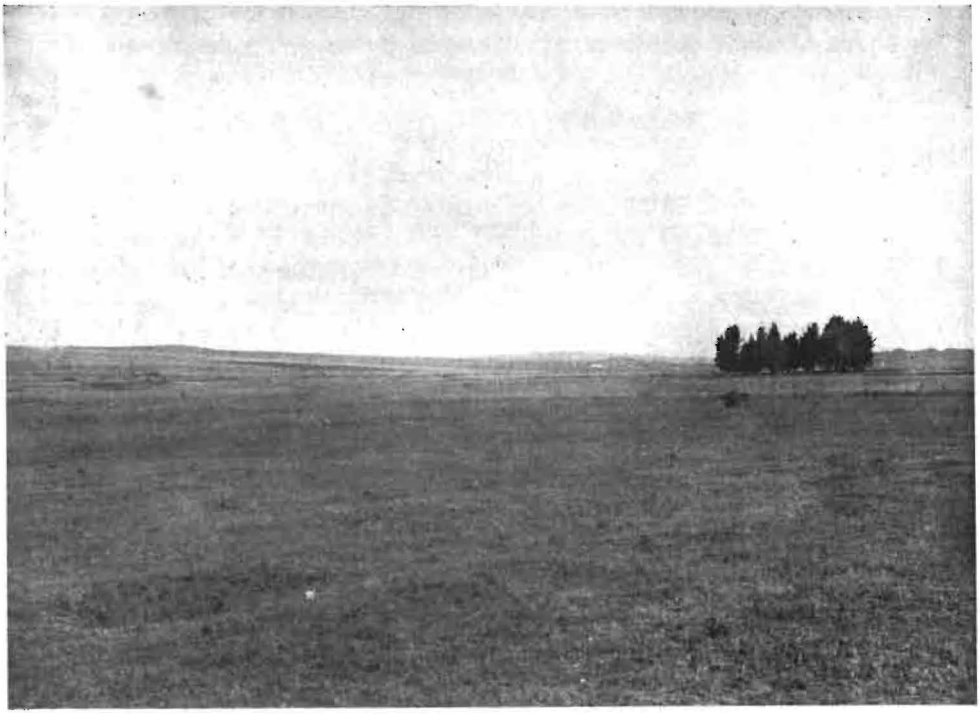

Fig. 17.

SITE OF BRITISH PRISONERS' CAMP, WATERVAL, NEAR PYRAMIDS, PRETORIA.

It was here that Vet.-Lieut. F. H. Shore was confined. The site was previously occupied by the Stables of the Veterinary Research Laboratory, 1897, under (Sir) Arnold Theiler. — Note the cemetery.

2. *2nd Dragoon Guards.*—During the year 1902 the wastage of horseflesh was terrible, no fewer than 748 animals out of the 775, with which they opened the campaign, being lost. [Letter 37/4 of February 29th, 1932, from O.C.]

3. *Royal Scots Greys.*—According to a record of the Regiment (1899–1901) by Q.M.S. James McElligott ([56]), Lieut. H. A. Sullivan. A.V.D., accompanied "B" squadron and Headquarters on S.S. *Ranee* from Glasgow in November, 1899. Veterinary officers who were attached during the war were Lieut. J. W. Brownless (a C.V.S.), and C.V.Ss. E. T. C. Ensor and Gamble.

4. *3rd Hussars.*—The V.O. was Lieut. W. M. Millar. The regiment arrived at Durban on December 29th, 1901, and January 5th, 1902. "During the sea voyage between Bombay and Durban the heat below decks was very trying to the horses, especially on board the *City of Vienna,* where they could not be brought up on the upper deck, and the accommodation and means of ventilation left much to be desired. Notwithstanding these drawbacks, the casualties were exceedingly small."

During a "drive" in the N.E. Orange River Colony at the end of February, a heavy loss of horses resulted, those from India being far from acclimatised. On this "drive" alone 70 horses died from exhaustion and exposure.

The casualties among the horses from January to May 31st, 1902, were : died, destroyed, etc., 407; returned to remount and sick depots, 575. Total, 982 horses. On leaving in October, 1902, the horses were handed in at the Remount Depot, Mooi River, Natal. [Letter 3316/2/3 of 18.3.32 from Lt.-Col. W. R. Tylden-Wright.]

5. *6th Inniskilling Dragoons.*—Smith (p. 227) states that "regimental losses during the war are most difficult to obtain and in many cases . . . no record appears to have been kept. There is one remarkable exception. Capt. A. S. Head ([57]), A.V.C. (R), who served with the Inniskilling Dragoons . . . kept the most careful notes . . .". His information is shown as a table by Smith (p. 228), but it is sufficient here to state that from November, 1899, to June, 1902, the regiment covered (measurements in a *straight* line) 6,116 miles and 3,750 horses were expended. Of 4,290 horses used, there were 4,170 admissions, and of the latter as few as 163 were due to bullet wounds and 3 to shell fire.

6. *9th Lancers.*—The regiment suffered heavily on the march to Bloemfontein and when inspected by the Acting P.V.O. at the beginning of April, 1900, had half of its strength ineffective (Smith, p. 54).

7. *10th Hussars.*—"A" Squadron and one troop of "B" Squadron proceeded from Aldershot by rail to Birkenhead on 3rd November, 1899, and embarked per S.S. *Ismore* on the 4th November, 1899, accompanied by 200 horses.

([56]) Kindly furnished by the O.C. (19.12.34).

([57]) See "The wear and tear of horses during the South African War" by Lieut. A. S. Head. *Jl. Comp. Path. & Ther.,* 1903.

On the morning of 3rd December, 1899, the ship struck on the reefs at Columbine Point, Paternoster Bay, 90 miles north of Table Bay. The alarm was sounded and troops were mustered on the troop decks, and marched to their respective life boats; the most perfect discipline and order prevailed. By daybreak the work of disembarkation in the ship's boats had commenced. The ship's officers assisted nobly in the work, but the crew disgracefully deserted in one of the boats, and were the first to reach shore.

The inhabitants of Paternoster village had observed the ship's lights, and sent out fishing boats and assisted in the work of disembarkation by guiding the boats through a navigable course among the formidable reefs.

Every effort was made to rescue the horses, but with scant success. The decks, on which were the stables, being a considerable height above water, it was necessary to force them to take the plunge. The system of lowering them was quite out of the question, as the boilers were flooded and all machinery rendered unserviceable. The ship was breaking up and time would not permit of manual methods. With a few exceptions, despite the endeavours to induce them to follow the boats, the horses that took the plunge immediately swam out to sea. Some were however towed ashore from the boats, and a few found their way to land during the day, but several of these were so badly cut by the rocks that they had to be immediately destroyed.

Only 19 of the whole number were saved, but all the men were brought safely to shore.

Field forges and vehicles and vast quantities of forage, rations, harness and saddlery, tents, blankets, and stores were lost.

The rescued horses were sent by march route to rail-head and thence by rail to Stellenbosch. The troops were bivouacked on the shore, and food and drinking water were obtained from Paternoster village.

On the 5th December, 1899, the troops marched to St. Helena Bay, where the transport *Colombia* had been sent to take them to Cape Town, where they disembarked on the 7th December, 1899. They entrained for Stellenbosch Remount Siding the same day. From this date until 19th December was occupied in drawing tents, stores, saddlery, equipment, arms, and clothing to replace that which had been lost.

Argentine remounts were received, and were trained with such success that they were fit to take their place in the ranks when the Detachment left by rail on the 21st instant, and joined the Regiment at Arundel. [Letter 32/1 of 15.10.34 from Lt.-Col. C. W. M. Norrie, O.C.]

8. 14*th Hussars*.—On the outbreak of hostilities the regiment was " ordered to the seat of war at once, [but] this was countermanded, owing to an outbreak of pink-eye". The regiment, however, sailed during the

middle of December, 1899. [Short Regimental History of 14th/20th Hussars.]

9. *16th Lancers.*—An interesting account of the horses used by the 16th Lancers is contained in a letter from Brig.-Gen. M. L. MacEwen, C.B., dated September 30th, 1934, and kindly provided by Col. G. Clark, O.C. 16th/5th Lancers. He states : " The Regiment sailed from Bombay for South Africa in three ships, taking all their horses with them. They were then mounted entirely on walers, except a few Arabs taken as officers' 3rd chargers. We anchored off Durban for a few hours and then went on to Port Elizabeth where all horses were slung overboard into lighters and landed quite comfortably as the sea was calm.

" The walers had been fed on grain in India and did not take at all kindly to oats on reaching the front, opposite Magersfontein. That, combined with want of water during the Relief of Kimberley, pursuit from Kimberley, and march on Paardeberg, fairly did them in.

" We then had a pretty big draft of Argentines, real bad stuff at about £5 a piece ! You could neither attack nor run away on them !

" After that mixed drafts from the remount depots—of all grades.

" Our few Arabs lasted out everything else, but could only have carried the weight as chargers. The local Basuto ponies were as good as or better than the Arabs, but were hard to get."

10. *17th Lancers.*—The regiment departed from England for service in February, 1900; a total of 505 horses was embarked. Of these " 40 succumbed during the voyage . . . the losses being mostly due to catarrhal fever". [Letter from Adjutant 17th/21st Lancers, 15th October, 1934.]

11. *18th Hussars.*—Lieut. F. H. Shore, A.V.D., was one of the two veterinary officers ([58]) present at Talana (October 20th, 1899), where he was taken prisoner. He was sent to Waterval Prisoners of War Camp at Pyramids, near Pretoria.

(f) Remount Depôt, Mooi River, Natal ([59]).

By **F. J. Carless,** formerly C.V.S., A.V.D., Mooi River.

The depôt was opened, early in the first month of hostilities, by Col. R. Stevenson, Inspector of Remounts, primarily as a collecting depot for locally purchased horses and mules. The farm " Mount Victoria," about four miles east of Mooi River, in area about 3,000 acres, was rented from Mr. R. D. Hall. Stable accommodation was limited to a large cow-

([58]) The other, Lieut. Bartlett, A.V.D., took part in the subsequent retreat to Ladysmith (Smith, p. 10).

([59]) See Smith, pp. 23, 84, 125, 201, and 225.

shed and about a dozen loose boxes, but plantations of pines and wattles provided shelter in addition. The farm was efficiently fenced and well watered. Very soon, however, owing to return to the depot of many debilitated animals from the front, and heavy purchases of untrained mares and geldings, the grazing accommodation proved insufficient and an adjoining farm "Shackleton" (2,800 acres) was rented from Capt. W. H. Stevenson and some 4,000 acres of Town Lands was enclosed and divided into paddocks. At this time (December-January) the strength of the depôt was from 2,000 – 3,000 horses and mules. Sick lines were established, but with no hospital accommodation beyond the ordinary field equipment. The original staff consisted of Col. R. Stevenson, Inspector of Remounts, Capt. R. Sparrow, an officer of Dragoons, Capt. G. M. Williams, A.V.D., Lt. H. M. Durrant, A.V.D., and F. J. Carless, C.V.S./A.V.D., with a staff-sergt.-farrier and several farriers from Imperial Regiments (the latter being replaced early in 1900 by civilian farriers), conductors, and dressers. D. Quinlan, C.V.S./A.V.D., joined the staff early in 1900 and was in charge of the sick lines for some months, under most difficult conditions. Apart from debility and lameness, there were frequent cases of glanders, mange, etc. Horses and mules had by this time begun to arrive from overseas. The Officer in Charge and one of the veterinary officers took over these animals on arrival at Durban, and the Show Ground and stabling at Lord's Ground were used as receiving depôts, whence the animals were drafted as quickly as possible to the Mooi River depot. Durban Show Ground soon became crowded with casualties. Cases of ship pneumonia, laminitis, wounds, etc., very soon occupied all available stabling. Large kraals were erected for the accommodation of new arrivals from oversea prior to testing with mallein. Glanders was very frequent amongst shipments from Spain and other European ports and in one case, in a cargo of 1,200 mules ex Gibraltar, active glanders was found on every deck on arrival of the ship at Durban. In this case about a thousand reacted to the mallein test and were destroyed and buried in large open graves on the Eastern Vlei adjoining the Show Ground. Several thousand animals, mostly glandered mules, were destroyed and buried there. Col. R. Stevenson returned to England in December, 1900. His position was filled by Majors de Vaux and Noble and the Mooi River depôt came under command of the newly appointed Director of Remounts, Capetown. About the same time I was invalided to hospital and sent to England, discharged "permanently unfit," June, 1901. In spite of every effort I have failed to get reliable data to fill in the interval between my enforced retirement and the end of the war. Of the veterinary officers, the senior men whom I can call to mind were Capt. G. M. Williams, followed by Capt. Leaning, Capt. J. Andrews and Capt. Aitken. The two latter officers would have been employed mostly in dealing with post-war work. The depôt was not finally closed until 1914. At the close of the War enormous numbers of loot horses came

for disposal. As might be expected, the majority were in a pitiful condition. Glanders was rife and sarcoptic mange particularly virulent. It would be no exaggeration to put the actual deaths through "mange" infection at tens of thousands. It is estimated that over 30,000 carcases were buried at Mooi River, but of course these embraced deaths from all causes.

(g) Military Veterinary Officers who have Died in South Africa.

It will be noted from the dedication that the intention is to honour not only the regular officers A.V.D. and the C.V.Ss. attached thereto, who died during the Second Anglo-Boer War, but *all military veterinarians* who have lost their lives in this country.

Further particulars are given herewith :—

Fig. 18a.

GRAVE OF F. F. CRAWFORD, A.V.D., FORT NAPIER, PIETERMARITZBURG.

Regular Officers A.V.D.

Vet.-Lieut. H. Wilkinson died at Pietermaritzburg on October 7th, 1895, and was buried at the Military Cemetery, Fort Napier, but no trace can be found of his grave. [Letter of 12.8.35 from Secretary, Natal Soldiers' Graves Association.] See Table III.

Vet.-Major F. F. Crawford died at Pietermaritzburg from enteric fever on January 16th, 1900, and was buried at the Military Cemetery, Fort Napier. His resting place is shown in fig. 18a. See Table III.

Vet.-Capt. J. Loughlin died of hepatitis at Wynberg on November 2nd, 1900, and according to Mr. C. H. Wadlow, M.R.C.V.S. (see *Vet. Rec.*, 26.8.1933) was buried at Wetton Road Cemetery. In August, 1935, the gravestone was repaired by the Cape Soldiers' Graves Association. [Letter 24.9.35.] See Table V.

Major A. E. Richardson. See *Vet. Rec.*, 28.11.36.

C.V.Ss. attached A.V.D.

Hirst, Wm. H., died of enteric fever at Boshof, O.F.S., on May 5th, 1900; but the Magistrate there reported [his 9/1/2 of December 12th, 1933] that neither the name nor the grave could be traced in his district.

Fig. 18 b.
GRAVE OF D. C. BARNINGHAM, I.Y., PRETORIA.

As at Boshof there are graves of unknown soldiers "it would seem possible that the grave of W. H. Hirst might be one of these, particularly as two or three . . . refer to persons who were buried during May, 1900" ([60]).

Ensor, E. T. C., died of enteric fever at Kroonstad, O.F.S., on June 9th, 1900. The Magistrate, Kroonstad, reported [his 9/3/2 of November

([60]) Letter dated 10.7.35 from Secretaries, Maintenance of Graves Trust Fund, Bloemfontein, O.F.S.

25th, 1933] "that the grave of C.V.S. Ender . . . is kept in good order," and "the surname should be Ender and not Ensor, as appears in the official list of graves which contains many errors." He adds that "the deceased died on the 10th June, 1900, and not on 9th June".

Kidd, W., died at Wynberg, C.P., on October 29th, 1901 [*Vet. Rec.* of 9.11.01 states Oct. 27th, 1901], and according to Mr. C. H. Wadlow, M.R.C.V.S. [see *Vet. Rec.*, 26.8.1933] was buried at Wetton Road Cemetery. Kidd and Armstrong are in a common grave—30 bodies. [Letter of 24.9.35 from Cape Soldiers' Graves Association.]

MacGregor, C. This must be the C. McGregor described as Sergeant and Veterinary (regimental number 25,868) on the monument to the Scottish Horse at Kensington, Johannesburg ([61]). His grave at Ventersdorp, where he died on January 22nd, 1902, is described by the Magistrate [his minute of December 5th, 1933] as being "in reasonable order".

Armstrong, R., died at Wynberg, C.P., on February 2nd, 1902, and was buried in a common grave at Wetton Road Cemetery, where Mr. C. H. Wadlow, M.R.C.V.S., located the site [see *Vet. Rec.*, 26.8.1933].

