VOLUME XIV. NUMBER 2.

JUNE, 1943.

THE JOURNAL OF THE SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION.

Edited by E. M. ROBINSON

for the

EDITORIAL COMMITTEE:

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PRINTERS:
CAXTON PRINTING WORKS (PROP, LTD
PRETORIA — 5720.

CONTENTS.

The Journal of the S.A.V.M.A. is owned and published by the S.A. Vet. Med. Assn., of which it is the official organ. It appears quarterly and is devoted to matters of veterinary importance generally.

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SOME ASPECTS OF SEX-PHYSIOLOGY AND THEIR RELATION TO FERTILITY OF ANIMALS — A REVIEW.*

JOHN QUINLAN, N. C. STARKE and H. P. STEYN.
Onderstepoort.

This paper is not a general review of recent publications on the sex physiology of animals. The discussion is confined to researches which have been completed or which are in progress at Onderstepoort. However, a few relevant references are made to works published elsewhere (by workers outside South Africa).

The ever increasing incidence of infertility and sterility of animals in this country suggested the necessity for a detailed study of its aetiology . Consequently, in 1926, one of us (Q) approached the late Director of Veterinary Services, Sir Arnold Theiler, who immediately provided facilities to proceed with the study. One of us (Q) had been interested in this question for several years and had read the literature then available on human and animal sterility. The majority of these works pointed to diseases of the female as the most culpable aetiological factor; infertility of the male had not then been carefully studied. Following the indications given in works on human gynaecology and the excellent publications of Williams on the diseases of the genital organs of animals, our first researches were confined to the pathology of the female genitalia. That study included observations on the cause, clinical indications, macroscopic and microscopic pathology, and the treatment of bovine sterility; the cytology and pathology of the ductless glands of sterile females were also included.

These studies, published in 1929, threw light on much difficulty in relation to the causes of infertility but there remained a hiatus which had to be filled if our knowledge was to be complete. The filling in of this hiatus has taken 12 years and it is now nearly completed. The great complexity of the problem was visualised only as the study progressed.

The results of our first researches showed that there were factors, other than those associated with genital pathology, concerned in infertility. An analysis of the facts available showed that our knowledge of sex-physiology was somewhat incomplete; there were so many physiological questions one was unable to answer, and it became necessary to study the sex-physiology of the male and female before the study of infertility could make further progress.

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* Paper read at the Annual meeting of the South African Veterinary Medical Association during September, 1942.

Our researches on sex-physiology were begun in 1929, and have been continued without intermission since then. Our first publication in 1930, concerned with the sheep, awakened interest in this most interesting study in other countries and a comparison, under varying environmental conditions, has been made possible.

The results have been most valuable in explaining aetiological factors, associated with infertility, hitherto not understood. Since some of the findings are of great interest to everyone concerned with animal production, it is considered that a discussion of certain of these researches might be of interest to a meeting such as this. You will realise, however, how cursory the review must be in the time available.

Reports of all the work completed have been published in the *Onderstepoort Journal,* the *Journal of Agricultural Science* and the *Journal of the South African Veterinary Medical Association,* and, no doubt, have been read by those interested. Consequently reference will be made only to the general scope of the study, with some more detail in connection with certain recent findings.

Articles on the following aspects of sterility and sex-physiology have been published or are nearing completion:—

(1) Sterility of cows in South Africa.
(2) The corpus luteum menstruationis and verum of cattle and sheep.
(3) The duration of motility of the ovine spermatozoon in the different divisions of the female genitalia.
(4) The longevity of the ovine spermatozoon in vitro.
(5) The fertilisable life of the ovine ovum following ovulation.
(6) The influence of vasectomy on sheep.
(7) The H-ion concentration of the vaginal secretion of the sheep during oestrus, dioestrus and pregnancy.
(8) The H-ion concentration of ovine semen.
(9) The influence of Prolan and Provetan on ewes:
    (a) on anoestrus,
    (b) on fertility.
(10) The nature of oestrus following a period of sexual inactivity.
(11) The influence of dry rations, lack of exercise and lack of sunlight on fertility of cattle.
*(12) The influence of dry rations, lack of exercise, lack of sunlight and delayed breeding on the fertility of cattle.
*(13) The influence of environment and nutrition on sexual maturity, oestrus and the ovarian cycle of cattle.
*(14) The scrotal and intratesticular temperature of the bull and ram and their relation to skin, body and air temperature.
*(15) The sperm picture of rams of different breeds as an indication of their fertility.

*(16) The fate of morphologically changed spermatozoa in the genital tract of the ewe.

(17) The influence of the vaginal temperature at the time of coitus on conception.

(18) An attempt to fertilise caprine ewes with ovine rams.

*(19) The influence of the vaginal reaction at the time of coitus on the sex of the offspring.

(20) Artificial insemination of ewes with fresh and stored semen.

These studies showed that the duration of life of spermatozoa varied in the different divisions of the ovine female genitalia, where they were conveyed after exposure of the uterus through a laparotomy opening. Spermatozoa were found to remain motile in the vagina up to 20 hours, in the cervix up to 48 hours, in the pars indivisa, uterine cornua and the Fallopian tubes up to 12 hours. There was no indication of a sperm swarm moving forward to attack the available ovum. Rather did it appear that the cervix acted as a sperm reservoir from which there was a slow issue of actively vital spermatozoa while the descent of the ovum into the Fallopian tube was being awaited. In view of the short time, approximately 6 hours following ovulation, during which an ovum appears capable of being fertilised, this action of the cervix would appear to be the only one compatible with a physiological explanation. Ovulation in the merino, under the environmental conditions prevailing in the Karroo, takes place about the 36th to the 40th hour following the onset of oestrus, and copulation later than the 42nd hour resulted in impregnation of only 10% of cases. In fact coitus at the 33rd hour already showed reduced fertility.

The earliest time at which spermatozoa were found in the Fallopian tubes, following normal copulation, was 6 hours. One of us (N.C.S.) recently found spermatozoa in the tubes 5 hours after copulation. High fertility followed copulation up to the 30th hour of oestrus.

Therefore it became apparent that there was an optimum time for copulation in relation to oestrus and ovulation. There was also a sterile period following the 42nd hour, when the ovum was incapable of being fertilized although some females would still allow copulation at the time.

Under the environmental conditions prevailing at Armoedsvlakte it appears that the duration of oestrus, as distinct from pro- and met-oestrus, is shorter in beef cattle than in milking breeds. The indigenous Afrikaner heifers showed an average duration of 7.88 hours, the Sussex 9 hours, the Friesland 11.67 hours and the Red Poll 14.0 hours. Ovulation takes place around the 24th to the 30th hour following the onset of oestrus. This duration of oestrus is shorter than the standard range recorded in other countries. However, it is much longer than that recorded for the Zebu in Kenya by Anderson (1936).

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* Indicates incomplete or unpublished work.