Sykes, G. Died on March 14th, 1902, and was buried at Charlestown, Natal. The N.C.O. in charge South African Police on November 20th, 1933, reported [his minute C.T. 43/3] that the grave was "in good order".

Davies, H. E. H., died at Deelfontein, C.P., on April 10th, 1902, and was buried in Deelfontein Military Cemetery. Thanks to the Magistrate, Richmond, C.P. [his letter of November 29th, 1933], the grave was put in "a good state of repair".

Parker, J. M. This C.V.S. attached to the 7th Dragoon Guards, died on August 15th, 1902, and is buried in the Old Cemetery, Worcester, C.P. The grave is in good condition. See fig. 18c.

The above names are shown in Table VI. See also *Vet. Rec.* of August 26th, 1933.

Imperial Yeomanry.

Vet.-Lieut. E. A. L. Fenner came out as V.O. 13th Batt. I.Y. "He was taken prisoner with us at Lindley, O.R.C., and died . . . at Vrede from dysentery and pneumonia and we buried him there". [Letter 6.6.35 from Mr. R. S. Garraway, M.R.C.V.S.] The number of the plot in Vrede

([61]) In a list of veterinarians who served in the Imperial Yemanry during the Second Anglo-Boer War, C. MacGregor's name is mentioned among the C.V.Ss. Another veterinarian who served in the Scottish Horse was Col. J. R. D. Beech who "in Sept., 1888, surrendered his veterinary for a combatant commission as 2nd Lieut. in the 21st Hussars . . . he returned from retirement to take part in the War in South Africa, where he commanded a regiment of Scottish Horse" (Smith, 1927, p. 184). Also R. S. Garraway, M.R.C.V.S., of Pretoria, served as a farrier sergeant in the 13th Batt. I.Y. and buried the V.O., E. A. L. Fenner, who died at Vrede.

Cemetery is 117 and according to the Magistrate [his letter of Nov. 14th, 1933], the only inscription on the iron cross is " 13 I.Y. Let. *[sic.]* E. Fenner, 23.6.00 ".

Vet.-Lieut. D. C. Barningham died at Pretoria on Dec. 7th, 1900, and his gravestone in the Old Cemetery, Pretoria, is in good state of preservation. See fig. 18b.

Another name of interest to the veterinary profession is that of *R. P. Williams,* who although not qualified, was " one of the first College (Class C, Royal Veterinary College, London) students to volunteer for

Fig. 18 c.
GRAVE OF J. PARKER, C.V.S., A.V.D., WORCESTER.

Fig. 18 d.
GRAVE OF R. P. WILLIAMS, VENTERSBURG.

the front, and shortly after enlisting in the Imperial Yeomanry he was . . . . made a Farrier-Sergeant. He was the first to be killed in action ". [*Vet. Rec.*, 1.9.1900.] His death took place near Ventersburg, O.F.S., on August 24th, 1900, and his grave is shown in fig. 18 d. The Magistrate, Ventersburg, kindly sent the photograph [November 22nd, 1933].

Fig. 19.

MURAL TABLET ERECTED IN 1904 BY R.C.V.S., LONDON, TO THE V.O's. WHO DIED IN THE SECOND ANGLO-BOER WAR (1899–1902).

The last veterinarian who died when on active service was *Capt. J. A. Worsley, S.A.V.C.*, whose death occurred on August 21st, 1914, at Prieska, C.P. His name, however, does not appear on the War Memorial Tablet, Royal College of Veterinary Surgeons. His grave [report of Magistrate, Prieska, dated November 24th, 1933] " is in good repair ". [See *Jl. S.A.V.M.A.* III (3) : 1932.]

(h) Conclusion.

One of the most concise statements concerning the Second Anglo-Boer War may be seen in " *A Short History of the 13th Hussars* " ([62]), where it is recorded :—

> Though this country [United Kingdom] at one time had 250,000 men in the field, the largest army which she had ever sent on active service, the tactics of the Boers made impossible anything resembling those fierce bloody battles which are a feature of the Napoleonic or Crimean Wars.
>
> There were few pitched battles, the Boer never fought unless the odds were in his favour, but for nearly three years he twisted and turned and wriggled and ran, always with a sting in his tail, always elusive, breaking every rule of war, without a base, without communications, but slowly teaching his enemies his own game until one day they could play it almost as well as himself . . . it was merely a quarrel between two independent and virile peoples over the possession of a tract of land as big as Germany and France, and victory as ever was on the side of the big battalions.
>
> The value of this campaign to the British Army was enormous. Whether we study the cavalry of the British Expeditionary Force or its retirement from Mons in 1914, or the tactics of the Colonial Mounted Infantry in Palestine three years later, wherever we look we see the fruits of the teaching of Louis Botha and Christian de Wet (p. 48).

Whatever other branches of the Army learned, there is no doubt that the campaign amply demonstrated the rottenness of the veterinary organisation. The evidence for this statement has been derived from Sir Frederick Smith's History. There is no doubt that the Second Anglo-Boer War was the most tragic period in the history of veterinary science.

Thanks, however, to the splendid spirit shown by many members of the profession, both Civil and Army, the position gradually improved, and later, with the sympathy of the War Office, the Army Veterinary Service reached the peak of its development during the Great War. This period of rejuvenation will be evident in the following Chapter.

Of chief interest in Chaper II has been the attempt to provide information regarding veterinarians, both regular and volunteer, who served during the campaign.

Only one diary was made available for this compilation, *viz.* that of Lieut. J. Moore, A.V.D., now General Sir John Moore. [See *Jl. Royal Army Veterinary Corps*, Aug., 1931, p. 182.]

(*To be continued.*)

([62]) Published by Gale & Polden in 1934 and kindly made available by the O.C., 13th Hussars.

NOTES AND NEWS.

On the 1st July last the Society for the Prevention of Cruelty to Animals, Johannesburg, opened a Clinic and Hospital at Booysens. Persons who cannot afford to pay for professional services may bring their animals to the Clinic for free treatment, and where necessary such medicines as may be prescribed and such nursing as may be requisite are also free.

Persons availing themselves of these privileges must sign a form stating that they cannot afford to pay a veterinarian. Owners offering to pay for treatment are advised to consult a veterinarian and are usually supplied with the name and address of the practitioner in their district. It is probable that members of the profession will be given the opportunity of sending dogs to the Hospital for treatment and taking the fees, the Society charging for boarding only.

The buildings, erected at a cost of nearly £5,500, comprise the office Block and the Kennel Attendant's Quarters, Surgery, and European Waiting Room. It is hoped that as more funds become available the equipment of the Surgery will be completed; at present this includes only the bare necessities. The Kennels can accommodate about 40 dogs and 40 cats and there is ample room for expansion.

At present the Clinic is open for one hour daily, while emergency cases receive attention at any time. So far the Clinic has been made use of only by Europeans, but it is hoped that when it becomes better known Natives also will bring their animals for attention.

Mrs. J. A. Robinson, B.V.Sc., is the veterinarian in attendance at the Clinic.

* * * *

Mr. B. M. Horwitz, B.V.Sc., who has hitherto been in private practice in Port Elizabeth, has been appointed Municipal Veterinary Surgeon of that city.

* * * *

The engagement is announced of Miss Vera Amos, M.R.C.V.S., to Mr. L. R. Morford, M.R.C.V.S.

* * * *

Mr. E. C. Nelson, M.R.C.V.S., has been appointed Manager and Veterinary Surgeon at the Municipal Abattoirs, Cape Town.

OBITUARY.
CHARLES ELIAS GRAY.

CHARLES ELIAS GRAY, born 10.4.64, entered the telegraph service in Edinburgh at the age of 16, but left seven years later to study at the Royal (Dick) Veterinary College. While in the telegraph service, of which his father was Controller, he was a member of the 24th Middlesex (Post Office) Rifles and when the call came for volunteers for the relief of General Gordon, members of the Edinburgh section, which was attached to the R.E., were selected for active service. Gray was one of these and his service included a year in Upper Egypt and the Sudan (1884–85). For this campaign he received the Egyptian medal (clasp Nile, 1884–85) and the Khedival Star. Shortly after his return he decided to take up veterinary studies and graduated in 1890 at the Royal (Dick) Veterinary College.

" Finding there was no chance of saving money as an assistant at home " he sailed for the U.S.A., where he had a " hard time finding employment ". Ultimately he became assistant to a British graduate, and later started for himself in Philadelphia. His capital proving insufficient, he returned " home for three weeks and then sailed for South Africa, travelling steerage for economy, London to Cape Town." He arrived at Cape Town at the end of 1895, hoping to secure a position as veterinarian, but as there was no opening he obtained employment (1.1.96) with the Chartered Co. as a telegraph operator, being stationed at Macloutsie. Two months later rinderpest (or Zambesi cattle disease, as it was then called) made its appearance and the authorities, having made no provision for veterinary services, appealed to the Colonial Veterinary Surgeon, Cape Colony (Hutcheon, D.) for assistance. Hutcheon naturally drew their attention to the fact that a veterinarian was in their employ and so Gray was summoned to Buluwayo in order to take over veterinary control. He was thus the first professional man to see a case of rinderpest, south of the Zambesi River. The policy adopted was slaughter with the payment of compensation. Along with this the permit system was initiated.

Just as the scourge was abating, the Matabele Rebellion (1896) broke out and as Gray " could do nothing more professionally " he "joined a troop of volunteers (H Troop, Bulawayo F.F.) raised by Selous . . . and served with it throughout the rebellion making" himself "generally useful . . .". For his services in the rebellion Gray received the Matabeleland medal (1896). " There being no veterinary work to do, I re-entered the telegraph service, and served in different parts of Rhodesia (ending up as postmaster, Victoria) until Koch established himself at the Victoria

Compound, Kimberley, when I was sent there to gain an insight into the new methods . . . and was subsequently employed as a Government Veterinary Surgeon in the Victoria district . . . where rinderpest was still smouldering." (1) He was then seconded for six months to the Cape Government on rinderpest duty in the Transkei, returning later to Salisbury, where he took charge of veterinary services (2). By compulsory inoculation with serum, rinderpest was eradicated by the end of the year.

The years 1900–1903 proved very trying for Mr. Gray, for with the entry of the Rhodesian Field Force (3) through Beira and Umtali and its stay in Southern Rhodesia, infectious diseases (*e.g.* lungsickness and glanders) became difficult to control. Later, from 1901 onwards, East Coast fever (or Rhodesian redwater, as it was then called) made its appearance. For his services to the Rhodesian F.F. at Marandellas and Beira, Gray received the Queen's medal with the clasp " Rhodesia ".

On Mr. Stewart Stockman's return to England, Mr. Gray became P.V.S. Transvaal, his service dating from April, 1905. With amalgamation of the four colonies in May, 1910, Gray was appointed P.V.S. of the Union of South Africa, his appointment as such dating from 1.1.1911. In 1917 he led a commission of veterinary surgeons, furnished by several Colonial Governments, to German East Africa to combat rinderpest, which as a result of military operations was spreading southwards towards the north-eastern border of Northern Rhodesia.

After a long term of faithful service, during which stock diseases were rampant owing chiefly to wars, Mr. Gray retired from the Union civil service on 9.5.21 and since then settled in Jersey, Channel Islands, where he died at St. Helier on the 11th June, 1937.

Throughout his service career Gray took a keen interest in volunteer-

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(1) At the beginning of 1897, while at Kimberley, Gray worked with G. W. Lee and Osborn O'Neil, all of whom returned to S. Rhodesia together, Lee to Salisbury and O'Neil to Umtali. Gray's permanent appointment as a Government Veterinary Surgeon dated from early in 1897. After this came Sinclair, J. M., Gorman, J., Edmonds, C. R., Bruce and Bevan. Then Gray transferred to Transvaal C.V.D.

(2) At this time there was a man, Ernest Sprague, farrier-sergeant-major in the B.S.A.P. He had studied at the " Dick " Veterinary College under Walley and had been to America, and for these reasons was considered by many as a veterinarian. After serving through the Boer War he settled in Pretoria as a veterinary surgeon and unfortunately contracted " blood poisoning " after operating on a horse which he declared was suffering from epizootic lmphangitis. This animal subsequently reacted to the mallein test! Gray and Garraway were at the funeral.

(3) Redwater had been recognised in Southern Rhodesia since the early 'nineties and when East Coast fever appeared in 1901, it was believed by the veterinary authorities to represent a virulent form of redwater; hence the name " Rhodesian Redwater ". East Coast fever first appeared in the Union in what is to-day the Barberton District in May, 1902, and was identified as such the following August. In 1903 Koch declared it to be "a distinctly different disorder."

ing and in professional matters. Soon after his transfer to Pretoria he joined the Northern Rifles as a trooper, but from May, 1906, to May, 1907, he acted as V.O. with the rank of Veterinary Captain. During the Great War he was sanitation specialist to the S.A.V.C. with the rank of

CHARLES ELIAS GRAY (1864–1937).

P.V.S. Transvaal, 1905; P.V.S. Union of South Africa, 1911–1921; President T.V.M.A., 1911–1913; Acting First President S.A.V.M.A., 1919–1920.

Lieut-Colonel. From 1911 to 1913, Gray was President of the T.V.M.A. and owing to Mr. Montgomery's absence in England in 1919–1920, he acted as the first President of the S.A.V.M.A.

H. H. C.

THE ASSOCIATION.

Secretary's Report for the Year ending 31st March, 1937.

Council.—Four meetings of Council were held during the year. These were well attended.

Membership.—The membership on 31st March, 1937, was 163—an increase of six. During the year the Association lost three members by death, *viz.*, Sir Arnold Theiler, Mr. J. D. Borthwick, and Mr. W. J. B. Green, while two resigned. Eleven new members were elected.

Activities.—The activities of the Association during the past year aimed primarily at promoting the welfare of the profession as a whole, but due attention was also paid to matters affecting not only sections of our members but in some cases individual members themselves.

Among the subjects of importance to private practitioners that were dealt with are the following : a uniform scale of charges for veterinarians; the witness fees paid by the Dept. of Justice for expert evidence; the supply of certain drugs direct to stock owners; and cruelty to animals with special reference to operative surgery. The investigations of complaints lodged by some members has revealed the fact that Act No. 16 of 1933, which was passed to protect registered veterinarians, is quite powerless to prevent certain forms of quackery, and the Association has obtained a promise from the Veterinary Board that certain amendments to the Act will be introduced in the near future.

Our members in the Government Service were vitally affected by the new scales of salary that were introduced during the year. In last year's report reference was made to a proposal by the Pro-technical Section to the Public Servants' Association for the adoption of a uniform scale for all technical officers. The application of the suggested scale to the Veterinary Division would have involved a lower commencing salary and smaller increments, with very doubtful benefits at the end of the scale.

Representatives of the Association on the Pro-technical Section strongly opposed this proposal and it was hoped that the matter had been dropped. Unfortunately, however, it was revived with renewed intensity during the present year, and it was only as a result of the determined opposition offered by our representatives that the Veterinary Division was excluded from the scheme.

It is sincerely trusted that this matter will now be allowed to rest, otherwise it cannot but have unpleasant consequences. Those of our members who belong to the Public Servants' Association have the right to expect that that Association will at least desist from attempting to harm them even if it cannot do them any good.

It is evident that in the endeavour to obtain the new improved scales for veterinarians, no assistance was obtained from the P.S.A., and credit for the attainment of these scales must be assigned mainly to the Director of Veterinary Services. The increments pertaining to the new scale are somewhat smaller; but on the other hand the commencing salary and the top notch are appreciably higher, and the affected members may well be satisfied with what they have obtained.

Municipalities are gradually realising the value of veterinary services in abattoirs and in dairy and transport supervision, and the Status Committee of our Association took a prominent part in the creation of two part-time municipal posts during the year. There are good prospects of two new full-time municipal posts being created in the course of the next year.

Courtesy Title.—The proposed adoption of the courtesy title was discussed very fully at the October, 1936, General Meeting, when it was decided to hold another referendum during the coming year, after members had had an opportunity of giving the question very careful consideration.

General Meetings.—At the Spring, 1936, General Meeting a resolution was taken abolishing the Autumn General Meeting. It was also decided to extend the Spring Meetings to three days and to have a major subject for discussion at every meeting. The meetings thus promise to be even more interesting and instructive in the future.

Finance.—Reference to the balance sheet will show that, despite the exceptionally heavy expenditure in connection with the Arnold Theiler Memorial Number of the Journal, the deficit of £22 15s. shown last year was converted into a surplus of £57 0s. 3d.