In the case of animals with a short oestrus period, the time of ovulation is interesting. It occurs subsequent to oestrus thereby allowing the spermatozoa sufficient time to reach the Fallopian tubes while the ovum is still capable of being fertilized. The mare with a long oestrous period ovulates late during oestrus and mating to be successful must be timed accordingly. These findings would conform to the observations of Knaus (1931) in relation to ovulation and conception in humans.

The importance of these observations cannot be over-stressed in animal breeding, where coitus is controlled in stud animals. Mating and insemination during the optimum period have improved fertility enormously, while mating outside that period, indicated by physiological researches as the "fertile period," has invariably been sterile.

The literature is rich in references to light, exercise and nutrition as prerequisites for high fertility. It may be so in certain animals, but our researches have shown that cattle remain sexually normal when placed for 13 years in an environment enforcing lack of exercise, lack of sunlight and dry rations. Some of the experimental animals still survive at an age of 14½ years and have had 12 calves. However, the calves of these cows, when retained in a similar environment, showed delayed sexual maturity, although oestrus and the ovarian cycle were normal in rhythm when maturity was reached.

The result of this observation was rather puzzling, as it was quite unexpected. Therefore delayed breeding was super-imposed on the existing environment and immediately produced infertility. Heifers bred at 5 years were infertile, and even at 4 years difficult conception was experienced. Those mated at three and three-and-a-half years bred normally. The majority of the infertile heifers showed ovarian cysts. The cause of this failure to breed is attributed to the atrophy of disuse, as the cytological picture showed atrophy of the uterine mucosa concurrent with cystic degeneration of the Graafian follicle. Such an occurence is probably attributable to endocrine disturbances such as occurs in women during middle age.

An interesting observation was made recently by Gunn. He established infertility by feeding rations deficient in vitamin A, and was able to cure the condition by supplying the deficiency, but he was also able to restore fertility by the use of injections of pregnant mare serum.

A most interesting phenomenon in animals that experience seasonal sexual activity is the change over which occurs when they are transferred from the Northern to the Southern Hemisphere. There is an almost immediate reversion of the sexual season, and animals with a short gestational period, i.e. 147 to 152 days, such as the sheep, will sometimes breed twice in the first year, after transfer, whereas normally they would breed only once. Afterwards they adopt the sexual habits of acclimatised sheep. A similar reversion is seen in mares. The cause of this change over is not understood, but it is apparently an environmental exteroceptive stimulus to the pituitary gland.

Anoestrus in animals with a seasonal ovarian cycle, is due to a low level output of the gonadotrophic and follicular hormones. It is almost entirely independent of nutrition, provided, of course, that there is not actual malnutrition. It is apparently Nature's method of controlling the animal population. A somewhat similar phenomenon has been described by Llewellyn (1932) as occurring in the Eskimo, both male and female, during winter. At the end of the anoestrous period and commencement of the breeding season the return to sexual activity is, in many cases a gradual process. The high level secretion of the gonadotrophic and the follicular hormone, necessary to produce full oestrus and ovulation, is a gradual occurence. Further, individual females react differently, some with delayed response, to the exteroceptive stimuli which initiate sexual activity after a period of quiescence.

The practical significance of oestrus without ovulation will be realised by veterinarians connected with thoroughbred studs. Failure to ovulate is the cause of prolonged oestrus periods, up to 15 to 30 days. Fortunately hormone treatment usually precipitates ovulation in such cases.

It is now possible to cause an immediate return to sexual activity, during the anoestrus period, by the use of extract of pituitary and pregnant mare serum. Use is made of this treatment to overcome seasonal anaphrodisia and that associated with corpus luteum persistens, a cause of infertility frequently encountered in animals. The use of these hormones, as a method of combating breeding difficulties, is well beyond the experimental stage, but further work is needed before exact dosage is established. Unless the dosage is correct, oestrus without ovulation or ovulation without oestrus may be experienced. There are indications that the dosage does not depend on the age and size of the animal, rather does it depend on the degree of activity of the pituitary of the individual at the given period. There is, as yet, no method of determining the pituitary output. Sexual activity may actually be prevented by an overdose; so that the greatest care must be exercised in prescribing hormonal treatment.

Attention has recently been drawn to the possibility of the reaction of the vagina at the time of coitus determining the sex of the offspring. The work of Warren (1940) and Durfee gives, in considerable detail, the results of experiments which, to the uninquisitive (or uncritical) mind, would appear conclusive. Extensive observations have however been carried out on rabbits and sheep with apparently negative results. Observations on the pH of the vaginal secretion of sheep gave a mean value of 6.6; but a vaginal reaction above 7, that is on the alkaline side, although reducing the percentage fertility, did not seriously affect pregnancy. There are indications that oestrus and pregnancy have a tendency to increase acidity.

A concurrent observation on ewes, in which the vaginal temperature was raised by forced exercise, indicated that within a range of 100.2°F to

106.2°F at the time of coitus, there was no significant interference with conception. The duration of the artificially created high temperature was, of course, short, and there appears to be no method by which such elevated temperature can be maintained without interference with general health. However, owing to the extreme sensitiveness of spermatozoa to slight elevation of temperature, there is little doubt that their fertilising power would be impaired by a prolonged stay in such environment.

Until recently, studies on infertility in humans and domestic animals had given little consideration to the male as a possible culpable factor, but the works of Moench (1931) and Laegerlöf (1934) have greatly increased our knowledge of male infertility. During our observations it soon became evident that a large percentage of males, although apparently clinically sound and copulating normally, were temporarily or permanently sterile. This necessitated an inclusion of the male in our study. Observations were begun on the semen: the volume of the ejaculate, its physical characters, colour, density, reaction, motility of the spermatozoa, longevity *in vitro,* sperm count, and morphology were considered.

As the work progressed it was found that these observations would not complete the study, and it became necessary to include different breeds of rams on the same level of nutrition during all seasons of the year under similar environmental conditions. It also appeared probable that changes in air temperature may play an important role in infertility and a study of the scrotal and intratesticular temperature was included.

The results of these observations have recently been published or are reaching conclusion. They have shown that the exogenous and indigenous breeds of sheep react differently to high environmental temperatures in so far as certain physiological functions are concerned.

The exogenous breeds are less resistant to trying climatic influences than are the indigenous breeds. However, it is, remarkable how efficiently the scrotum and the dartos muscle act as a temperature regulating mechanism for the testicle. It has been shown that the temperature of the scrotal skin of the bull is 2°C to 4°C lower than the skin of the body, and that the testicle is similarly lower than body-temperature within a wide range of air temperature from 4°C to 40°C (39°F to 104°F.). The scrotal-skin and testicular temperatures remain within a very limited range of constancy in normal animals. Testicles placed in such a position as to approximate body-temperature rapidly become aspermatogenic and remain so until normal topography is restored. This is the cause of the cellular changes in abdominally retained testicles, in which the spermatogonia and Sertoli cells are not recognisable. The interstitial cells remain unaffected. The efforts made by the scrotum and dartos to keep the testicle at normal temperature are extraordinary. One of us (Q) has displaced the testicle of the bull and

placed it in contact with the abdominal wall, away from the influence of the scrotum. The temperature of the displaced testicle almost reached body-temperature, i.e. between 2.5°C and 3°C, above its normal, but the scrotum sweated profusely *(perspiratio insensibilis)* and, through evaporation from its surface, reached the low level of 6°C below normal, and 8.7°C lower than intratesticular temperature.

In making these latter observations assistance was rendered by Miss G. Riemerschmid and Mr. J. Elder, Section of Solar Radiation Survey, who perfected a thermo-couple for skin readings and a thermo-needle for intratesticular readings. These instruments are extraordinarily sensitive to changes of temperature and a great improvement on instruments previously used by them.

The constancy of the testicular temperature at which normal spermatogenesis is possible indicated how wrong it was to exclude the male from our first observations in a subtropical country. Gunn (1942) has recently shown a marked change in the spermatozoal picture of rams during intensely hot spells.

The spermatozoa of animals are extraordinarily sensitive to heat. Their survival at room temperature under summer conditions is a matter of hours; while motility has been observed up to 100 days in ovine spermatozoa maintainde between 2°C and 4°C: longevity between 50 to 82 days is nothing exceptional. It can be taken that longevity is an indication of the vitality of spermatozoa, but in itself it is no indication of fertility; other physical factors must be considered as well.

The pH of the ejaculate is an important factor in determining fertility. Normal ovine semen shows a reaction of 6.3 to 5.8., but there is a rapidly increasing acid reaction during storage, unless the motility of the spermatozoa is reduced by cooling. When testing fertility by the artificial insemination of ewes, the most successful results were obtained with semen within a pH range of 5.0 to 6.5. These observations would be compatible with the results obtained on the H-ion concentration of the vaginal secretion of ewes in which increased acidity developed during oestrus.

In view of these results it appears that sufficient attention may not have been paid to the reaction of semen as an aetiological factor in infertility.

The activity of spermatozoa is not always an indication of their capacity to impregnate, although active motility is necessary for fertilisation.

The density of semen is also important in judging fertility. The variation in density has been extraordinarily wide, ranging from 6,900,000 to 50,000 per c.mm. The number of spermatozoa in the ejaculate of the same ram may vary considerably within a period of a week. Changes in environmental conditions appear to influence spermatogenesis very rapidly. We have observed fertile rams, transferred from Ermelo district to the Pretoria District during cold rainy

weather, to show infertility within a week. The lowest level of spermatozoa per c.mm. compatible with fertility has not yet been ascertained, but the study will be completed soon. Gunn estimates a density of 50,000 per c.mm. necessary for fertility.

The morphology of the stained sperms is of the greatest importance, and without a morphological examination it is impossible to gauge the fertilising capacity of semen. The greatest care is necessary in the preparation and staining of semen smears, as certain changes are easily produced in preparation which may influence the abnormal count considerably. For routine examination the staining process recommended by Williams, has been found most satisfactory.

The important morphological changes seen in abnormal spermatozoa are recognised without difficulty, but the importance of each change is not yet entirely recognised. It may be taken as a general rule that 240 to 250 abnormal spermatozoa per 1,000 indicates sterility and 160 to 180 per 1,000 indifferent fertility.

Scientific breeding and artificial insemination have indicated the necessity for submitting males to a frequent test for fertility. One can assess, with fair reliability, the degree of fertility of a male after a few detailed examinations of his semen. The economic loss from breeding from sterile and lowly fertile males can be entirely obviated by such examination. Semen for examination can be obtained after normal copulation, but, in the case of the ram, it is better to use electric ejaculation or the artificial vagina, by which methods uncontaminated material is obtained. These latter two methods are those used by us in obtaining semen for artificial insemination of ewes. They have given satisfactory fertility with semen stored up to 12 hours and pregnancy has been obtained with semen stored up to 72 hours.

Our methods of producing pregnancy by artificial insemination in sheep are notably artificial. Semen is obtained as described above and is transmitted into the cervix of the oestrus female, in diluted form, at the optimum pH. Several females can be inseminated from the same ejaculate. Oestrous ewes are picked out from the flock by vasectomised teasers, and semen can be stored until a convenient time for insemination. Further, there are indications that the operation will become more artificial within a short time; that is, when the correct dosage of the hormones governing ovarian activity has been ascertained. Then, oestrus in the female, a phenomenon which must now be awaited and which occurs at intervals throughout a certain season of the year, could be produced as desired.

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MOVEMENTS OF OFFICERS.

The following transfers of veterinary officers in the government service have taken place since the last journal was published.

W. D. Malherbe	from	Potchefstroom	to	Onderstepoort.
C. J Erasmus	„	Bethlehem	„	Potchefstroom.
J. A. Badenhorst	„	Flagstaff	„	Bethlehem.
P. H. Brown	„	Bedford	„	Flagstaff.
C. W. A. Belonje	„	Kokstad	„	Aliwal North.
J. M. de Wet	„	Upington	„	Kokstad.
P. P. Hugo	„	Allerton	„	Upington.
W. C. Viljoen	„	Umtata	„	East London.
G. L. Muller	„	Malmesbury	„	Worcester.

RESIGNATIONS.

Mr. F. J. D. Hempstead, government veterinary officer, East London, has resigned from the government service to take up private practice with Mr. J. G. Boswell in Johannesburg.

Mr. J. H. Schoeman, government veterinary officer at Vryheid has resigned to take up private practice with Dr. J. G. Bekker in Johannesburg.

Mr. J. G. van der Wath has resigned from his post as lecturer in physiology at Onderstepoort to take up farming in the Ermelo district.

A SKIN CONDITION IN CATTLE PROBABLY ASSOCIATED WITH PHOTOSENSITISATION.

J. I. QUIN, Onderstepoort and J. L. DORÊ, Dundee.

A condition, as yet undescribed in South Africa and of unknown origin has recently been investigated in the Natal Highlands (Glencoe-Dundee-Utrecht areas). It appears to be fairly widespread as cases were seen on six out of seven farms visited. Moreover, it has also been reported from several neighbouring districts. Its incidence, however, is peculiar in that the cases thus far reported have all been of isolated, sporadic nature and without any tendency to occur as outbreaks in the usual sense of the word. According to information gathered, it appears to be confined mainly to the summer months, although individual cases may be encountered at other times of the year. Of the cases recently investigated, all were found to be amongst Friesland females, ranging from young heifers to old cows. One case only was seen in a grade Ayrshire cow and one case in a Friesland heifer calf about 3 months old. The condition is usually encountered in animals grazing on the open grassveld. No specific poisonous plant or other form of poisoning has as yet been discovered on the veld to account for these cases.

Symptoms.