A disturbing feature of the balance sheet, however, is the large amount outstanding in arrear subscriptions. The collection of arrear subscriptions involves much extra clerical work and fairly heavy, unnecessary expenditure. I would therefore appeal to every member to assist by forwarding subscriptions when due.

Benevolent Fund.—This fund increased from £30 to £115 8s., mainly as the result of an appeal made by the President at the last General Meeting, which resulted in voluntary donations to the amount of £45 1s. 6d. being received. The balance of £115 8s. does not include £100 invested with the Reserve Fund.

The fund is not yet sufficiently strong to carry out benevolent work on a large scale, but during the past year financial assistance was granted to two very deserving cases—widows of colleagues—and this was greatly appreciated by the recipients.

Group Endowment Insurance.—The scheme has now been in working for a year and yielded a nett profit of £10 4s. 7d. It is trusted that all members who take out new endowment policies with the S.A. Mutual

will do so through this channel; which benefits both the Association and the individual member.

My thanks are due to all members for their loyal support during the year; to the President for his guidance in all matters, to the members of the various committees for their assistance; and to the Assistant Secretary, Mr. v. d. Wath, who has shared my duties conscientiously.

(Sgd.) *S. W. J. van Rensburg,*
HON. SEC.-TREAS., S.A.V.M.A.

Report of the Finance Committee for 1936–37.

Members of Committee : B. S. Parkin, A. D. Thomas, C. J. v. Heerden, G. van der Wath.

The books of the Association were duly audited on 31.3.37, and the following statements reflect the positions of the various funds on that date :—

A. INCOME AND EXPENDITURE.

Receipts.				*Expenditure.*			
Credit Balance on 1.4.36	£45	7	9	Printing and binding	£292	18	3
Subscriptions	339	16	3	Transferred to Benevolent Fund	103	19	6
Donations to Benevolent Fund	45	1	6	Dinner	45	14	7
Dinner	42	0	9	Clerical Assistance	30	0	0
Sale of Reprints	28	2	0	Petty Cash	22	0	0
Sale of Journals	24	9	3	Donation to Griffith Evans Fund	5	5	0
Advertising	25	12	0	Wreaths	2	2	0
Contributions for Publications by Dr. Curson	17	15	6	Auditing	2	2	0
				Refund on account Dr. Curson	0	12	0
				Natal Branch	5	0	0
				Bank Charges	4	19	1
				Credit Balance in Bank on 31.3.37	53	12	7
	£568	5	0		£568	5	0

B. BALANCE SHEET.

Subscriptions paid in advance	£7	8	6	Credit Balance in Bank on 31.3.37	£53	12	7
Printing Vol. 8, No. 1 of Journal	31	10	0	Arrear subscriptions	102	19	0
Contributions : Publications Dr. Curson	17	15	6	Petty cash on hand	0	19	2
Natal Branch Balance	14	17	0				
Clerical Assisctance	2	10	0				
Credit Balance	57	0	3				
	£157	10	9		£157	10	9

Last year this statement showed a debit balance of £22 15s. Notwithstanding the exceptionally heavy expenditure on Volume VII No. 4 of the Journal, the deficit has been converted into a surplus of £57 0s. 3d.

An unsatisfactory item in the statement is the large amount outstanding on account of arrear subscriptions, and an attempt will be made to reduce this sum appreciably during the current year.

C. Reserve Fund.

The Reserve Fund amounts to £1,182 8s., which sum was invested in Union Loan Certificates in 1934. Of this amount £100 plus interest, which was voted to the Benevolent Fund in 1933, must be credited to the latter fund.

D. Benevolent Fund.

Credit Balance on 1.4.36	£30	0	0	Donations to Widow A	£10	0	0
Credited from Cash Account	103	19	6	,, ,, ,, B	10	0	0
Interest	1	8	9	Credit Balance, 31.3.37	115	8	0
	£135	8	0		£135	8	0

E. Group Endowment Insurance Fund.

Receipts.				*Expenditure.*			
Credit Balance on 1.4.36	£1	13	0	Premiums paid to S.A. Mutual	£371	8	8
Premiums collected	386	18	11	Bank Charges	2	3	0
Erroneously credited	0	9	0	Cr. Balance in Bank on 31.3.37	15	9	3
	£389	0	11		£389	0	11

The nett profit on this account is £10 4s. 7d., *i.e.* £15 9s. 3d. less premiums paid in advance (£4 15s. 8d.); and the sum of 9s. which was erroneously deposited to the credit of this account. In accordance with a resolution taken by Council on 18th October, 1936, the nett profit is to be transferred to the Benevolent Fund.

(Sgd.) *S. W. J. van Rensburg,*
Hon. Sec.-Treas., S.A.V.M.A.

Editorial Report for 1936,

Members of Editorial Committee: P. J. du Toit, A. D. Thomas, J. H. Mason, M. Sterne, C. Jackson.

STATEMENT.

To Advertisements	£29	0	0	By Printing Charges No. 1	£29	8	0
,, Subscriptions	18	3	11	,, ,, ,, No. 2	24	10	0
,, Sale Journals	4	18	0	,, ,, ,, No. 3	56	11	6
,, Sale Reprints	0	2	6	,, ,, ,, No. 4	116	5	0
				,, Reprints	24	12	6
				,, Envelopes, 1,500	4	10	0
				Total Expenditure	£256	17	0
				Revenue from Journal	52	4	5
	£52	4	5	Actual charges on Association funds	£204	12	7

CIRCULATION.

Copies.		*Sundry Copies.*	
Members	600	Current and Back Nos.	30
Cadet Members	111	Advertisers' Vouchers	28
Exchange	232	Specimen and Complementary	26
Subscribers	168	Book Review Vouchers	7
Free Mailing List	20		
	1,131		91

COMPARISON OF ACTIVITIES DURING LAST 6 YEARS.

Year.	*No. pages per Volume.*	*Total Cost.*	*Total Revenue.*
1931	170	£157	£41
1932	214	209	62
1933	244	217	66
1934	278	229	72
1935	314	304	85
1936	252	256	52

The chief feature of the editorial activities for the year 1936 was the publication of the Arnold Theiler Memorial Number (Vol. VII, No. 4), the most ambitious issue (pp. 119) which has so far been attempted and which received favourable comment from both professional and lay sources. Due acknowledgement of the excellent spirit in which not only members but other well-wishers co-operated in this project has been made in the Journal. In the publication of this Number, a joint meeting of the Finance and Editorial Committees gave the Editor virtually a free hand as regards expenditure.

Notwithstanding the relatively heavy cost entailed (£116 5s.), the expenditure for the year was kept within moderate limits, chiefly by

economy in the preceeding numbers of the volume. Largely because of the publicity which the Press has been kind enough to accord the Memorial Number, the sales of this number promise to exceed those of previous issues; but the revenue from this source does not fall under the present financial year.

During the year under review, Dr. A. D. Thomas relinquished the Editorship of the Journal, after five years of unremitting labour, during which period he effected outstanding improvements in its standard and make-up. Dr. Thomas further achieved considerable success in the most difficult task which the Editor has to face, *viz.* that of persuading members other than those who are stationed at Onderstepoort to give the Journal the benefit of their knowledge, observations and experience. It is felt by the Editorial Committee—and no one will take this remark amiss—that a Journal which is over-weighted by contributions from Onderstepoort is not truly reflecting the entire activities of the Association and the ideals of the Profession as a whole, and it will continue to be our policy to encourage G.V.Os., Private Practitioners, and Municipal Veterinarians to see to it that their work takes its rightful place in the contents of the Journal.

For the Editorial Committee,

(Sgd.) *Cecil Jackson,*

EDITOR, *Jl. S.A.V.M.A.*

Report of the Hon. Librarian for the Year ending 31st March, 1937.

In accordance with the recommendations made in my last report and confirmed by Council on 18.6.36, the necessary arrangements for the housing of the Library at the Students' Hostel at Onderstepoort have made good progress. It is hoped to transfer the books during July next. In this matter Dr. du Toit, Director of Veterinary Services, has given the Association every assistance, and the shelving is being provided without cost to the Association.

The Hon. Librarian will continue to supervise the Library until such time as the Cadet Members have become familiar with its administration, and Members may continue to borrow literature as before.

Owing to the disorganisation entailed by the transfer of the Library, it is convenient to postpone giving details of new accessions until a later report.

At the Empire Exhibition, the Hon. Librarian co-operated with the Division of Veterinary Services in arranging a display of "Some of the most Important Contributions from South Africa to the Literature of Veterinary Research." In this exhibit the publications of the S.A.V.M.A. occupied a prominent place. At the close of the Exhibition the Director of Veterinary Services decided that the cabinet and its contents should be housed permanently in the Divisional Library at Onderstepoort.

(Sgd.) *Cecil Jackson,*

HON. LIBRARIAN.

Jl. S.A.V.M.A.
VIII (4) :
1937.

Epidemiology.

By E. M. ROBINSON, Dr. Med. Vet., D.V.Sc., F.R.C.V.S.,
Onderstepoort.

In recent years a great deal of attention has been paid to the study of the epidemiology of diseases. An enormous amount of information has been collected and it has been submitted to the statisticians for criticism, with the result that some of the most important points in relation to the study of epidemics have now been sorted out from the tangle of miscellaneous observations. A recently published book *Epidemics and Crowd Diseases* by Greenwood, of the London School of Tropical Medicine and Hygiene, is a fascinating study of the problems facing the epidemiologist. Greenwood is a statistician and makes use of mathematical formulæ in his book, but one needs little knowledge of mathematics to follow the author and the style in which the book is written is very fine. In Dible's (1932) *Recent Advances in Bacteriology,* a chapter is devoted to a summary of the work done in recent years on epidemiology, written in a simple and easily understandable style. Finally, last year the Medical Research Council in England has issued a publication on *Experimental Epidemiology* by Greenwood, Topley and others, giving a full summary of their work over the last fifteen years.

Although a great deal of information has been obtained in the past from the study of epidemics, particularly in man, many problems have arisen which have proved very difficult to solve by a study of naturally-occurring epidemics. It was these problems which led British, German, and American investigators to seek for an experimental method of studying epidemics. It is not pretended that it is possible to reproduce experimentally conditions exactly such as are met with in nature, but a study of experimental epidemics has thrown much light on some of the obscure problems arising out of the study of naturally-occurring epidemics. The method has been to work with large colonies of mice, although other laboratory animals have been used in some cases. In the case of mice, one can work with large numbers of animals, introducing virus or bacterial diseases amongst them and studying the results. The various epidemiological factors can be varied at will in order to study their effects.

The problem at issue is to study the conditions underlying epidemics. The spread of a disease and the spread of infection are not quite the same thing; for instance in a disease like cerebrospinal meningitis (spotted fever) in man, one may have a high percentage of people harbouring the causal organism (carriers) but very few cases of the disease. It is very

probable that the distribution of an infective organism in a population is far greater than the prevalence of the disease. In epidemic spread of disease two main factors play a part : the virulence of the infection and the resistance of the host. Topley states that we may expect four different types of disease to occur when an infection starts in a population : (a) fatal infections, (b) infections clinically recognizable as cases of the disease, (c) atypical infections in which there is some departure from the normal, and (d) latent infections without symptoms.

Experimental epidemiology tries to control these variable conditions by experiments in which the various factors can be ascertained. A warning must be issued against arguing from mouse experiments to the much more involved human or animal epidemics, but nevertheless certain similarities between them can be observed. Most of the experiments on mice have been done with salmonella or pasteurella infections and with a virus infection called ectromelia. Some investigators have even gone to the length of breeding pure lines of mice by close inbreeding over a period of some years, in order to eliminate variable characters in the mice themselves; but this is probably unnecessary, as human or animal populations are usually unselected. Topley in his experiments confined himself to unselected mouse stocks.

Description of an artificially produced epidemic.

When an epidemic was started in a herd of mice by adding a few infected individuals (*S. enteritidis*) a lag period followed by a heavy mortality occurred, but the curve of the mortality then flattened out and in about three months the mortality ceased, fewer and fewer cases occurring. Some of these epidemics developed more slowly than others. The milder epidemics developed more slowly from the beginning.

In the study of epidemics, three phases can be recognized : (a) the pre-epidemic, (b) the epidemic and (c) the post-epidemic.

The Pre-epidemic Phase.—This may be considered as being the time from the introduction of the infection to the outbreak of the main epidemic. Isolated and scattered deaths occur amongst the susceptible animals, but a long latent period occurs before the main outbreak develops and this period appears to be of constant occurrence. It may be one or two months in some cases. If one starts an epidemic with three infected mice and adds three susceptible individuals every day, or in some cases irregular batches of susceptible mice, the main epidemic does not start until there are about a hundred mice in the colony. The same thing occurred in a pasteurella outbreak which broke out accidentally in a herd of one hundred and twenty mice which had been collected for another experiment. Defoe in his history of the " Plague " mentions a similar occurrence in an outbreak in man.

The Epidemic Phase.—The addition of susceptible animals to a

population already infected, but in which the epidemic has died down and a stage of equilibrium has been reached, always results in a fresh outbreak of the disease, involving both newcomers and old residents which had survived.

An epidemic can therefore be kept going indefinitely. If increments to a population are made regularly, the mortality does not maintain a regular level, but waxes and wanes. The periods of heavy mortality are preceded by periods of quiescence, when the population gradually increases until the concentration is considerable and conditions become suitable for a fresh flare-up. These epidemic waves are best seen when the additions are made slowly, as the periodicity is less marked when a high concentration is maintained in the cages. As long a period as ten months may elapse between flare-ups. For the occurrence of a severe epidemic a large population is necessary. In small groups, after a few cases have occurred, the infection dies down but if the groups are reconstituted into a big group the mortality increases again. The possible factors which govern the onset and course of an epidemic are the dosage of the infection, the virulence of the organism, and the resistence of the host. Resistance refers to the general resistance of the herd, not to that of the individual. This host resistance probably plays no great part. The probable explanation of the recrudescences in old herd survivors and of the heavy general mortality is an increase in the dosage of infection which has previously been falling. According to Webster, there is very little change in the virulence of an organism during an epidemic, though in endemic infections a low virulence may be noted. However, Topley considers that variations in virulence during an epidemic do occur and that the virulence of an organism is increased by passage. The mortality in epidemics varies with the virulence of the strain. With one particular strain the course of the epidemics is very similar. When one adds large batches of mice to an infected herd at irregular intervals one may note a sudden rise in the death rate, but on the other hand nothing at all may happen. The suggestion is that the mortality depends on the disease balance between the hosts and the organism at the time of the addition.

The Post-epidemic State.—A simple epidemic tends to be self-limiting and to come to an end. The survivors possess a greater resistance and the question arises as to whether they had this from the first or developed it during the epidemic. One has to assume that, in some individuals, a state of equilibrium between the organism and the host occurs, because when small infected groups are amalgamated into one big one, although the mortality increases, it does not do so to any great extent such as would occur if fresh mice were added to the infected ones. Sixty to 100 per cent. of mice surviving epidemics of *S. aertrycke* infection can be shown to harbour the organisms in the spleen, although outwardly the animals appear to be healthy. Survival is therefore due to a sub-

clinical infection and a sort of symbiosis occurs; but later the infection is finally got rid of. Tuberculosis and syphilis can be quoted as examples of similar symbiosis. Ultimately this equilibrium ends in favour of the patient, but this probably only applies to a few infections. Some think that natural selection eliminates those with a weak resistance. Topley does not consider natural selection important, but that survival is due to active immunization during the epidemic. In favour of this idea is the finding of organisms in the tissues of survivors and the reactions of the latter to serological tests. The sera of 75 per cent. of the survivors of an *S. aertrycke* epidemic agglutinate the organism. A wide dissemination of the infecting organism occurs, so the increased average resistance of the survivors points to active immunization, and it is apparently more important than selection.

Since acquired immunity has a tendency to bring an epidemic to an end, what occurs if an attempt is made at an earlier period to induce immunity, as is done in human or animal medicine ?

The result of infecting populations of immunized and unimmunized or mixed mice is that the mortality is much less in the immunized or mixed lots. Topley carried out an experiment to see what influence immunization at different stages of an epidemic would have on the course of the epidemic. The highest survival rate was seen in mice which had received two injections of vaccine prior to the experiment. Mice vaccinated even twice after exposure to infection did not show any greater immunity than did the controls. Topley has made the observation that when one inoculates mice with paratyphoid vaccines, the best immunity is obtained after four injections; but if one goes on to eight inoculations, the immunity, instead of increasing, decreases; and if twelve are given it decreases still further.