According to owners, the first symptoms usually noted are those of abdominal colic accompanied however by diarrhoea. The animal loses appetite and this is followed by a rapid loss of condition. All unpigmented white patches of skin appear swollen and tender, those along the back being more severely affected than the white areas on the ventral aspects of the body. White skin on the legs and especially unpigmented coronets and hoofs may be markedly affected, thereby causing tenderness in the feet, and a stiff gait. (Fig. 1).

FIG. 1.

The swelling of the skin is soon followed by hardening and subsequent cracking of the superficial layers accompanied by the oozing of yellow fluid. In time there follows extensive sloughing of necrosed skin leaving a tender surface which bleeds readily (Fig. 1 and 2).

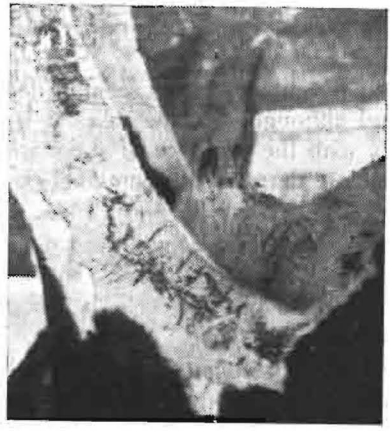

FIG. 2.

Afflicted animals are definitely sensitive to sunlight as shown by their eagerness to move into shade whenever this is available. Depending on the degree of skin damage and the duration of the photosensitive state, the progress of recovery may vary. Thus in the milder cases there is little or no scarring of affected areas which soon become covered with a growth of normal white hair. In more severe cases on the other hand scarring of unpigmented areas may be intense and at times accompanied by hyperkeratinization and the formation of horny scales. Such animals may be grossly disfigured, as the hairless parts remain permanently hairless. In some cases the hoofs are shed. All lesions described above are limited to the white areas and are sharply demarcated from pigmented skin, which appears to be completely resistant.

During the acute stage there may be some evidence of jaundice, which is noticeable especially on the skin of the udder and teats, if unpigmented. The milk flow of affected cows is reported to be definitely lowered, even during subsequent lactations. The condition is rarely if ever fatal, thus affording scant opportunity for post-mortem examination.

As little is known concerning the distribution, primary causation and pathogenesis, any further information concerning this condition in the different areas of the Union will be gratefully received by the Director of Veterinary Services, Onderstepoort.

FILARIASIS IN DOGS IN UGANDA.

J. CARMICHAEL and F. R. BELL,
Veterinary Research Laboratory, Entebbe.

Microfilaria are not infrequently seen in blood examinations of dogs in Uganda and although in some cases it is difficult to associate a symptom complex with the presence of these worms, there are other cases where an obstinate dermatosis is undoubtedly the result of filaria infection.

The clinical cases which have come to our notice have all been large dogs — Alsatians, ridge-back lion-dogs and similar breeds, and the presence of what are described as "veld sores" by the owner is the usual reason for seeking advice.

The lesions consist of swollen, moist, desquamated areas of skin which later become necrotic, with or without sinus formation.

They are extremely resistant to any form of local treatment and so far attempts to eliminate the microfilaria from the blood by chemotherapeutic measures have failed completely.

The following case seen recently will illustrate the type of clinical condition one is faced with.

Subject — Alsatian dog about 6 years old.

History — The first skin lesion was noticed in November, 1941, and the condition became progressively worse until January, 1942, when he was presented for treatment.

Clinical Examination — The dog was in good condition and temperature, pulse, respiration, etc., were normal. Multiple dermatitic lesions were present on the legs, feet, and head where the hair was short. They consisted of typical desquamated swollen moist areas with erosion of the epidermis in places, from which numerous small sinuses discharged a blood-stained serous fluid (see Fig.). Early lesions are at first localised swellings about the size of a shilling, over which the hair stands erect. Later there is an exudation of lymph with crust formation, squamous thickening and sinus formation. The mucous membranes were normal or slightly injected. This picture on previous experience immediately suggested filaria infection which was confirmed by blood examination.

A more detailed blood examination revealed the following:—
Hb. 51% (Sahli-Hellige) or 7.4 grms. per 100 c.c.
R.B.C. 3,100,000 per c.m.m.
W.B.C. 8,600 per c.m.m.
Neutrophils 88%.

Lymphocytes 7%.
Monocytes 2%.
Eosinophils 3%.

Neutrophils mainly mature, but a few band forms and an occasional metamyelocyte.

Course of condition. — The dog had been under observation for three months without any amelioration of the condition. The lesions are insensitive and show a definite cycle from their commencement to healing, with the result that lesions in all stages are present at the same time. Lesions may coalesce and leave a hairless scar about three inches square.

FIG. 1.

Continued examination of the exudate and the depth of the sinuses has not revealed the presence of either adult filariae or microfilariae.

The dog, which was used to a very active life, has maintained its condition fairly well as it has been, of necessity, kept in a kennel.

Regular examination of blood smears showed the continued presence of microfilariae. The blood was consistently negative for protozoan parasites.

The Hb. and R.B.C. after 3 months improved to 72% (10.4 grm. per 100 c.c.) and 5,250,000 respectively. Cultures from the lesions yielded pure growths of *Staph. albus.*

The unsheathed microfilariae were numerous in the blood and gave an average measurement of 240μ long and 4.5μ wide.

Treatment — Local dressing consisted of eusol, iodine, acriflavine 1/100, dusting powders, mercurochrome 1/500, and all were used without success.

Fouadin, as used by *Underwood and Wright (1934)* was unobtainable so antimosan was used. Two courses of antimosan in five doses

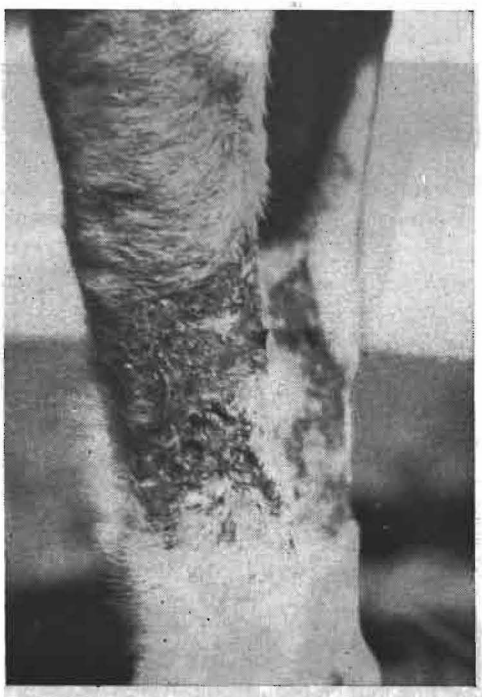

FIG. 2.

of 3 c.c. of a 5% solution at a two day interval were given intravenously without eliminating the microfilaria or causing any improvement. A course of soluseptasine, 2 c.c. twice daily subcuntaneously for five days, was also without effect.

Novarsenobillon was also given. An autogenous vaccine prepared from the Staph. albus isolated from the lesion also failed to cause any improvement.

The dog was eventually destroyed. A careful search failed to reveal any adult worms.

Experimental. — Subinoculations of blood into native dogs set up a blood infection without causing any symptoms. A clean dog kennelled with an infected one contracted infection, possibly through mosquitoes or other insects.

Discussion. — This condition simulates filarial dermatosis described in other species by many authors. The lesions are like those described by Bubberman and Kraneveld (1933) in the condition known as "cascado" in the Dutch East Indies due to the presence of the adult worms of *Stephanofilaria dedoesi.* The condition described above differs in the fact that as yet no adult worms have been found in the epithelial layers and also in that there is no pruritus sufficient to cause aggravation by rubbing. It would appear that the lesions are produced in a similar way, that is, the presence of great numbers of microfilaria in the sub-epithelial capillaries cause *embolic* necrosis.

Chitwood (1934) described a similar condition in the United States, where the lesions are produced on the underside of the abdomen in cattle due to *S. stilesi* infection. Kemper (1938) has described a similar filarial dermatosis of adult female sheep in New Mexico due to infection with Elaeophora schneideri.

Filaria from yet another genus of the family Filariidae, the genus Parafilaria, *P, multipapillosa* and *P. bovicola* produce dermatitis lesions of a similar type in equines and bovines.

The common filaria of dogs, however, belong to the genus Dirofilaria and the most common parasite, *D. immitis* does not occur in Africa. This parasite does not usually produce dermatosis although Vaills (1934), reporting on four cases in dogs, describes a cutaneous syndrome, where numerous microfilariae in the capillaries of the skin cause an ulcerous dermatitis. The larvae isolated do agree in some respects, especially anatomically, with *D. immitis* but it is much shorter. The length of the larvae approaches more closely that of *Acanthocheilonema drancunculoides* which has been found in the peritoneal cavity of the dog in Africa.

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Note.

For the sake of completeness it might be added that *Stephanofilaria haeli* Buchley, 1937, causes lesions on the legs of cattle in the Malay

Penisula and S. assamensis Pande, 1936, causes "hump sore" of cattle in Assam.

The microfilariae in smears submitted by the authors correspond very closely to microfilariae which are fairly common in dogs at Pretoria. The latter sometimes occur in large numbers, but no symptoms or lesions have ever been associated with them. Careful autopsy and a search extending over several hours repeatedly failed to reveal an adult worm anywhere in the body. These microfilariae correspond fairly closely in their morphology to *Mf. immitis,* but are somewhat shorter (*Mf. immitis* measures about 218 — 329μ usually about 260μ).

It is possible that the microfilariae found in the cases of ulcerative dermatitis described above are not identical with those found in dogs at Pretoria, but it would be advisable to study the pathology of the dermatitis further before a definite conclusion is drawn as to its aetiology.

Subinoculation of microfilariae would not produce adult worms, as the microfilariae have to develop in an intermediate host before development in the final host can occur. Such inoculated microfilariae can, however live a long time in the new host, as has been demonstrated by Fülleborn and others.

H. O. MONNIG,
Onderstepoort.

AN OUTBREAK OF ANTHRAX AMONGST KOEDOES.

S. W. DE VILLIERS, DE AAR.

What may be described as an outbreak of very rare occurence, namely, that of anthrax amongst koedoes, took place in the Karibib District, S.W.A., during the month of June, 1942.

This article is a preliminary report, as investigations are still proceeding and will no doubt be further reported upon by my successor, Dr. Holtz.

On the 15th July, 1942, I had occasion to do Bull-Inspection work (Cattle improvement) on the farm Lievenberg, Karibib District. The owner, Mr. Redecker, notified me that koedoes were dying, not only on his farm, but also on adjoining farms. Towards sunset of the same day a native, acting as herd-boy, informed us that a dead koedoe was lying near the road leading to one of the cattle outposts about five miles away. We proceeded to the spot indicated and found a koedoe bull approximately 5 years old, judging by the teeth and growth of horn.

By the time we arrived it was too late to hold a post-mortem but the following observations were made:— There was no blood from the nostrils, the mucous membranes of the eyes were slightly redder than usual but not the bluish-purplish-red associated with HCN poisoning, there were no signs of external injuries, nor were there any swellings on any part of the body, the abdomen was slightly distended, and blood from a cut in the ear appeared to be dark in colour with no signs of coagulating.

A blood smear was taken and examined on my return to Head Quarters, Omaruru. To my surprise I found that it showed anthrax. Being desirous of having my diagnosis confirmed, I forwarded the smear to the Senior Veterinary Surgeon, Windhoek, who was able to do so. In his report the S.V.S. stated. " A few putrefactive bacteria were also found and it was observed that the red cells were dotted with granules and in the stage of breaking up." The Director of Veterinary Services, Onderstepoort, also examined the smear and found it to be positive for anthrax.

There is one very peculiar feature about the smear. Whereas in cattle an anthrax smear usually shows numerous bacilli in any particular field, the bacilli in the smear from the koedoe were so sparsely dotted here and there that they could easily have been missed.

On the 22nd July another smear from a dead koedoe was submitted by the same owner. This smear also proved to be positive for anthrax. Numerous putrefactive organisms were present and some spores.

From investigations made, the following points must be mentioned:

(1) The first deaths amongst koedoes were observed during the month of June, but there may have been cases earlier.

(2) Deaths have taken place on the following farms: Lievenberg, Westfalenhof, Okamitundu and Namatsaus.

(3) On these farms so far dead koedoe bulls only have been found.

(4) The anthrax is apparently confined to the koedoes only, as no bovines, equines, ovines or caprines have succumbed to the disease.

(5) Since koedoes graze "high" as a rule, instead of "low," the causative agent is in all probability not ingested.

(6) Good rains have fallen and the veld, comprising trees, bush, shrubs and grass, was excellent for grazing purposes. All the dead koedoes found were in good condition.

(7) As far as is known koedoes are not bone-eaters.

(8) Infection by contaminated water cannot be ruled out, but other domesticated animals not having died, and having access to the same water, excludes this as a likely source of infection, unless it be that the particular strain affects koedoes only.

(9) The possibility of infection by some blood-sucking insect, such as the horse fly, must be taken into account, and I personally incline to this theory.

The owner of the farm, Lievenberg, has been supplied with bottles containing glycerin and has been requested to forward not only further blood smears of koedoes found dead but also portions of skin, etc.

The total destruction of all koedoes could be considered, but there is the danger of spreading the disease still further, owing to the dispersal of koedoes in all directions. It may be that the disease will have to run its course, but if it is found that other domesticated animals become infected as well, total destruction may be found to be the only solution.

In conclusion I may add that I have just received a report from the G.V.O. Omaruru, to the effect that cattle have died from anthrax on the farm Uitdraai. This farm is in the same vicinity of those mentioned above. As this is the first outbreak of anthrax in the Karibib District for over five years, it may be more than a mere coincidence. It would be interesting to note further developments in view of the fact that although koedoes have died in numbers on the four farms, no deaths amongst domesticated animals have taken place form anthrax on those farms for a period of approximately four months.