Greenwood and his co-workers (1936) in their summary of their work on *Experimental Epidemiology* state, with regard to the general character of epidemic processes as revealed by herds of mice in close and continuous contact and subject to intermittent or continuous immigration of susceptibles, that :—

(1) The disease will never die out normally. It is possible for a disease to become extinct if very small numbers of animals are left, of which none is a carrier or sick. This would be purely an accident under natural conditions and probably rarely occur.

(2) The form of the mortality curve depends on the rate of immigration. With a low immigration rate there will be well-separated waves and quiet intervals. With high rates of immigration one gets minor fluctuations or an almost steady death rate.

(3) The condition of equilibrium is unstable and, when it is disturbed, violent fluctuations will occur before some kind of an equilibrium is re-established.

(4) In the epidemics studied, initiated by virulent and infective strains and in which the disease takes its characteristic and fatal form, the rate of mortality in the early days of herd life is very high. Fifty per cent. of the entrants are dead in 25 days and 75 to 80 per cent. after 50 days. The early mortality is always of this order, a decrease sets in by about the 25th day, and by the 60th day a low mortality level has again been reached. The mortality rate does not fall to that of the normal cage mortality of healthy mice. In ectromelia, a virus disease of mice, the return to a low level nearer to that of healthy mice occurs.

Mechanisms determining these events.—These have been discussed to a certain extent in an earlier part of this paper. The resistance of surviving mice is a fluctuating factor and does not remain at a steady level. It would seem that some mice have a very effective mechanism of some kind, hindering access of bacteria or viruses to the tissues, and remain uninfected in spite of constant exposure to infection. Their ability to escape infection is never absolute. It is probable that in an epidemic the fluctuations which are observed in mortality are not due to any fixed cycle of bacterial or virus development, to seasonal factors, or to any single determining cause. The characters of the bacterial strain seem to influence the level of mortality and the proportion of fatal infections and the degree to which infection occurs. Virulence and infectivity vary independently, but a bacterium may possess both. It has been shown that a strain of low epidemicity may give rise to a variant of high epidemicity during the course of a long continued epidemic. The difference in virulence or infectivity of different bacterial strains of the same species is therefore of first importance in determining the general characters of an epidemic. The possible variations in these characters are a potential cause of changes in the prevailing type of an epidemic.

It has been shown that natural immunization is more effective against virus diseases than against bacterial diseases and that the same applies to artificial immunization. Immunity is never complete and even immunized mice will ultimately die of the disease. The immunization of all entrants to an infected herd will bring the epidemic to a close. Artificial immunization does not have much effect on the *infection* rate and many of the immunized mice in both bacterial and virus diseases are infective for healthy mice. Even better methods of immunization would probably not eliminate infection from herds and so render immigration of non-immunes safe. It has to be remembered, however, that in the mouse infections the exposure was severe and may not be comparable with what occurs in human epidemics.

Interference with the course of an epidemic by modifying the conditions of contact.—Dispersal of a herd, in order to be effective in stopping an epidemic, must be carried out in the early stages. If some means could

be found to lessen the intimacy of contact of animals in an infected herd, it would almost certainly be found that a decrease in the infection rate would occur.

In giving examples of epidemic fluctuations in human disease, one may quote the case of measles. In this disease the periodicity of the epidemic is probably due to periodic changes in the constitution of the population exposed to risk, leading after each epidemic wave to a gradual re-accumulation of susceptibles. This corresponds to the cases in the mouse experiments where there is a wide space between the waves of heavy incidence when there is a slow rate of immigration, or to the effect produced by adding susceptibles to a population surviving from an epidemic. In nature the accumulation of susceptibles is largely by births. In boarding schools or schools in general, it is probably the immunization of the non-immunes term by term which determines the course of events. It is this—the ever-varying immunological state of a herd—which determines the intervals of the epidemic waves.

Sublethal or latent infection is the essential factor involved in the immunization of human or animal populations. Regarding the relative value of inborn and acquired immunity, well over 1,000 years exposure to measles, which is most dangerous before puberty and so should be highly selective, has not lowered its incidence at all. A wave of disease obviously immunizes a population, but it is probable that many who never show symptoms become immunized. In mouse experiments, no seasonal factor could be found influencing mortality but of course in nature one has many factors such as heat and cold, moisture and dryness, and weather conditions affecting the individual's health. Opening and closing of schools would be independent of such factors as weather conditions.

The character of epidemic strains is of great importance. An example is recorded where 11 cases of cerebro-spinal fever with one death occurred during 15 months followed by a carrier rate in the local population of over 50 per cent. for seventeen months with no further cases.

Another problem is that one bacterium may be associated with another in an epidemic, or one virus with another, or a virus with a bacterium. In the character of an epidemic this may play a part as important as the variation of a single bacterium. The effect of dispersal of an infected herd is comparable with the closing of a school infected with the disease, and the result would probably depend on the time in the epidemic when the school was closed. If early, when there were only a few cases, the advantage to the scholars would be great. If late, the advantage would be slight, but not neglible. Only when one knows the relation of cases to carriers can one judge what the result will be on dispersal.

As has been mentioned, there is little hope of eradicating diseases by immunization. One may maintain the herd in a state in which no

obvious cases occur, but a relaxation of the immunization will result in a re-accumulation of susceptibles and a recrudescence of the disease. It would be more hopeful if one could immunize so that infection could be prevented, though the danger of reimportation if quarantine measures were relaxed would be a very real one. Active immunization is the only method at present which will exert any influence on the prevalence of an infectious disease. It is probable that the disappearance of an epidemic is due to several factors combined, not to any one single factor.

One of the most important points which emerges from these mouse experiments is that the influx of susceptible immigrants will confer a herd immortality on a disease. There may be long periods of quiescence, but only in very small herds will the disease ever disappear. It is impossible to end an epidemic simply by guarding against the re-introduction of further infection.

When a disease occurs in limited foci in a country and breeds true, or is actually absent, then, if one can close the frontiers to reimportation of infected material and stamp out the internal foci, it is theroretically and practically certain that the disease will disappear. If the disease is widespread it can no longer be controlled in this way. In the case of isolated outbreaks of swine fever or foot-and-mouth disease, slaughtering-out is a sound policy. If the disease spreads, slaughter may be too expensive and so the policy has to be modified. Numerous experiments would tend to show that if the immigration of infected animals from outside is prohibited, but contact of the survivors with healthy animals is permitted, the disease will not die out, but will recur from time to time in a most perplexing way.

Greenwood (1935) points out that officials dealing with infectious diseases cannot bring themselves to believe that, when a disease has officially been declared eradicated, it can start up again without reimportation; and reimportation is generally blamed for these recurrences. It has been suggested that the possibility cannot be excluded that infective material from a carrier, after passage through one or two susceptible animals, may be in the position to cause the disease in animals which had previously had it and acquired some degree of immunity. This may explain recrudescence of foot-and-mouth disease without reimportation of infected material and one may encounter what appears to be a spontaneous outbreak.

In considering human and animal epidemics one has to realise the large number of factors which may influence the course of an infectious disease. The virulence and infectivity of the organism, its resistance to external conditions, the mode of infection, the type and character of the intermediate host (if there is one), the existence of carriers, latent infections, labile infections, climatic factors, predisposing factors, and the influence of immunization methods are only some of the factors which

may exert an influence and must therefore be taken into account. There is very little doubt that diseases have existed which have completely disappeared, but the historical records of antiquity are not sufficiently accurate to enable one to describe any of them, apart from the famous sweating sickness of the middle ages, which appeared in the fifteenth century and, after causing several severe epidemics, disappeared completely. From the very good accounts which exist it is fairly certain that this disease was not one of the better known ones, such as plague or typhus.

The occurrence of new diseases not previously recognized has been noted in recent years. Two of these may be mentioned—post-vaccinal encephalitis and post-influenzal encephalitis. Zinsser (1935) mentions the increased virulence of measles in recent years and the comparatively mild course of scarlet fever. It would appear that vaccination against a disease may alter its virulence, and he quotes diphtheria in America, where, since the general inoculation campaigns in schools, cases of a virulent type have occurred in susceptible children. When measles starts in a population which has no previous experience of it, it takes a virulent form and it is known that it caused a very heavy mortality in the South Sea Islands when it first appeared there. The acclimatization of population to diseases is a commonplace occurrence and one may instance the resistance of the white races as a whole to diseases such as tuberculosis and syphilis. The black races show a much lower resistance to tuberculosis than the white. The records of syphilis show that when it first broke out on a large scale in the fifteenth century, it had a far greater virulence than it has now, which is usually explained by a saturation of the population with it.

Diseases such as plague no longer appear to be capable of causing large-scale epidemics. Why this should be the case it is difficult to say, as it has been introduced into populations where fleas were present in large numbers. Zinsser (1935) suggests that the increased domesticity of the rat and plentiful food supplies for it may tend to prevent it wandering, so the disease remains localized.

A further possible means of spreading diseases has come into existence in recent years in the motor-car and the aeroplane, and it has been feared that diseases like yellow fever, endemic in certain parts of Africa, may be spread by aeroplanes carrying the insect vectors.

An attempt has been made in this paper to give a review of some of the more recent observations which have been made in the study of epidemiological problems. The introduction of the experimental method is an important step forward and we may expect many important observations to be made in the future which will throw light on some of the features of epidemic diseases which are at present obscure.

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Note on the Occurrence of *Hatertia gallinarum* in the Korhaan.

By H. H. MÖNNIG, B.A., Dr. Phil., B.V.Sc., Onderstepoort.

It is of interest to record that the nematode parasite, *Hatertia gallinarum* (Theiler, 1919), known hitherto only in the domestic fowl, has now been found in the "vaal korhaan", *Heterotetrax vigorsi.*

This worm and its life-cycle was described in 1919 by Theiler, who found that the termite, *Macrohodothermes mossambicus transvaalensis* was the intermediate host. The worm was first discovered in fowls around Pretoria, later at Kroonstad, then in other parts of the Union and as far north as Elizabethville, Belgian Congo.

The distribution of this parasite, restricted as it is to (southern) Africa, made one suspect that it is one of those which our domestic animals have acquired from wild animals in this country. For years every opportunity was seized to examine worms of bustards, which seemed to be the most probable hosts, but unfortunately they are royal game. Several species of *Hatertia* have been described from these birds, and recently *H. gallinarum* was collected at Kendrew, C.P., and sent to us for identification by the Government Veterinary Officer at Middelburg, C.P.

It is very probable that other species of bustard can also carry this worm. Sclater states that the "vaal korhaan" has not been found north of the Limpopo and it is therefore likely that there must be another host in the region of Elizabethville.

THE ARMY VETERINARY SERVICE IN SOUTH AFRICA 1881—1914 (Continued).

By H. H. CURSON.

CHAPTER III.

The A.V.S. in South Africa, 1902—1914.

(a) Introduction. (b) Demobilisation (Table VII). (c) Army of Occupation (Tables VIII and IX). (d) Stables. (e) Annual Reports of A.V.S. (f) Statistics relating to Army Animals in South Africa (Tables X and XI). (g) Special Features in Reports. (h) Special Orders and Reports. (i) Veterinary Hospitals. (j) Animal Management. (k) Miscellaneous. (l) Conclusion.

(a) Introduction.

An excellent résumé of the development of the A.V.S. between the Second Anglo-Boer (1899–1902) and Great (1914–18) Wars is given by Blenkinsop and Rainey (1925), who, in describing the period as one of preparation, state (p. 1), "The veterinary experiences of the South African War were valuable chiefly for the undeniable evidence they gave of the need for a properly organised Army Veterinary Service".

By 1902 it must have been obvious to everyone that there was something amiss with the veterinary department of the Army (63). Apart from the tremendous wastage of horse-flesh, the following facts, among others, must have convinced the authorities that the organisation of the A.V.S, a professional branch, should be entrusted to its own heads :—

(a) Lack of officers on outbreak of hostilities;

(b) Supply of field veterinary hospitals *from India;*

(c) The appointment in 1899 of the Director of Transport and Supply as "the head of the Remount and Veterinary Services" the latter being placed *under* the former (Smith 1927, p. 203) (64);

(d) "The lack of authority in their own hospitals and over their own subordinates" (*Idem*);

(e) "The appointment of a combatant officer as Inspector of Veterinary Hospitals in South Africa" (*Idem*);

(63) It is significant that only one veterinarian, Col. F. Duck, Director-General, A.V.S., was called to give evidence before the Royal Commission on the War in South Africa! He was not in South Africa during 1899–1902!

(64) The Report written by the Remount Dept. after the War was never published. Deficiencies, amounting to "30,000 animals in a year," nullify the statistics therein. See, however, résumé of official Remount Report 1901–1902 in *Transvaal Agr. Jl.* Vol. I, Jan., 1903.

(f) Veterinary officers were *ineligible* to purchase remounts ([65]); and (g) The scattering of disease after the war, even to Great Britain.

Accordingly, " in November, 1902, on the suggestion of Major-General H. Thomson, the Director-General . . . . a committee was assembled, under the Earl of Hardwicke . . . to enquire into the conditions affecting the officers of the A.V.D. and to suggest remedies ". (Blenkinsop and Rainey, 1925, p. 1) ([66]). The findings of the Committee were sanctioned by the Royal Warrant of October 5th, 1903, and with similar sympathetic treatment, the A.V.S. by 1914, had become the finest veterinary organisation in the world, and was so efficient that it coped successfully with all demands made during the Great War.

The actual reforms introduced by the Hardwicke Committee included (a) the conferment of non-compound rank on all serving officers, (b) increased pay and pensions for most ranks, (c) accelerated promotion, and (d) the creation of an Army Veterinary Corps from among the N.C.O's and men already in the Service.

There were thus an Army Veterinary Department, comprising the officers, and an Army Veterinary Corps for the rank and file, and it was not until 1906 that amalgamation took place, the designation A.V.D. becoming obsolete.

Other reforms included the creation of hospitals, the training of personnel, the accumulation of mobilisation and reserve stores, and the compilation of records and instructions. Among the last mentioned were Standing Orders (1906), a handbook on *Animal Management* (1907), and the *Veterinary Manual* (*War*) (1914). The names of the Director-Generals during this period of evolution are shown under (e) Annual Reports of A.V.S.; but the brunt of the work fell on Major (now Major-General) E. R. C. Butler, now of Kokstad and a member of the South African Veterinary Medical Association.

" In 1907 the first attempt was made to create a separate Veterinary Directorate under the Q.M.G. instead of the Director of Transport and Remounts . . .", but it was not until the appointment of a new Q.M.G. in 1913 that this was approved by the Army Council (Blenkinsop and Rainey) ([67]).

([65]) The officers of the Remount Dept. draw up mobilisation schemes and state what horses are necessary, but unlike the veterinary officers have no particular training.

([66]) The action of the Council R.C.V.S., especially the initiative of Mr. J. F. Simpson, in drawing the attention of the War Office to the grievances of the A.V.D., must ever remain as an example of what can be done by systematic perseverance.

([67]) See article " Retrospect and Prospect " by Sir John Moore, *Jl. R.A.V.C.*, Nov., 1932.

(b) Demobilisation.

Before describing conditions concerning the Army of Occupation, it is necessary to refer to the process of demobilisation, an unusually strenuous period for the A.V.S., especially when not properly organised.

Smith (1912–14, p. 297) mentions that "on the day peace was declared there existed 131,700 army horses, 76,600 mules, 12,800 donkeys and 74,200 oxen" ([68]). Of these "there were 28,700 sick horses and mules . . . and . . . certainly not less than 10,000" oxen, among which communicable diseases were rampant. As the expense of maintaining such a large number of animals was tremendous, the War Office naturally wished to dispose of the surplus as speedily as possible.

In order to assist the Boers to resume their farming operations, the Imperial Government sanctioned the expenditure of £3,000,000 and Repatriation Departments were created in the Orange River Colony and Transvaal. In this way the military authorities disposed of 23,700 horses, 51,800 mules, 5,700 donkeys, and 60,900 oxen; and as a result disease was scattered more thoroughly throughout the Subcontinent. To cope with the situation veterinary surgeons were appointed to the Repatriation Departments, but as an idea of the hopelessness of the position Smith (p. 282) mentions that there were 25,000 doses of mallein for 131,000 horses and mules! The chaos resulting from the lack of organisation generally is described under the Repatriation Departments and their Veterinary Organisation in the *Veterinary Record* of September 27th, 1930.

Horses were allowed to return to the United Kingdom too, in spite of veterinary opinion, and as a consequence not only mange but also epizootic lymphangitis were introduced into Great Britain.

"Seventeen officers were returned to India before the end of 1902" as well as "the whole of the Indian Hospitals, together with their personnel". (Smith, p. 299.) The A.V.S. officers who remained in South Africa are shown in Tables III and V.

Of greatest interest to us in South Africa was the fact that many C.V.Ss. remained in the country as members of either the Repatriation Departments, Civil Veterinary Divisions, or S.A. Constabulary.

A list is appended (see Table VII).

(c) Army of Occupation.

The distribution of the cavalry is shown in Table VIII but other branches of the Army employing horses and mules ([73]) have also to be considered from a veterinary aspect. In addition to the centres shown in Table VIII the following were also of military importance: *Cape Colony* —Cape Town, Wynberg, and Bowkers Park; *Natal*—Pietermaritzburg

([68]) There is a great discrepancy in the various estimates. See Smith, p. 229 and 282.
([73]) e.g. chiefly Artillery, Mounted Infantry, Remounts, Royal Engineers and Army Service Corps.

TABLE VII (69).

NAMES OF CIVIL VETERINARY SURGEONS ATTACHED A.V.D. WHO DID *NOT* RETURN TO UNITED KINGDOM SOON AFTER SECOND ANGLO-BOER WAR.

Name.	Qualified.	South African Constabulary (70).	Repatriation Department (71).	Department of Agriculture (72).	Other positions.	Further remarks.
Armstrong, R.	Glasgow 1895	—	—	—	—	† Wynberg 13.2.02 *Vet. Rec.* 26.8.33.
Aitchison, D. A. D.	London 1900	—	—	O. 1903	—	—
Brand, J.	N. Edinb. 1890	1902	—	—	—	Came to S. Africa from New Zealand.
Bell, J. H.	London 1889	1901–04	—	T. 1904–08	—	† Dublin 24.6.13 *Jl.* III (3) '32.
Buck, J.	N. Edinb. 1895	—	—	—	Pp. Kimberley	—
Bowhill, T.	N. Edinb. 1886	—	—	C. 1903–06	Col. Brit. Inst. Grahamstown, 1902	† Durban 1.9.25 *Vet. Rec.* 1.5.26.
Cannon, G. T.	London 1899	—	—	T. 1903–04	—	—
Conacher, P.	Edinburgh 1889	—	—	T. 1902–21 x	—	—
Christy, J. M.	Edinburgh 1889	1901–03	—	T. 1903–16 x	—	† Pretoria 29.6.17. *Jl.* III (3) '32.
Crawford, J.	Glasgow 1898	—	1902–03	—	—	—
Chalmers, J.	London 1900	—	1902–03	T. 1903–31 x	—	*Jl.* May 1931.
Carless, F. J.	London 1890	—	—	—	Pp. Mooi River	Was Pres. of the Mooi River Rifle Assn. which body formed nucleus of Murray's Horse, 1899.
Clark, C. C.	N. Edinb. 1881	—	1902–03	—	—	—
Cordy, C. H.	London 1890	—	—	N. 1902–07	—	In Natal C.V.D. from 1897.
Davies, H. E. H.	Glasgow 1895	—	—	—	—	†Deelfontein 10.4.02. *Vet. Rec.* 26.8.33.
Dunphy, J. P.	N. Edinb. 1900	—	—	T. 1903–08	—	—
Dale, T.	N. Edinb. 1889	—	1902–03	T. 1903–17 x	—	† Durban 15.7.17. *Jl.* III (3) '32.
Dunning, F. J.	N. Edinb. 1901	—	1903–05	T. 1905–34 x	—	—
Ensor, E. T. C.	London 1898	—	—	—	—	† Kroonstad 9.6.00. *Vet. Rec.* 26.8.33.
Edgar, J. I.	Edinburgh 1895	—	—	T. 1902–28 x	—	—
Evans, W. G.	London 1901	—	1902–03	T. 1903–14 x	—	—
Forrest, J.	Edinburgh 1900	—	—	—	Pp. Capetown	—
Fairclough, J.	N. Edinb. 1899	—	—	—	Pp. Johannesburg	—
Fern, E.	N. Edinb. 1891	—	—	—	Pp. Port Elizabeth	In private practice at Port Elizabeth since 1895.
Fyrth, W.	London 1891	—	—	N. 1905–06	—	—
Gavin, F. C.	London 1889	—	—	—	Mun. Johannesburg	*Jl.* Nov. 1929.
Grist, A. G.	London 1892	—	—	O. 1903–28 x	—	—
Goundry, C.	Edinburgh 1897	—	—	C. 1902–12 x	—	† Malmesbury *Jl.* III (3) '32.
Goule, A.	London 1871	—	—	N. 1908–18 x	—	† At Sea 12.9.18. *Jl.* III (3) '32.
Hodder, A.	Edinburgh 1898	1901–03	—	N. 1905–06	—	—
Hirst, W. M.	London 1898	—	—	—	—	†Boshof 5.5.00. *Vet. Rec.* 26.8.
Hill, W.	Glasgow 1887	—	1902–05	O. 1905–06	—	† 14.2.06 Senekal. *Vet. Rec.* 9.6.06.
Hamilton, J. R. R.	N. Edinb. 1894	—	1902–04	O. 1904–22 x	—	—
Hogg, T.	Edinburgh 1900	—	—	—	Pp. Johannesburg	—
Jackson, A. F. S.	Glasgow 1899	—	1902–03	—	—	—
Jowett, W.	N. Edinb. 1898	—	—	C. 1906–11 x	—	Reappointed 1912–14.
Joyce, J. F.	Edinburgh 1899	1901–06	—	O. 1909–26 x	—	† Kroonstad 30.8.26. *Jl.* III (3) '32.
Jarvis, E. M.	London 1894	—	—	S.R. 1901–07	—	*Jl.* Nov. 1929.
Kidd, W.	N. Edinb. 1888	—	—	—	—	- Wynberg 29.10.01.
Kellett, E. B.	N. Edinb. 1889	1901	—	—	T.T. Police, 1902	† Johannesburg 6.9.32. *Jl.* III (4) '32.
Lindsay, F.	London 1895	—	—	T. 1903	—	—
MacDonald, R.	Ontario 1891	—	—	—	Pp. Germiston	*Jl.* IV (1) 1933.

MacNae, A.	N. Edinb. 1899	—	1902	T. 1902–34 x	—	Until 1915 at Standerton Stud Farm.
MacGregor, C.	Edinburgh 1886	—	—	—	—	† Ventersdorp 22.1.02. *Vet. Rec.* 26.8.33.
May, G.	London 1901	—	—	T. 1902–34 x	—	—
McKie, W.	Glasgow 1887	—	—	O. 1909–11 x	—	—
Neill, J.	Glasgow 1892	—	—	C. 1904–06	—	† Vryburg 14.12.06. *Cape A. Jl.* XXX.
Neale, C. R.	Edinburgh 1893	—	—	—	—	† 1921. *Vet. Rec.* 26.8.33. Came to S.A. from New Zealand.
Oliver, H. O.	London 1898	1902–06	—	O. 1906–07	—	† Germany 1918. *Jl.* III (3) '32.
O'Neill, O. A.	Edinburgh 1903	—	—	N. 1902–06	—	In S.R. C.V.D. from 1897–1901.
Parker, J. M.	McGill 1890	—	—	—	—	† Worcester 15.8.02. *Vet. Rec.* 26.8.33.
Pollard, J.	N. Edinb. 1897	—	—	T. 1902–06	—	—
Peddie, J.	Edinburgh 1893	1901–03	—	—	Mun. Johannesburg 1903–05	† 5.8.32 Johannesburg. *Jl.* III (4) 1932.
Pakeman, W. G.	London 1892	—	—	C. 1903–16 x	—	† Aliwal North 20.1.16. *Jl.* III (2) 1932.
Pilkington, J. K.	London 1881	—	—	—	Pp. Johannesburg	—
Pye, W. M.	Edinburgh 1891	—	1902	—	Pp. Johannesburg	† Pretoria 1.2.05 *Vet. Rec.* 4.3.05 In private practice Jo'burg from 1896.
Robinson, J. M.	Unqualified	—	—	—	Pp. Pretoria	† Pretoria 11.4.37.
Runciman, B.	N. Edinb. 1900	1902–06	—	—	Pp. Johannesburg	—
Sykes, G.	Edinburgh 1900	—	—	—	—	† Charlestown 14.3.02. *Vet. Rec.* 26.8.33.
Stokes, E. E.	Edinburgh 1899	—	1902–03	—	Pp. Port Elizabeth	† 27.11.25 Port Elizabeth. *Vet. Rec.* 20.3.36.
Stockman, S.	Edinburgh 1890	—	—	T. 1903–05	—	† Glasgow 2.6.26. *Jl.* III (3) '32. Came from India as P.V.S. Transvaal.
Smith, J. I.	Glasgow 1898	1902–06	—	T. 1906	Mun. Johannesburg, 1907	*Sun. Agr. Jl. S.A.* Jan. 1930.
Stevens, W. S.	Edinburgh 1901	—	1902	—	—	—
Sturge, H.	London 1894	—	—	T. 1902–05	Pp. Potchefstroom	† Potchefstroom 10.7.1932.
Skues, F. M.	Edinburgh 1897	—	1902–05	O. 1905–21 x	—	† Bloemfontein 18.8.21. *Jl.* III (3) 1932.
Sharpe, C. M.	London 1899	—	—	N. 1902–07	—	—
Stranaghan, D.	Edinburgh 1890	—	—	—	Pp. Potchefstroom	† Johannesburg 22.12.31. *Jl.* III (1) 1932.
Stokoe, R.	London 1901	—	1902	—	Pp. Johannesburg	—
Tate, J. M.	London 1899	1901–08	—	T. 1908–14 x	—	† Plumstead 26.12.33. *Jl.* V (4) '34.
Tasker, H. K.	Edinburgh 1897	—	—	O. 1904–09	—	† 23.1.09 drowned.
Tamblyn, D. S.	McGill 1901	—	—	—	Pp. Johannesburg	—
Thomas, R. P.	London 1896	—	—	—	—	In private practice at Capetown before War. † 1914 at Mombasa, B.E.A. Was in service of B.E.A. Adm. *Vet. Rec.* 12.4.13.
Webb, H. M.	N. Edinb. 1898	1901	—	T. 1902–25 x	—	—
White, F. F. G.	London 1894	—	—	—	Pp. Pretoria	
Walker, J.	Edinburgh 1896	—	—	T. 1903–18 x	—	In private practice Capetown before War.

(69) *Abbreviations*: x Transferred to Union C.V.D. on amalgamation of four Colonies 31.5.1910.
Jl. = *Jl. S.A.V.M.A.*
Pp. = private practice.
O., T., N., C., S.R. = Orange River Colony, Transvaal, Natal, Cape Colony, and Southern Rhodesia.
Mun. = Municipal service.

(70) For History of South African Constabulary, see *Vet. Rec.* 22.10.32.

(71) For History of Repatriation Department, see *Vet. Rec.* 27.9.30.

(72) No history yet compiled.

(Fort Napier), Mooi River, and Howick; *Transvaal*—Barberton; *Orange River Colony*—Bloemfontein (Tempe), Glen, and Ladybrand.

As the South African Garrison was reduced so were some of the above stations closed (*e.g.* Standerton in February, 1909), until in 1913 there remained troops (and details) only at Cape Town, Pietermaritzburg, Mooi River, Potchefstroom, Pretoria, Bloemfontein, and Harrismith.

TABLE VIII.

CAVALRY REGIMENTS AND GARRISON CENTRES FROM 1903-1914.

	1902	
1st Dragoon Gds.	In S.A.	At Potchefstroom until Oct. 1903.
2nd ,, ,,	In S.A.	At Roberts Heights Aug. 1902-Dec. 1907.
3rd ,, ,,	In S.A.	At Harrismith June 1902-July 1904.
4th ,, ,,		At Middelburg (Cape) 1905-1908.
5th ,, ,,	Left Mar.	At Tempe Mar. 1904-Nov. 1908.
6th ,, ,,	Left Aug.	At Tempe Dec. 1908-Dec. 1912.
7th ,, ,,	In S.A.	At Ladysmith until 1904.
1st Dragoons	Left Sept.	At Roberts Heights Oct. 1911-Feb. 1913, then to Potchefstroom until Aug. 1914.
2nd ,,	In S.A.	At Middelburg (Tvl.) until 1904.
3rd Hussars	Left Oct.	At Roberts Heights Dec. 1907-Nov. 1911.
4th ,,	—	At Potchefstroom Nov. 1905-Nov. 1909.
5th ,,	Left S.A	—
6th Dragoons	Left S.A.	—
7th Hussars	In S.A.	At Krugersdorp Oct. 1902-July 1904, then to Potchefstroom until Nov. 1905.
8th ,,	In S.A.	At Roberts Heights June 1902-Oct. 1903.
9th Lancers	Left. Mar.	At Potchefstroom Nov. 1906-Nov. 1910.
10th Hussars	Left Sept.	At Potchefstroom Dec. 1912-Aug. 1914.
11th ,,	—	—
12th Lancers	Left Sept.	At Potchefstroom Nov. 1910-Dec. 1912.
13th Hussars	Left Sept.	—
14th ,,	In S.A.	At Kroonstad until March 1903.
15th ,,	—	At Potchefstroom Nov. 1909-Dec. 1912.
16th Lancers	In S.A.	At Middelburg (Tvl.) until Oct. 1904.
17th ,,	Left Sept.	—
18th Hussars	Left S.A.	—
19th ,,	In S.A.	At Standerton June 1902-April 1903, then to Kroonstad until Jan. 1904.
20th ,,	In S.A.	At Heilbron until March 1903.
21st ,,	—	—

There were also regiments of Mounted Infantry, the 2nd, 4th, 5th, 6th, and 8th being frequently mentioned in the earlier veterinary reports. Middelburg (Transvaal), Standerton, and Harrismith were their stations.

Remount depots were situated at Mooi River, Glen, and Pretoria.

Also employing many horses and mules were the Royal Artillery (both Horse and Field), Army Service Corps, and Royal Engineers.

TABLE IX (75).

LIST OF VETERINARY OFFICERS AFTER COMPLETE DEPARTMENTALISATION OF A.V.S. SENT TO SOUTH AFRICA AFTER CONCLUSION OF SECOND ANGLO-BOER WAR (31.5.02 — 1914).