HISTORY OF THE S.A.V.C. 1910 - 1939.

(Continued from Vol. XIII. No. 4, December, 1942).

CHAPTER I.

GERMAN S.W. AFRICAN CAMPAIGN (1914 — 1915).

INTRODUCTION.

This campaign is of particular interest not only because it was the first in which the S.A.V.C. participated, but also because equines played an important role therein, both for transport and riding purposes. Smith (1916), whose report on the campaign is reproduced in Blenkinsop and Rainey's *History of the Great War: Veterinary Services* (1925), states that "the total animal establishment on mobilisation comprised 8,000 horses and mules," but the number eventually increased to 160,000 animals.

A summary giving the number of troops *in the field* on 15.3.1915, extracted from the *Official History of the Union of South Africa for the Great War* (1924), furnishes the following data:—

Force	*Artillery*	*Mounted*	*Infantry*	*Administrative*	*Total*
Northern	741	12,773	5,864	1,491	20,869
Central	575	3,858	5,583	855	10,871
Southern	117	8,438	——	631	9,168
Eastern	40	2,016	——	420	2,476
Total	1,473	27,085	11,447	3,397	43,402

Complicating hostilities were several factors, the most important being: that the campaign was interrupted owing to insurrection in the Union, the semi-arid nature of the country generally, a strip of desert up to 80 miles in width along the littoral, the need for transporting all supplies, both human and animal, to the territory, and the lack of wagons for transport purposes.

It will be necessary first to give a background of the movements of the various forces employed in the subjugation of the country before passing on to veterinary considerations. The former is based on data culled from the *Official History* (1924) referred to above or from Collyer's account of the campaign (1937), and the latter are chiefly gleaned from Smith's report (1926).

Strategic Considerations.

The campaign may be conveniently divided into the following phases :—

(i) *Preliminary*: The initial steps directed against the southern portion of German South-West Africa between the end of August, 1914 and the latter half of October, 1914;

(ii) *The Rebellion*: This lasted from the 9th October, 1914, to the end of January, 1915. During this period forces engaged under (i) were withdrawn to the Union; and

(iii) *Resumption and completion of hostilities*: This occupied the period between the end of December, 1914, and the date of surrender of the German forces, viz. 9.7.1915.

Part of the activities of the S.A.V.C. falls into the post-campaign period i.e., up to the end of 1915 and included work undertaken both in South-West Africa and the Union.

(i) Preliminary Operations.

These included the despatch of three forces designated "A," "B," and "C" particulars of which follow:—

"A" force under General Lukin, and comprising 2,420 ([14]) men of all ranks, was disembarked at Port Nolloth between 1st and 16th September. After a rail journey of 50—60 miles to Steinkop the troops proceeded by road north-east across desert country to occupy Ramans Drift and Houms Drift on the Orange River. Apart from long lines of communication, including sea, rail and road, Lukin's transport consisted of "indifferent wagons and donkey teams" (p. 31), so much so that on 22.9.1914 he wired D.H.Q., Pretoria, "that three weeks must elapse before sufficient supplies could be accumulated on the Orange River to enable an advance .... to commence" (p. 32).

Nevertheless an attempt was made to advance towards Warmbad with the result that the Union troops were badly defeated at Sandfontein, 24 miles north of the Orange River on 26.9.1914.

On the day of the action rations for the troops at Sandfontein "were 36 hours overdue" (p. 39) and reserve ammunition could not be sent forward owing to lack of transport.

Subsequently Lukin took up a position at Gudous Drift (approximately 10 miles west of Ramans Drift), but in view of the poor state of the horses of the 5th S.A.M.R. this regiment returned to Steinkop. Finally, as a result of the Rebellion Lukin left Gudous with his force

([14]) According to Collyer (1937) the units represented were:— 8th (Citizen) battery — 4 guns; 2nd and 4th (Permanent) batteries, each 4 guns; 10th Infantry (Witwatersrand Rifles); 1st — 5th regiments S. African M.R.; Ammunition column and 1 section Engineers. The **Official History** makes no reference to the 1st — 4th regiments S.A.M.R. Page numbers with no reference to author, imply Collyer.

for the Union on 23.10.1914, leaving behind the Witwatersrand Rifles, presumably disposed along the railway.

"B" force under Lt.-Col. Maritz and numbering 1,000 of all ranks (with no artillery) mobilised at Upington during September. It was soon manifest that Maritz was in league with the enemy. On 2.10.1914 he moved from Upington for Van Roois Vlei, where a week later he "disarmed and betrayed to the enemy the few officers and men of his force who had refused to join him" (Official History, p. 15). His subsequent movements form part and parcel of the Rebellion which affected not only the North-West Cape Province, but also the Orange Free State and Transvaal.

"C" force commanded by Col. Beves and totalling 1,824 men ([15]) was disembarked at Luderitzbucht on 18.9.1914 without opposition. It remained there until the resumption of the campaign in South-West Africa.

(ii) Rebellion.

It has been noted that on 9.10.1914 Lt.-Col. Maritz had disarmed loyal troops at Van Roois Vlei. On 15.9.1914 General Beyers, Commandant-General of the A.C.F., had resigned his appointment, along with Major Kemp, who was District Staff Officer at Potchefstroom, the important military centre. All three, with General Christian de Wet, were to play important parts in the Rebellion.

Beyers commenced his activities in the Rustenburg district, but on being attacked by General Botha on 27.10.1914 at Commissie Drift, he proceeded with his following by way of Koster to Vleeskraal in the Bloemhof district. On 2.11.1914 he left Vleeskraal travelling south, and crossing the Vaal River near Kingswood Station, he hoped to effect a junction with Conroy, who was in rebellion in the North-West Orange Free State. On 7.11.1914 Col. Lemmer, with Government troops, encountered Beyers at Verlatenskraal Drift near Hoopstad and inflicted heavy loss. Ten days later Col. Celliers repeated the blow, and as a result the rebels (Beyers and Conroy having joined forces) suffered severe loss, 487 being captured and 257 voluntarily surrendering. Beyers himself escaped and travelling east fled in the direction of Reitz. Later he doubled back and crossing the Bloemfontein-Johannesburg railway line near Ventersburg on 8.12.1914 was attacked by Field Cornet de Necker's force in the Hoopstad district. He again escaped and shortly afterwards was drowned in the Vaal River while attempting to cross it in flood.

Kemp, who had joined Beyers at Vleeskraal at the end of October 1914, proceeded westwards with his force to meet Maritz on the German border, travelling via Schweizer Reneke (3.11.1914) and Kuruman (8.11.1914). He attempted to cross the Orange River at Kheis Drift

([15]) The units represented were:— 7th (Citizen) battery — 6 guns; 1 squadron 5th M.R. (Imperial L.H.); 8th Infantry (Transvaal Scottish); 11th Infantry (Rand L.I.) and 1 section Engineers.

on 18.11.1914 but was prevented by loyal troops. Ultimately he effected a junction with Maritz at Schuit Drift on 20.12.1914, after being harrassed by Government forces *en route*.