Name.	College.	Qualified.	In South Africa. From	To	Retired.	Died	Notice of Death. P=Photo. X=Career.
*Fail, F.	Edinburgh	1898	16.5.03	21.12.03	6.1.20		
*Moore, F. A. S.	London	1900	,,	28.12.08	24.4.24		
*Ludgate, W.	Edinburgh	,,	,,	21.12.03	30.4.26	6.5.27, Ayr	D.G./A.V.D. letter of 11.11.35.
*Plunkett, R. A.	London	1899	,,	15.4.09	24.4.24		
*Jelbart, W. A.	Edinburgh	1901	,,	11.11.03	16.6.28	7.5.34, Dursley	*Jl. R.A.V.C.* Aug. '34.
*Nicholas, I.	London	,,	,,	10.3.08		29.9.25, India	*Vet. Rec.* 10.10.25X.
*Pallin, S. F. G.	,,	,,	,,	27.1.04	13.7.29	18.2.30, Shaldon	*Vet. Rec.* 1.3.30X.
*Tapley, J. J. B.	,,	1899	,,	1.5.08			See *Jl. R.A.V.C.* Nov. 1933. P.X.
*Allen, H.	,,	,,	,,	21.12.03	21.8.29		
*Oliver, E. S.	,,	1901	,,	29.3.08	17.6.23		
*Greenfield, H.	,,	,,	,,	21.12.03	23.10.28		
*Schofield, W. E.	,,	,,	,,	22.1.08			
Webb, E. C.	,,	1902	,,	21.1.04		30.5.34, Winnipeg	Dr. F. Bullock's letter of 19.8.35.
Wadley, E. J.	,,	1901	,,	29.3.08	4.2.21		
*Steevenson, J. R.	,,	1902	,,	21.1.04	18.2.30		
*Neale, W. R.	,,	1899	,,	31.1.08	30.1.24		
(a) *Leaning, A.	,,	1901	,,	8.7.04	5.5.31		
*Gibbs, H. E.	,,	1902	,,	21.1.04			
Kirby, H.	New Edinburgh	,,	11.5.04	19.12.09		6.1.31, Naina Tal, India	*Vet. Rec.* 24.1.31X.
Burridge, T. E.	London	1904	12.10.05	31.1.11			
Marriott, T.	Glasgow	1883	,,	3.9.09	4.9.09		
(b) Harris, P. J.	Edinburgh	1896	,,	3.1.11	24.4.24		
Holness, H. J.	London	1903	,,	30.11.10			
Haigh, T.	,,	1901	15.5.06	14.6.07	1.6.19	28.1.29, Bedford	*Vet. Rec.* 9.2.29.
Turnbull, D. O.	,,	1903	6.7.06	15.5.08	16.5.08		
McKenzie, K. M.	Glasgow	,,	10.9.06	13.12.11		6.10.25, India	*Vet. Rec.* 31.10.25X.
Brookes, W. T.	London	,,	,,	21.2.08	22.2.08		
Andrews, J. O.	,,	,,	1.7.07	9.3.13			
Audas, R. S.	,,	,,	27.11.07	5.10.09	6.10.19		
Jones, H. L.	,,	1903	,,	27.1.11	28.1.11		
Taylor, W. H.	,,	1905	,,	7.2.13.		12.2.19, Active Service	*Vet. Rec.* 1.3.19.
Leckie, V. C.	,,	1906	,,	17.12.12			
Hodgins, A.	Dublin	,,	1.2.08	9.3.13			
Stewart, H. A.	Edinburgh	1905	,,	28.2.13	26.9.23		
Bone, H.	,,	1906	,,	7.2.13			
Tindle, R.	,,	1904	,,	29.12.12	13.5.20		
Wright, W. N.	,,	1890	29.5.08	26.11.12	27.11.12	7.8.33, Torquay	Dr. F. Bullock's letter of 19.8.35.
Taylor, E.	,,	1884	7.2.09	5.3.14	24.3.18	7.10.31 Kensington	*Vet. Rec.* 24.10.31. *Jl. R.A.V.C.* Nov. 31X.
Appleton, A. F.	New Edinburgh	1881	Oct. 1909	Oct. 1913	5.11.13		
Hearne, E.	Dublin	1905	20.10.09	19.9.14		3.2.32, Malta	*Vet. Rec.* 13.2.32X. *Jl. R.A.V.C.* May '32X.
Anthony, W. S.	London	1895	31.3.10	13.5.10			See *Jl. R.A.V.C.* Feb. 1930P.
*Rainey, J. W.	Edinburgh	1904	14.12.10	5.8.13	6.8.13		
Tillyard, G. E.	London	1907	,,	19.9.14			
Bright, W. F. L.	,,	,,	17.12.10	18.3.13		22.11.21, India	*Vet. Rec.* 10.12.21X.
Pallin, W. A.	New Edinburgh	1894	6.5.11	12.8.13	25.10.29		See *Jl. R.A.V.C.* Nov. '29X.
Nuthall, C. E.	London	1883	19.9.13	19.9.14	9.7.19		

(a) A. Leaning was again in South Africa from 21.2.05 to 22.1.08.

(b) P. J. Harris was again in South Africa from 3.5.13 to 19.9.14.

* Served as C.V.S. in Anglo-Boer War (Smith 1927, p. 259) ; but Gibbs and Rainey qualified after the Campaign!

(75) See Tables III and V for details of V.O's. who remained in S. Africa after 31.5.02.

mentions (letter Feb. 24th, 1927) that as far back as the eighties, following a " very serious outbreak of Horsesickness " (probably 1887), circumstances (*e.g.* " Thoroughbred horses . . . . kept for racing and shed purposes were never attacked with the disease ") led him " to the conclusion that a main factor in its not attacking these animals was the housing question. I put the matter to the test and advised that closed up properly ventilated and sound floored stables should be provided with suitable drainage. The G.O.C. at the Cape made an inspection at Pietermaritzburg in consequence of my report and directed that a stable according to my plans should be prepared, the result being that in this stable we never had Horsesickness when outbreaks occurred in

Fig. 20.

ARTILLERY BARRACKS, PRETORIA.

The P.V.O's office was on the extreme left, ground floor. The photo was probably taken before the Second Anglo-Boer War. An additional block was built in front of the above in 1926-7.

other stables . . . ." Here is evidence that Rowe was responsible for the first military stable as opposed to a shed.

The stables at the Artillery Barracks, Pretoria, were built of brick in 1896 for the horses of the Staats Artillerie, but are now used for stores. Blenkinsop (*Annual Report of A.V.S. for* 1906) states that the stables " *recently* built in the Middelburg (Cape) cantonments, on the Indian plan, with two rows of horses facing *inwards* towards a central passage and a deep verandah behind them, is preferable to the stables in the Transvaal and Orange River Colony, where the two rows of horses face outwards—an arrangement suitable for permanent build-

Fig. 21.

FULL DRESS UNIFORM LIEUTENANT, A.V.C. 1911 (*).

The above taken from Plate J. facing p. 232 of Smith's *History of the R.A.V.C.*, shows :—

Headdress. Infantry helmet with ball, first adopted for lieutenants (and captains) in 1891. The plate was an "eight-pointed star, within the garter, in silver was A.V.C. in monogram on green enamel" (p. 252).

Tunic as in 1883 except that the cuffs and collar were of maroon cloth, the departmental badge was worn on the collar, and the buttons "bore the words Army Veterinary Corps".

Belts and sword slings as in 1904 were of gold lace with maroon stripe.

Overalls. Blue with double stripe of maroon cloth.

(*) The Dress Regulations distinguish between administrative rank (A.V.S.) and executive officers of A.V.C. Note that although Khaki service dress was worn during the Second Anglo-Boer War, it was not officially adopted until 1904.

ings in a cold climate, but not for stables built of corrugated iron and exposed to a semi-tropical sun. Such stables are very cold in winter, and the sun heats the iron and makes them into ovens in the summer." (p. 51).

The roofs in double-row stables would be of gable pattern, the floors of concrete and the mangers of corrugated iron (76).

The stables forming part of a veterinary hospital were no doubt of the general type referred to above and as an example a photograph is shown of those at Roberts Heights. (Fig. 24). However, Blenkinsop states that at Middelburg (Cape) the Veterinary Hospital consisted of " makeshift shelters . . . . only intended . . . . until some more satisfactory arrangements could be made. At present the healthy animals . . . . are occupying good hygienic stables, while the sick are crowded into sheds with mud floors, no drainage, and insufficient shelter from sun, dust and cold." (p. 52).

The importance of hard floors was thoroughly proved in 1904 during the campaign against epizootic lymphangitis. The percentage of admissions was reduced in units having stables with concrete floors.

(e) Annual Reports of A.V.S.

Since much of the subsequent information is obtained from the *Annual Reports of the A.V.S., viz.* from year ending 31.3.1904 to 31.3.1913, a few comments concerning the publication are indicated. The reports have necessarily a limited circulation and in South Africa there is not a single complete set. Thanks, however, to Dr. Fred Bullock the reports mentioned below were made available :—

Year ending	General Report signed by	S. African Report by	General Report addressed to	S. African Report Size (pages)
31.3.1904	H. Thomson, D.G., A.V.S.	Col. F. Smith, P.V.O.	Dir. of Transport & Remounts	24
31.3.1905	,, ,,	,, ,,	,,	47
31.3.1906	,, ,,	Col. J. A. Nunn ,,	,,	18
31.3.1907*	F. Smith ,,	Lt. Col. L. J. Blenkinsop ,,	,,	38
31.3.1908*	,, ,,	P.V.O., S. Africa	,,	59
31.3.1909*	,, ,,	,, ,,	,,	47
31.3.1910*	,, ,,	,, ,,	,,	39
31.3.1911*	R. Pringle ,,	,, ,,	,,	37
31.3.1912*	,, ,,	,, ,,	Quartermaster-General	35
31.3.1913*	,, ,,	Asst. Dir. Vet. Ser.	,,	35

(76) The mangers at the Artillery Barracks were made of cast iron. A few are still to be seen at the Transport stables, Roberts Heights.

* Although for the year ending March 31st, the cover of the report for year ending 31.3.1907 merely states 1906 and so on! The size of the page is also no longer foolscap but royal octavo.

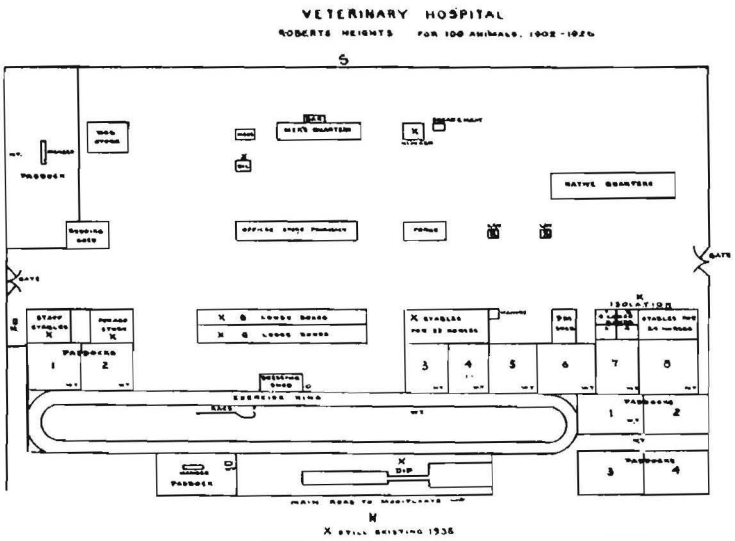


Fig. 22.

PLAN OF VETERINARY HOSPITAL, ROBERTS HEIGHTS.

The Veterinary Hospital was built after the Second Anglo-Boer War and used by the A.V.C. until 1914. It was then used by the South African M.R. until its disbandment in 1926. The stables were then converted into native quarters.

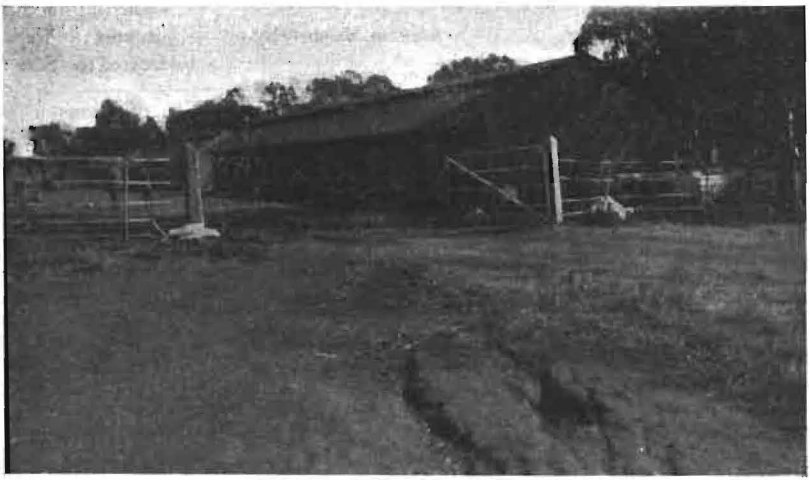


Fig. 23.

MAIN ENTRANCE, VETERINARY HOSPITAL, ROBERTS HEIGHTS.

The full title of the report is *The Annual Statistical and General Report of the Army Veterinary Department*. From and including the year ending 31/3/1906 the word "Service" replaces "Department". The printers throughout the series are Messrs. Harrison and Sons, who do the work for His Majesty's Stationery Office. The Report for the year ending 31.3.1910 (*i.e.* with 1909 on cover) was issued as a Command Paper, "so that it was presented to both Houses of Parliament, and obtained the same circulation as is accorded the Army Medical Report. In the next administration, however, the report was reduced to bare figures and tables" (Smith 1927, p. 230), which remark applies particularly to the South African Command. The A.V.S., especially during Major-General Smith's period as D.G., investigated many causes of wastage, and it seems a pity that such researches are not made available by veterinary educational institutions.

Of particular value is the statistical information, and for this purpose the return is divided in 13 classes of ailments, based mainly on the anatomical system involved, *e.g.* Class 1, general diseases comprising (*in* 1904) among other conditions, horsesickness, anthrax, and biliary fever. Class 2 included the affections of the respiratory apparatus, Class 3 those of the circulatory system, Class 4 the urinary, Class 5 the generative, Class 6 the digestive, Class 7 the liver and spleen, Class 8 the nervous system including tetanus, Class 9 the tegumentary, embracing mange, Class 10 the locomotory, and Class 11 zymotic diseases *i.e.* communicable diseases. These were glanders, variola, strangles, influenza, rabies, lymphangitis and epizootic fever. Class 12 included maladies of the visual apparatus including specific ophthalmia and Class 13 surgical diseases and accidents.

Each of the above classes was again subdivided into a varying number of affections, the minimum being 8 for zymotic diseases (farcy being enumerated), and the maximum 39 for the locomotory system. No reference was made to osteoporosis.

By 31.3.1907 certain improvements had been effected in the nomenclature, *e.g.* osteoporosis was placed in Class 1, and Class 11, now entitled specific diseases, included biliary fever, tetanus and horsesickness. The designation liver and spleen (Class 7) was now lymphatic system, liver conditions having been transferred to Class 6.

TABLE X.

(f) STATISTICS RELATING TO ARMY HORSES IN SOUTH AFRICA.

Year ending	31.3.04	31.3.05	31.3.06	31.3.07	31.3.08
Strength	11,781	6866	6257	6083	5603
Admitted to treatment %	132.79	130.08	120.72	117.8	149.11
Died and destroyed %	6.13	6.48	3.37	4.8	4.8

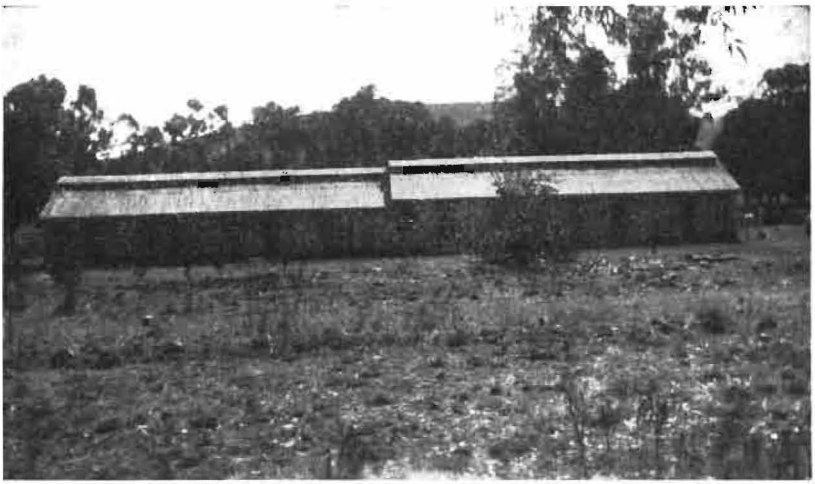


Fig. 24.

CORRUGATED IRON STABLES, VETERINARY HOSPITAL, ROBERTS HEIGHTS.

Fig. 25.

OLD VETERINARY HOSPITAL, ARTILLERY BARRACKS, PRETORIA.

This is now used for stores.

TABLE X—(*continued*).

Year ending	31.3.09	31.3.10	31.3.11	31.3.12	31.3.13
Strength	4807	4275	4199	4076	3281
Admitted to treatment %	122.9	114.26	104.8	112.63	104.38
Died and destroyed %	3.46	4.49	2.97	3.06	3.04

The total mortality does not of course represent the only wastage. There are castings for disease and other causes (77). The following figures are instructive :—

	31.3.09	31.3.10
Castings for disease	6.63	4.37
Castings for other causes	15.87	7.04

TABLE XI.

STATISTICS RELATING TO ARMY MULES IN SOUTH AFRICA.

Year ending	31.3.04	31.3.05	31.3.06	31.3.07	31.3.08
Strength	7250	4024	3414	2623	2515
Admitted to treatment %	66	74.36	66.91	55.68	55.7
Died and destroyed %	4.14	4.36	2.92	3.33	3.29

TABLE XI—(*continued*).

Year ending	31.3.09	31.3.10	31.3.11	31.3.12	31.3.13
Strength	2473	1914	1729	1585	1338
Admitted to treatment %	48.24	43.57	42.1	47.2	47.3
Died and destroyed %	1.93	4.96	2.71	1.82	3.2

The relative figures regarding total wastage for the periods corresponding to those given for horses, Table X, are :—

	31.3.09	31.3.10
Castings for disease	.2	23.3
Castings for other causes	1.73	4.85

(*To be concluded.*)

(77) The cause of casting was not regularly recorded until October, 1903, when in conjunction with the Assistant Inspector of Remounts . . . . the following classification was adopted :—(a) unsound, (b) undersized, (c) bad conformation and unsuitable, and (d) defective vision.

Interesting Clinical Case in a Male Spaniel.

By J. B. QUINLAN, F.R.C.V.S., Dr.Med.Vet., D.V.Sc., Onderstepoort.

Subject.—A black spaniel dog, aged 9 years, weighing 30 lb., arrived in the clinic on the 11th May, 1937.

History.—The dog was a house dog, and a great pet of the 12-year-old son of the owner.

The veterinary surgeon who sent the dog to the hospital stated that the animal passed blood-stained urine for a period of 20 days, since the 22nd April. No other symptoms had been noticed, except frequent attempts at micturition during which drops only of blood-stained urine were passed. This had gone on since the first examination, but the symptoms gradually became accentuated with the passage of small blood clots and deeply blood-stained urine.

Status Praesens.—The condition was good and the coat smooth. The circulatory, digestive, locomotory, and nervous systems showed nothing abnormal. There was slight pain on pressure over the prepubic region. The bladder could be palpated through the abdominal wall as a somewhat firm oval body. Catheterisation showed that it contained urine mixed with blood. A radiogram, without contrast filling, showed a clear picture of the outline of the bladder (see illustration).

Diagnosis.—Newgrowth in the bladder.

Prognosis.—Guarded until nature of the new-growth could be established histologically.

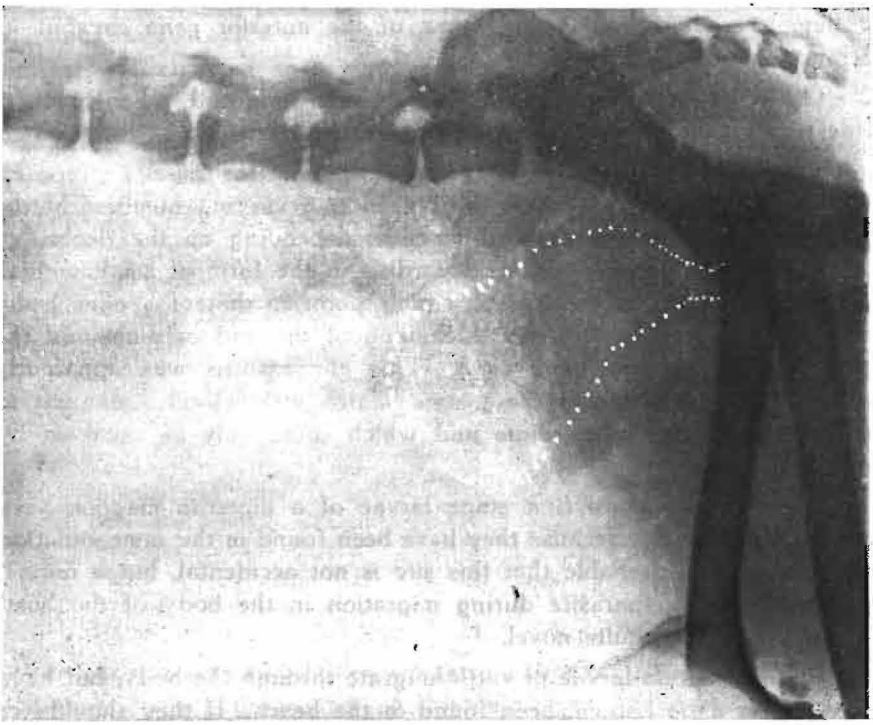
Treatment.—On the 12th May a prepubic laparotomy was performed under general anaesthesia : Eukodal 2 ccm. subcutaneously, followed 1 hour later by 3 ccm. of pernocton intravenously.

The bladder was withdrawn through the wound. About 25ccm. of dark, blood-stained urine was removed by puncture with a hypodermic needle. The empty organ showed a local circumscribed thickening, somewhat circular in outline, about 5 cm. in diameter, over the fundus. The subperitoneal vessels over this area were markedly distended and very tortuous. The thickened area presented a dark red appearance. The bladder was opened by a median incision, about 6 cm. long, on its dorsal surface. The whole mucosa showed hyperaemia ; the thickened area presented a number of nipple-like projections which were oozing blood. The wall of the thickened area measured about 2 cm. This thickening showed distinct delimitation. There was little merging between it and the remainder of the wall. As the nature of the thickening was not known, it was decided to remove it. This was done by clamping it off with bowel forceps and excising it with a scissors. These manipulations caused marked contraction of the remaining portion of the organ so that suturing was difficult. When suturing (which was done by Czerny-Lembert sutures of fine catgut) was completed, the bladder appeared

like a finger about 7 cm. long and 2.5 cm. in diameter. The laparotomy wound was closed by three layers of fine catgut sutures. Anaesthesia persisted for about 10 hours.

The histological report on the section of the bladder removed, made by Dr. C. Jackson of this Institute is as follows :—

" Due to pronounced hyperaemia and oedema of the *propria mucosae*, the folds of the mucosa are entirely obliterated. The blood in the vessels shows neutrophilia. The superficial part of the propria is diffusely infiltrated by neutrophiles and erythrocytes, and at many points where the infiltration becomes concentrated the overlying epithelium is soaked with blood and infiltrated with neutrophiles. In these parts also there may



Radiograph showing outline of the bladder just prior to the operation.

be pronounced signs of oedema of the epithelial cells and superficial desquamation with the formation of minute erosions not extending into the depths of the epithelium.

Diagnosis : Acute localised haemorrhagic cystitis."

One wonders if entirely local lesions of this type are common in the dog, and if so whether partial incision of the bladder is justified as a routine form of treatment ?

The animal made an uneventful recovery. The urine appeared normal on the second day following the operation. The dog was discharged on the 8th June and at the time of writing, 3 months after the operation, still continues in good health.

A Note on the Occurrence of a Dipteran Larva on the Endocardium of the Bontebok.

By C. C. WESSELS, B.V.Sc., V.M.D., Bredasdorp.

While an investigation into an unknown disease in bontebok (*Damaliscus pygargus*) was being carried out in the Bredasdorp district, a sick buck, in which spinal paralysis was the outstanding symptom, was killed. At *post mortem,* the right atrium revealed chronic fibrous endocarditis. On closer examination there were found large numbers of a small, whitish, maggot-like worm attached to the surface of the endocardium as well as on to the intima of the anterior *vena cava* at its entrance into the auricle.

These worms were between 1 and 2 mm. long and, when touched, made twisting movements like those of oestrus larvae.

Subsequently four healthy bontebok were shot for museum purposes. At autopsy these also revealed the presence in varying numbers of the same maggot-like worms. In each case, depending on the degree of infection, there was a chronic endocarditis in the form of small whitish nodules, varying from the size of a pin's point to that of a pin's head, and causing a roughening and thickening of the endocardium and the intima of the entering *vena cava.* This endocarditis was apparently caused by the irritation of the worms, which were closely adherent to the surface of the endocardium and which could only be removed by forcible scraping.

These parasites are first stage larvae of a dipteran maggot, very probably an oestrid. Because they have been found in the same situation in five buck it is probable that this site is not accidental, but a normal one for the young parasite during migration in the body of the host, which is something quite novel.

The hypoderma larvae of cattle migrate through the body, but have never, as far as is known, been found in the heart. If they should get there it certainly would be accidental and happen rarely.

It is uncertain whether these larvae are of much pathogenic importance in the bontebok. However, in view of the fact that the unknown disease has not yet been cleared up, there is the possibility that these larvae, during migration in the body of the host, may cause injury to the spinal cord and so produce the paralysis.

As it is impossible to identify the species from these first stage larvae only, further attempts will be made to obtain as many developmental stages as possible and also to breed the fly from mature maggots.

ABSTRACTS.

The Presence of the Bovine Tubercle Bacillus in Human Tuberculosis.

(Ueber das Vorkommen der Perlsuchtbazillen bei menschlicher Tuberkulose : Eduard Gróh (1936). *Ztschr. f. Tuberkulose* vol. 74 : 263-270).

In cultures of the tubercle bacillus of human origin, Gróh can identify two morphologically differing germs, one of which he considers is the bovine tubercle bacillus. In his investigation, he examined 149 specimens, taken at autopsy from man. A portion of each sample was examined microscopically, after straining by Ziehl-Neelsen's method, and another was cultured on a medium similar to that devised by Loewenstein (an egg-asparagine-potato-starch medium).

In all but one specimen, " bovine " bacilli were demonstrated, even if only in small numbers. In smears from culture they might be found in every microscopical field or a prolonged search might be required before one clump was encountered. The accompanying table gives the frequency of the " bovine " and " human " bacilli in cultures from different types of tuberculosis of man.

	Bovine bacilli		
Organ affected	*Many*	*equal in no. to human*	*Few*
Lung	9	2	26
Kidney	9	10	28
Prostate and testes	2	2	1
Bone and joint	11	3	17
Meninges	5	3	4
Pleura	3	5	6
Appendix	—	—	1
Spleen	1	—	—

A correlation existed between the number of " bovine " bacilli in a culture and the pathogenicity of this culture for the rabbit ; the larger the number the greater the pathogenicity.

A possible reason why the " bovine " bacillus has not been observed more frequently in material of human origin is that the common method of isolation is to inoculate a suspension of the organ or exudate into a guinea-pig, to culture the spleen and the liver on glycerinated serum, to transfer the growth obtained thereon to glycerine-broth, and to use the culture in this medium to infect a rabbit. Because the " human " bacillus in artificial culture grows much more rapidly and luxuriantly than the " bovine ", the final test culture will consist predominantly of the " human " germ and therefore the disease in the rabbit will be of the classical human strain type, i.e. a slow but progressive tuberculosis.

The Importance of Bovine (Tubercle) Infection.

(Den bovine Infektions Betydning. K. A. Jensen (1935). *Ugeskr. Laeg.* 97 : 204).

During the last three years, 3,000 specimens from man were examined for the presence of the bovine and/or human type of the tubercle bacillus. The material came from four different areas : (1) Copenhagen (bovine tuberculosis presumably not prevalent), (2) "Islands" (bovine tuberculosis not prevalent), (3) East and North Jutland (bovine tuberculosis moderately prevalent), and (4) West Jutland and North Schleswig (bovine tuberculosis very prevalent). The data on the demonstration of each type of bacillus in tuberculosis of the lung, the bones, joints and genito-urinary apparatus, the cervical lymph-glands and the meninges are given, with the incidence for people of age groups 0—15 years, 15—30 years, and over 30 years.

The highest incidence of infection due to the bovine type of bacillus is found in those residing in districts where bovine tuberculosis is prevalent (e.g. pulmonary tuberculosis, area 2 : 15—30 years, 2.6%, area 4 . 15—30 years 16.9%); children are more frequently infected with the "bovine" bacillus than are adults (e.g. T.B. of cervical lymph glands area 3 : 0—15 years, 81.3%, over 30 years, 18.8%), and finally the order of the frequency of the bovine infection in man would appear to be (1) the cervical lymph-glands, (2) the meninges, (3) the bones, joints and genito-urinary organs, and (4) (far down the list) the lungs.

Frequency and form of tuberculous affentions in man, caused by the bovine bacillus.

(Fréquence et forme des affections tuberculeuses provoquées chez l'homme par le bacille bovin. L. Lange (1937). *Bull. de l'Office internat. d'Hygiène publ.* 28 : 317-328).

Lange has collected data on the incidence of bovine tuberculosis in man. A satisfactory abstract is difficult to make because of the large number of tables and figures given ; only a few will be quoted and the reader is referred to the original for details.

Incidence of bovine tuberculosis in man.

Germany (to 1936) 1,165 cases, 13.5% due to the bovine bacillus.
England (1932) 1,697 cases, 17.6% due to the bovine bacillus.
Scotland (1932) 1,226 cases, 26.0% due to the bovine bacillus.
Canada (1932) 436 cases, 10.5% due to the bovine bacillus.
Japan (1932) 100 cases, 4.0% due to the bovine bacillus.

The bovine organism is found most frequently in tuberculosis involving the cervical lymph-glands, the skin and the bones and joints and least often in the lungs. This type of germ is encountered most

often in children less than five years of age. There is not a single form of tuberculosis in which the bovine bacillus has not been found.

The percentage of bovine infection given for different countries varies so greatly that definite conclusions cannot be drawn, but the following general statements may be made :—

1. In countries like Japan, India, and (in a certain measure) Norway, the bovine bacillus does not play a big part in the causation of tuberculosis in man.

2. In Germany and perhaps Switzerland, a considerable number of infections are caused by it.

3. In England and Scotland, it is the cause of a large number of infections.

In the campaign against tuberculosis, the bovine bacillus must be seriously considered, but even if the disease were completely eliminated from cattle, the total number of human infections (taking all peoples into consideration) would be reduced only to a very slight extent. The most important and the commonest form of the disease in man, phthisis, is caused nearly always by the human type of germ.

The Incidence of Human Tuberculosis of Bovine Origin in Great Britain.

(L'incidence de la tuberculose humaine d'origine bovine en Grand-Bretagne). Sir Weldon Dalrymple-Champneys (1937). *Bull. de l'Office internat. d'Hyg. publ.* **29** : 329-336.

Over 90.0 per cent of tuberculous cervical lymph glands, 57.3 per cent of cases of lupus and 26.7 per cent of pulmonary infections in children of 5 years and under were caused by the bovine bacillus. Including all age groups, 50.0 per cent of cervical lymph-gland infection and 1.4 per cent of phthisis disease were produced by this type. In Scotland, of 13 cases of lupus in children of 5 years and under, all were caused by the bovine germ.

Infected milk is undoubtedly the principal cause of bovine tuberculosis in man, and although there is no certain figure for the number of cows giving infected milk one can deduce from the report of the Cattle Diseases Committee (1934) that this proportion is at least five cows per thousand. The Manchester Health Authorities have shown (1935) that there are 136 farmers of 1,035 supplying the town with milk who send in tubercle infected milk.

Pasteurization of milk is discussed, but it is noted that this is not always properly carried out. An ordinance was issued by the Minister of Health in 1936, giving clear and precise details of pasteurization methods.

Frequency of Human Tuberculosis in Scotland due to the Bovine Bacillus.

(Fréquence de la tuberculose humaine due au bacille bovin en Écosse. M. T. Morgan (1937) *Bull. de l'Office internat. d'Hyg. publ.* **29** : 337-341.)

Data, confirming those recorded in the previous abstracts, are given. The bovine bacillus is encountered most frequently in children, it is chiefly responsible for tuberculous cervical lymph glands in young people and it plays a small part in causing phthisis (all age groups included).

One important point comes out—the incidence of infection in man due to the bovine bacillus is higher in rural than in urban districts.

Frequency of Tuberculosis of the Bovine Type in Man in Holland.

(Fréquence de la tuberculose de type bovin chez l'homme dans les Pays-Bas.) A. Charlotte Ruys (1937) *Bull. l'Office internat. d'Hyg. publ.* 29 : 342-347.

The number of cases examined is not so large as that recorded in the previous abstracts, but again the percentage of bovine infection is higher in children than in adults.

In 1930 (Van Loghem and Vedder), 2 per cent of milk samples were found to be infected with the bovine germ and in Amsterdam the food control service records the following figures : 1931—2.4 per cent, 1932—2.2 per cent, and 1933—0.8 per cent. In Amsterdam, abattoir figures of tubercle infection in bovines were 35 per cent in 1933 and 34 per cent in 1934. Of 11,000 cattle tested with tuberculin, 40.8 per cent reacted. The possibility of infection in the country is greater than in the towns because more raw milk is drunk and further the milk often comes only from a few cows. If one of these animals is infected, the chance of contracting the disease is greater than if the milk were diluted in a large town supply.

Pasteurization of milk should be made compulsory.

The foregoing articles appeared in *Bull. off. Internat. Epiz.* (1937), Vol. 13, No. 9-10.

J. H. M

REVIEWS.

The booklet[1]) which records in considerable detail the findings of the Special Committee of the N.V.M.A. on small animal euthanasia is packed with valuable hints to the practitioner. The different ways of destruction are discussed fully, both from the humanitarian and from what might be termed the aesthetic point of view. The perfect method should cause neither physical nor mental distress to the animal and should be simple and clean. Unfortunately, no one method fulfilled all these desiderata. However, this exhaustive investigation of a large number of cases gives a clear lead as to the choice of a method in the different circumstances of daily practice.

In general, prussic acid—that old standby of the 'veterinary chemist'—is condemned because of the distress caused during the short period preceding death. But its deadly action makes prussic acid a suitable agent to use after preliminary anaesthesia or narcosis. In such cases only intraperitoneal or intrathoracic injections are recommended. The use of strychnine is deprecated—which should not surprise anyone who has observed its action on animals.