A month later (24.1.1915) these leaders attacked Upington but were repulsed. As it was now clear that the insurrection had failed, the rebel leaders in the North-West Cape Province agreed on 30.1.1915 to unconditional surrender of their commandos. Kemp gave himself up on the night of 2.2.1915, but Maritz departed for German territory and continued to assist the enemy.

De Wet, who led another large rebel force, went into rebellion on 28.10.1914, when he assaulted the local magistrate at Vrede. About this period there was great activity by other leaders in the North-East Orange Free State, N. W. Serfontein M.L.A. being particularly energetic. De Wet was at Lindley on 3.11.1914 and occupied Winburg on 9.11.1914. General Botha, however, had concentrated forces in the district and an action took place at Hoenderkop on 12.11.1914. De Wet escaped with a small force and fled in the direction of Vryburg, crossing the Rhodesian railway on 27.11.1914. Five days later he was captured at Waterbury in the Kuruman district.

Minor local disturbances occurred in other districts, e.g. in the South-West Transvaal; but the rebels under Wolmarans and Claassen were defeated by Col. J. J. Alberts at Treurfontein station on 29.10.1914.

The policy of the Government was to concentrate strong mobile columns against the chief leaders and to leave the smaller fry to local commandos. Thrust and determination characterised Government action and as a result of the furious pace, exhaustion and abandonment of rebel horses was a marked feature of the conflict. Kemp's westerly flight of approximately 1,200 miles in semi-arid country was a striking example of commando mobility.

Twenty thousand of the thirty thousand Government troops engaged in the Rebellion were mounted burgers.

(iii) Resumption of Hostilities.

Rebellion having collapsed in the Transvaal and Orange Free State and Kemp having fled to the Kalahari desert, the campaign against South-West Africa was continued, preparations being well advanced by the middel of December 1914.

Four main forces were created (two corresponding with the previous organisations) as follows:—

(1) Southern force (formerly "A' 'and "B"), now based mainly on Upington which was railhead; and
(2) Central force (formerly "C") with sea base at Luderitzbucht. Two entirely new dispositions were:—
(3) Eastern force based on Kimberley, which was railhead; and
(4) Northern force with sea base at Walvis Bay.

(1) SOUTHERN FORCE.

The command of the troops, including commandos, from Upington westwards along the Orange River now fell to Col. (later Lt.-Gen. Sir) J. L. van Deventer. At the beginning of January, 1915, the force comprised of about 5,000 mounted riflemen [including burgers from 29 commandos, Hartigan's Horse, 17th (Western Province) M.R., and 14th Dismounted (but actually mounted) Rifles] and 1 battery — 4 guns — of Artillery, operating in 5, and later 4, columns. Reference has already been made to the unsuccessful attack by the rebels, assisted by German troops on Upington on 24.1.1915. Later actions took place at Kakamas (4.2.1915), Nabas, near Ukamas (8.3.1915), Platbeen (27.3.1915), and Kabus (20.4.1915), as a result of which the enemy was driven steadily back north of Keetmanshoop which town surrendered to Col. Bouwer.

To assist in the transport of supplies, the railway was extended from Upington to Kalkfontein (the German rail terminus) as Col. van Deventer's troops advanced, and field troops were supplied from rail head by mule wagons.

(2) EASTERN FORCE.

Berránge, who had commanded one of the S.A.M.R. regiments of "A" force was placed in command of the Eastern column which comprised 4 regiments of S.A.M.R. totalling 1,200 rifles ([16]) and 1 section of Artillery.

After traversing the semi-arid country between Kimberley and Kuruman, he followed the dry bed of the Kuruman River as it passes westward through the Kalahari Desert. He then struck north-west to Rietfontein, on the German-Union border, where a detachment of his troops defeated the enemy on 19.3.1915. Successful actions followed at Koes (5.4.1915), Kiries West (16.4.1915) and at Kabus (20.4.1915), referred to above, where the Southern and Eastern forces united.

The line of communication was over 500 miles, and both motor and mule transport was used. Special arrangements were made for providing water and troughs at tank stations on the desert stretches.

(3) CENTRAL FORCE.

As the Southern and Eastern forces were driving the enemy northwards from Keetmanshoop towards Gibeon during the middle of April 1915, Brig.-Gen. McKenzie, with 3 mounted brigades ([17]) and 2 batteries — each 6 guns — (these constituting the Central force) had commenced his drive eastwards towards Gibeon from Aus. At Gibeon

([16]) The **Official History** states (p. 54) that Berrange had the 5th Regiment S.A.M.R. and 2 Commando regiments.

([17]) Including Natal L.H., Imperial L.H., Umvoti M.R., 1st and 2nd M.R. (Natal Carbineers) and 3rd (Natal) M.R. totalling 1,800 rifles.

an action was fought on 27.4.1915 the Germans losing 11 killed, 20 wounded and 188 prisoners.

Water and supply difficulties had held up the advance. As an example, it may be mentioned that from Luderitzbucht to Garub (6 miles west of Aus) water was obtained either by sea from Cape Town or condensed at the coast. The country being desert, supplies had been collected at Aus (evacuated by Germans at the end of March) prior to the advance on to Gibeon and for this the railway was available. During the operations leading to the occupation of Gibeon, McKenzie had about 100 mule wagons, which sufficed until the railway was repaired.

At the beginning of April the Southern, Eastern and Central forces were amalgamated as the Southern Army under General Smuts. On 5th May the Army was disbanded, as by that time the southern part of the territory was free of German troops. Soon afterwards, viz. on 12.5.1915, Windhoek was formally occupied by the Northern force, as the enemy had retreated north of a line drawn between Karibib and Okahandja.

(4) Northern Force.

The first move in the creation of the above force, which was to bring the campaign to a close, was the occupation of Walvis Bay on Christmas Day, 1914, by a contingent under Col. Skinner ([18]). On 3.1.1915 Swakopmund which had been abandoned by the enemy, was occupied. On 10.2.1915 General Botha arrived at Swakopmund and took command of the force, which had then been reinforced by artillery and mounted commandos.

After clearing away the enemy from about 20 miles of the port and so making it possible to construct a railway, Botha decided to advance to Windhoek along the Swakop River. This involved travelling along the dry river bed through the desert belt of approximately 80 miles in which the enemy had prepared defences. An alternative route was along the railway and in this connection, Collyer (p. 58) comments: " It was stated that D.H.Q. had allotted the transport of the Northern Force on the understanding that the view last mentioned (i.e. that the advance would be along the railway) was to be adopted."