The intravenous injection of a warm saturated solution of magnesium sulphate has given excellent results. The only drawback is the method of administration. This requires some skill and assistance. The results are exceedingly good and even if the vein is missed the first time little pain results.

A number of experiments were done with narcotic drugs, and the most useful appeared to be nembutal. Medium-sized cats and dogs could be adequately anaesthetized with comparatively large dosis injected intraperitoneally. This drug is best followed by prussic acid to hasten death.

Generally speaking, the volatile anaesthetics and the use of lethal chambers did not evoke marked enthusiasm. These methods—even when painless—cause considerable mental distress in some subjects and this is sufficient reason to avoid their use where possible.

The small captive bolt pistol was found very suitable in a number of tests. Death was painless and little skill was required. The greatest drawback was the bleeding after death. In cats considerable movement takes place after destruction of the cerebrum and these unconscious spasms may not look pleasant. However, the Committee recommends this method when the choice lies with the operator.

A very interesting chapter is that on destruction by electrical

[1]) *Report of the Special Committee appointed by the National Veterinary Medical Association to study the subject of Small Animal Euthanasia.* 1937. Issued by the N.V.M.A., London. Pp. 59; price 5/-.

means. The Committee investigated a number of instruments and gives its conclusions at length. The type of killer which consists of electrodes applied to the head was very effective if properly applied. Prussic acid should be injected when unconsciousness has ensued. However, the conditions for proper contact of the electrodes with the head are rather rigid, and as distressing symptoms may follow a misapplication of these instruments, the Committee does not recommend them wholeheartedly. Suggestions are made for improving both the instruments and the technique. The Committee was most impressed by some of the electrical cabinet methods for the large-scale destruction of unwanted animals. These are more suitable for dogs than for cats, but gave good results with the latter species. The method is quick, clean, apparently painless, and practically fool-proof with a well-designed cabinet. It must of course be understood that the method is not of much value for odd cases.

Practitioners are strongly advised to consult this book when in any doubt on the subject of the painless destruction of small animals.

M. S.

Britain is often said to be one of the few countries of the world where great social changes are effected without publicity or terrorism, or the loss of blood or political rights. The recent Report of the N.V.M.A. on the Veterinary Profession and its organization [2]) is so admirable, eminently sane, and sensible as to confirm such views.

Not so long ago the average layman, and sometimes the veterinarian himself, was ready to prophesy the gradual extinction of the profession. In England and Scotland, things certainly were bad. The Royal College was tumbling down and research starved for lack of funds. Wealthy old ladies would endow antivivisection societies, but not veterinary education. " It's just too plebeian, my dear." And that was that.

The Jeremiahs proved all wrong. In a changing world the veterinarian found himself more in demand than before. Only his duties were different. Horse-doctoring was superceded by abattoir supervision and similar pursuits.

Conservative to the core, the profession almost resented the creation of new avenues of employment. There is no doubt that expansion was fostered from without—by county councils and allied bodies. The layman was guiding the profession, and his lack of expert knowledge soon led to trouble.

[2]) *The Veterinary Profession and its Organisation for the Control of Animal Diseases.* Report of a Committee of Investigation of the National Veterinary Medical Association of Great Britain and Ireland.

Following the long deferred improvement in veterinary education and the partial acceptance of the responsibilities of research, the profession bestirred itself to achieve efficiency. The election of the committee of investigation was the outward manifestation of the urge to serve the nation better.

The report took eighteen months to prepare. Inquiries were pursued in every quarter. It was not a pleasant state of affairs that was revealed. There was friction between different branches of the profession, when there should have been none. Practitioners often found stock disease regulations pressing unfairly on them. Local authorities interpreted the law pretty well as they wished. In England, veterinarians usually could enter abattoirs only when disguised as " sanitary inspectors ". The anomalies attaching to public appointments were numerous and ludicrous.

As a substitute for chaos, the report offers a suggestion combining all the advantages of socialised veterinary medicine with the best features of private practice. The State will derive the maximum benefit from the minimum expenditure. The scheme can be in full operation with only a little delay. It will inconvenience very few. The private practitioner will remain the bulwark of the profession. Possibly his prestige will be higher than before.

Officials will be appointed solely by the State, for the control of infectious diseases, for the control of the importation and exportation of animals and their products, for the control of biological preparations, for the control of slaughter-houses, for the control of a wholesome milk supply, and for staffing the diagnostic and, possibly, most of the research laboratories. Parish pump politics will no longer be able to influence the purity of the milk or the degree of TB. infection allowed in the carcass. This withdrawal of power from local authorities is not so revolutionary. Even in die-hard America, the Federal veterinarians have the last word in the packing-houses and in the private hog-cholera serum " factories ".

With so many State servants being supported partly, no doubt, by municipal funds, the British Government will be able to encourage attendance at post-graduate courses. This fact alone should tend to raise veterinary education to a much higher level than at present, and the nation will benefit.

The gist of the scheme is given here, but every one should read the full report, which means so much to animal health.

Summary of the Scheme Recommended by the Committee of Investigation :

(a) There should be established a Department of Animal Health, preferably as a separate Department or, failing this, as a Department of an existing Ministry.

(b) The executive of this Department should consist of a principal veterinary

officer, an assistant principal veterinary officer, area veterinary officers, county veterinary officers, municipal veterinary officers, laboratory officers, and certain specialist officers.

(c) Panels of veterinary practitioners should be created.

(d) The principal veterinary officer should be directly responsible to the Minister of State.

(e) The area veterinary officers should be responsible to the principal and assistant principal veterinary officers, and should co-ordinate the activities of the county and municipal veterinary officers and area laboratories.

(f) The specialist services should each have individual chief veterinary officers, who would be responsible to the principal veterinary officer and the assistant principal veterinary officer of the Department.

(g) Laboratories will be required in each area, and should be under the supervision of veterinarians trained fully in laboratory procedures.

(h) The county veterinary officers should superintend, co-ordinate and at times check the routine work, which should be carried out by panels of veterinary practitioners. These panels of veterinary practitioners should form a permanent feature of the service, but every stock-owner should have the right to choose, or change his veterinary surgeon engaged in this work, provided he first consults the State Department.

(i) In some localities it may be necessary to have, in addition to the county or municipal veterinary officer, other whole-time officers for special duties especially in municipalities.

(j) The principal veterinary officer, assistant principal veterinary officer and area veterinary officers should be appointed by the Minister.

(k) The county, municipal, laboratory and specialist officers should be appointed by the State Department. The veterinary practitioners should be chosen by the stock-owner after consultation with the State Department.

J. D. W. A. C.

A little book [3]) arrived recently and although it will probably pass unnoticed by the popular press, its advent means more to the English-speaking world than all the gold of Ophir.

Poultry Diseases: Issued by the N.V.M.A.

This catches the breath and makes us pause to think. The womb of embryology was the egg of the bird. The geese of the Capitol saved Rome. The first bacterial vaccine was pumped into fowls. Cocks were used for the initial studies on hormones. The solution of polyneuritis heralded the great work on avitaminosis.

But the humble hen went on cackling and laying, seeking and receiving little for her labour. The horse doctors and the cow leeches were " practical men ". What skill was needed to treat a pullet with bunged-up eyes? To castrate a fiery prancing stallion in an open field was Science indeed.

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[3]) *Handbook on Poultry Diseases.* Issued by the National Veterinary Medical Association of Great Britain and Ireland. 1937. Pp. 90. Price (to non-members) 10/6d.

One day statisticians had a tale to tell of more ducks and geese, of still more fowls and turkeys, of bigger eggs and better eggs, and of poultry ousting mammals.

The horse and the cow doctors were not impressed, but when they found that laymen doctored birds they invoked the aid of the statutes, and then retired to rest. "You must not help fowls; we cannot help fowls; death to the fowls".

Use a sprat to catch a mackerel! The cow doctor later thought of this. Why shouldn't he "treat" fowls, to win favour in the byres and stables? After all, the diagnosis presented no difficulty—just roup or pip or liver or gall, or too much protein or too much forcing. This "interest" the journals aided and abetted. Garbled articles, garbled special articles, garbled special numbers appeared on cholera, pox, and helminthiasis. Coccidiosis became the joke of veterinary literature. Whenever there was something really funny to relate, it was written about coccidiosis, the author, of course, always "taking his solemn oath." The editors never "found" the other hundred and one diseases so, naturally, fowls did not die of them.

The War saw the end of the "practical men". Blotted out? No. They just hadn't the brains to survive. "Veterinary" joined "Science" colleges passed from circus rings to halls of learning and the younger generation could now fathom nature's secrets for the good of animal kind. This gave the fowl some chance, gave the research workers theirs, and ultimately gave the N.V.M.A. courage to produce its little book instead of passing resolutions.

The little book has many imperfections; they could scarcely have been avoided. Tradition dies slowly and the authors dare not frighten nervous colleagues. The time-honoured subjects are trotted out—T.B., cholera, B.W.D., typhoid, coccidiosis (still with the old errors), verminosis and the rest. The most important ones—oncology, nutrition, and housing—have been curtly dismissed in the grand old manner.

But don't let us be dismayed. The plan of campaign is subtle. The book is a wad of loose sheets, held together by string between two bits of cardboard. Surreptitiously new sheets will be inserted between these cardboard covers, and old redundant ones withdrawn.

Some fine morn the British practitioner will have a valuable aid to the diagnosis and control of avian diseases; even "coccidiosis" will be up-to-date. And he will not know how he got it.

Ex Brittannia semper aliquid novum.

It seems the poor little hen has achieved lasting fame.

J. D. W. A. C.

Realising so well the numerous pitfalls which beset the path of the beginner in veterinary practice and the disastrous consequences of any avoidable blunders during this critical period, Dr. P. Escande has thought fit to give his younger colleagues the benefit of his thirty years of experience.

His little book[4]) entitled "The First Steps of the Veterinarian—Advice and Clinical Observations" is replete with sound advice, common sense, and practical hints.

He starts by dealing with the human and social element—the relationship of the budding practitioner with his colleagues, with his clients, and with communal bodies. Then he goes on to deal very briefly with the most common ailments of domestic animals in his district, and their treatment. He does not claim to have written a treatise on medicine: he merely gives practical hints and suggestions to supplement or short-cut the theoretical knowledge which he assumes the beginner has acquired as a student. The author points out that the inexperienced are often confronted in textbooks by a bewildering mass of detail on diagnostics and choice of therapy, the best of which he may not be able to select at once. Dr. Escande therefore indicates only the treatment which in his hands has given the best results, and mentions outstanding symptoms which are helpful in rapid diagnosis and prognosis. He stresses the importance of the latter and rightly says that the owner is more concerned with the veterinarian's ability to give a correct prognosis than diagnosis.

A. D. T.

4) *Les Premiers Pas du Vétérinaire*, by Pierre Escande. 1937. Pp. 120. Vigot Frères, Paris.

First Conviction under Act 16 of 1933.

On the 27th August, 1937, G. H. Potts, of 23, Nelson Road, Booysens, was found guilty in the Magistrate's Court, Johannesburg, and fined £2 for contravening Section 17 (1) of Act 16 of 1933. This subsection reads as follows:—

> Any person not registered under this Act as a veterinarian who pretends, or by any means whatever holds himself out to be a veterinarian (whether or not purporting to be so registered) or uses the name of veterinarian or veterinary surgeon or any name, title, description or symbol indicating or calculated to lead persons to infer that he possesses a degree, diploma or other qualification as a veterinarian or veterinary surgeon or in veterinary science or that he is registered as a veterinarian under this Act, shall be guilty of an offence and liable on conviction to a fine not exceeding fifty pounds.

The accused, in a civil case in which he was the plaintiff, had stated under oath that he was "a veterinary surgeon, M.R.C.V.S." and that he had been qualified for 26 years.

THE ASSOCIATION.

Minutes of Council Meeting held at Polley's Hotel, Pretoria, on 2nd September.

Present: C. J. van Heerden (Chair), P. J. du Toit, H. O. Monnig, A. C. Kilpatrick, A. D. Thomas, G. v. d. Wath, and S. W. van Rensburg (Secretary).

The Secretary read a telegram from Mr. Amos expressing regret for his inability to attend, and apologised on behalf of Dr. Curson.

(1) Minutes of meeting held on 24th March, 1937, were taken as read and were confirmed.

(2) **Arising from these minutes:**

(*a*) *Scale of Charges.*—The Secretary reported that upon receipt of the desired information from the N.V.M.A., the Status Committee had drawn up a scale of charges which would be submitted to members engaged in private practice with a request that they submit their views thereon.

(*b*) *Expert witness fees.*—The Status Committee found that there appeared to be a lack of uniformity in the fees paid to veterinarians. It was therefore necessary to investigate the question further.

(c) *Complaint: Dr. K. Schulz.*—The Secretary submitted a letter dated 22nd June, 1937, from this member regarding his claim for out of pocket expenses in connection with his trip to Europe for treatment.

After a full discussion a committee consisting of the Chairman and Secretary was appointed to ascertain exactly what agreement had been arrived at between the Department and Dr. Schulz before the trip was undertaken. Council to decide subsequently whether legal advice should be obtained or not.

(3) **Revision of Constitution.** The Secretary submitted the report of the sub-committee appointed to revise the Constitution. It was decided to place the suggested alterations on the agenda for the General Meeting.

(4) **Municipal Appointment—Complaint.** The Secretary read a letter dated 5th August from a member and appended newspaper cuttings regarding a certain municipal appointment. Exception was taken to the undue publicity given in the press to the qualifications of one candidate and the alleged failure of the Municipal Council to consider the claims of some of the other candidates.

This matter was discussed very fully. It was decided that although Council need not necessarily approve of the methods followed in this

case, it was not justified in interfering with the decision of the Municipal Council, and that it would be futile to attempt to obtain from the newspaper concerned the source of its information.

(5) **Members in arrear.** The Secretary submitted the names of four members who were over three years in arrear with their subscriptions. Satisfactory explanations were given in two cases. It was decided to send registered letters to the other two and if arrear subscriptions were not paid in the interval, to submit their names to the General Meeting.

(6) **Resignations.** Letters of resignation were received from Maj. Gen. E. R. C. Butler, Messrs. Hutchinson, G. T. Cannon, J. Forrest and A. McNae. Decided to recommend to the General Meeting that the first three be accepted with regret. The latter two will be requested to reconsider their decision.

(7) **New Members.** The following were proposed: J. A. Badenhorst, J. M. de Wet, H. F. T. Hellberg and T. H. Sandrock. Decided to recommend their acceptance to the General Meeting.

(8) **Correspondence.** The Secretary submitted the following letters :—

(*a*) From Dr. M. Zschokke dated 24-6-37 re proposed formation of a South West African branch of the Association, and his reply dated 5-7-37.

(*b*) From the Registrar, University College of North Wales dated 29-7-37 re opening of Dr. Griffith Evans Memorial Wing.

(*c*) From Chairman, Veterinary Board, dated 2-7-37 re complaint by Major Harber. Dr. du Toit informed Council that this matter has now been settled in a manner satisfactory both to the Board and to Major Harber.

(*d*) From Mr. J. H. R. Bisschop dated 2-9-37 re lack of organisation in the S.A.V.C. Decided that Dr. du Toit should first interview the Adjutant-General before further action is taken.

(9) **General:**

(*a*) *Newspaper Article.*—The Secretary read a letter dated 22nd August, 1937, from a member, enclosing a newspaper cutting and asking whether such an article would constitute an infringement of any of the regulations framed under Act 16 of 1933. Council was of the opinion that, since neither the names of the veterinarians nor their addresses were given in the article no offence had been committed.

(*b*) *S.P.C.A.*—Dr. du Toit reported on an interview he had had with the Chairman of the S.P.C.A., Johannesburg, re veterinary attendance at the latter's clinic.

(c) *Book Fund.*—Dr. Thomas reported that the sum of approximately £100 had accrued from the profits of the Book Fund. He suggested that this be put into a special fund in order to create prizes for the advancement of veterinary science. Decided that Dr. Thomas draw up a scheme for consideration by Council at its next meeting.

(*d*) *Binding.*—£10 voted for binding purposes.

(*e*) *Prosecution.*—Mr. Kirkpatrick reported the first successful prosecution under Act 16 of 1933. Decided to get particulars of this case for publication in the Journal.

(*f*) *Auditor.*—On Mr. v. d. Wath's proposal Mr. J. L. v. Wyk was appointed auditor in succession to Mr. C. O. Wadner.

The Meeting closed at 11 p.m.

(Sgd.) *S. W. J. VAN RENSBURG,*
HON. SEC.-TREAS., S.A.V.M.A.

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