Botha, however, did not wish to sacrifice the mobility of his mounted troops and so preparations were hastened for establishing an advance supply base at Husab, 30 miles east of Swakopmund along the Swakop River. Infantry garrisons were placed not only on protective duty covering the railway construction, but also at strategic points, e.g. water holes, to secure the lower Swakop valley.

([18]) Comprising 7 guns artillery, 6 infantry battalions (2nd Transvaal Scottish, 2nd Kimberley Regiment, 1st Rhodesian Regiment, 1st Durban L.I., S.A. Irish and Witwatersrand Rifles) and Imperial L.H.

All mounted troops, except the Imperial L.H., were withdrawn to Swakopmund where they could be fed, and provisions, including fodder, were placed at convenient spots, e.g. water holes to augment the reserve at Husab.

By this time Botha had available for his first advance 2 batteries — each 4 guns — of artillery and 2 mounted (commando) brigades([19]) comprising approximately 8,000 men. Collyer (p. 60) states that "the only units which arrived at the scene of operations with their full transport were the 1st and 2nd Mounted Brigades, each of which had first line transport and a brigade train. The latter could carry one — capable of being stretched to two — day's supply." He adds that "Infantry Brigades, the Medical and Veterinary units and the machine-gun Brigade were all short of the transport allowed by their establishments."

We shall later see how the failure to provide for sufficient transport prevented the mounted troops from being used to the best advantage, viz. in regard to mobility and effecting surprise. To make matters worse, owing to the heavy sand along the coast, a team of ten mules could not pull a wagon, and "the addition of two mules to each team of ten — the regulation allowance — meant sacrificing a certain number of wagons" (p. 61).

"On 16.3.1915 after stripping every unit of its transport 5½ days supplies had been collected at Husab for the mounted force which was to make the attempt to reach Windhoek" (p. 62). As a result of the heavy and incessant work which took 16 days, the draught animals were returned to their units in a poor condition. Instead of 400 wagons having been available "the Northern Force had little more than one-tenth of that number" (p. 62).

Another factor which led to dissapointment later was the absence of grass-veld, supposed to commence at Riet, a point 30 miles east of Husab and therefore 60 miles from Swakopmund. By this time the 3rd Mounted Brigade under Col. Mentz had arrived at the port where fortunately it remained in reserve until conditions had been remedied.

On 18.3.1915 the advance began, and after engagements at Pforte and Jakalswater (on the railway) and Riet (in the Swakop valley) on 20.3.1915 the enemy was driven back, ultimately to concentrate north of the railway from Karibib to Okahandja. General Botha remained at Riet until the 24th id. but his supplies had entirely disappeared as had the hope that it might be possible to bring enough from Swakopmund to maintain a forward movement. The condition of the best horses would only allow of one more day's work (p. 78). In the circumstances "General Botha was forced to order his mounted troops back to Swakopmund where they could be fed from the ships" (p. 78).

([19]) During the advance Col. C. J. Brits and Col. J. J. Alberts commanded the 1st and 2nd Mounted Brigades respectively.

After an inevitable delay, General Botha decided to occupy Windhoek and to sieze the railway line between Karibib and the capital. This was to be the second advance.

Transport was again the stumbling block and once more units were stripped of their transport, this time to establish an advance supply depot at Riet. "Some additional transport had come from the Union, but the force to be fed was nearly three times as large as that which took part in the Riet-Pforte operations" (p. 85).

By 25.4.1915, 5 days supplies had been stored at Riet and "the return of the transport to the units would, if reliance could be placed on the timely arrival of considerable transport from the Union, leave 85 mule wagons and 30 donkey wagons — the latter in the end had to be put to work straight off the ship — to feed the force of approximately 13,000 troops ([20]) and 15,000 animals which was to move inland. With 400 wagons it might have been possible to maintain 2 — 3 days' supply with the troops" (p. 86).

On 26.4.1915 the advance began and by the 28th *id.* the 4 mounted brigades (1st, 2nd, 3rd and 5th) had concentrated at Riet. By 6.5.1915, after strenuous marching, the Union troops had secured possession of the railway from Karibib to Okahandja; and on 12.5.1915 Botha took over Windhoek. The Northern force however, was reduced to complete immobility .... and 18 days later (on May 23rd) was unable to move, though the local supply situation had improved" (p. 120).

The enemy in the meantime had retired along the railway to Tsumeb, Franke, the German commander, having previously planned a line of retreat from the rail head to the north via Namutoni on the Etosha Pan.

General Botha realised that in order to surround the enemy sufficient transport and supplies were imperative, so that rapid movement, without unnecessary delays, could be undertaken. A large amount of animal transport had now become available through the disbanding of the Southern Army, but in addition the railway had been reconstructed between Karibib and the coast; and "for the first time since General Botha had landed in the country, the supply question had ceased to be an apparently insoluble riddle" (p. 123).

The distribution of the wagons was as follows:—

1st Mounted Brigade	(Aukus to Namutoni	— 340 miles)	100([21]).
2nd „ „	(Wilhelmsdal to Tsumeb	— 280 „)	100([23]).
5th „ „	(Karibib to Otavi	— 230 „)	105([24]).
6th „ „	(„ „ „	— 230 „)	120([22]).

([20]) Collyer reports that at this time the Northern force had been compelled to slaughter oxen of the Heavy Artillery for supplies (p. 86).

([21]) Column commander was Brits; ([22]) Commander was Lukin.

([23]) Commander was Myburgh; ([24]) Commander was Manie Botha.

Right Wing 3rd Mounted Brigade (Wilhelmsdal
to Tsumeb .... — 280 „) 57([23]).
57([23]).
1st Infantry Brigade .... 50([22]).

On 18.6.1915 the Union forces totalling 13,000 men and 20,000 animals moved forward for the third advance and 21 days later the enemy was surrounded at Khorab near Otavifontein and the Union terms of capitulation accepted (9.7.1915).

The final advance involved many forced marches often without off-saddling and sufficient water, and examples of them are:— 5th Mounted Brigade from Okaputa to Kilo 500 (44 miles in 18 hours) and 6th Mounted Brigade from Omarrassa to Elephants Nek (36 miles in 15 hours).

Previously Col. Claassen's Standerton Commando, which had been resting at the coast on short rations, had marched 90 miles without water from Nonidas to Usakos. The record march of the campaign was that undertaken by the 2nd Mounted Brigade as follows:—

Left Swakop 6 p.m., 18.3.1915 and arrived Husab (40 miles) 8 a.m., 19.3.1915. Left Husab 8 p.m., 19.3.1915 and arrived Jakhalswater (38 miles) 5 a.m., 20.3.1915. In action for three hours and then returned to Husab for water, i.e. a total of of 116 miles in 36 hours.

(*To be continued*).

PHIMOSIS IN THE BULL — CASE REPORT.

C. W. A. Belonje
Kokstad.

Phimosis in the bull is common and as a rule caused by a narrowing of the lumen of the preputial orifice, or injury to the penis. The former can often be easily corrected surgically.

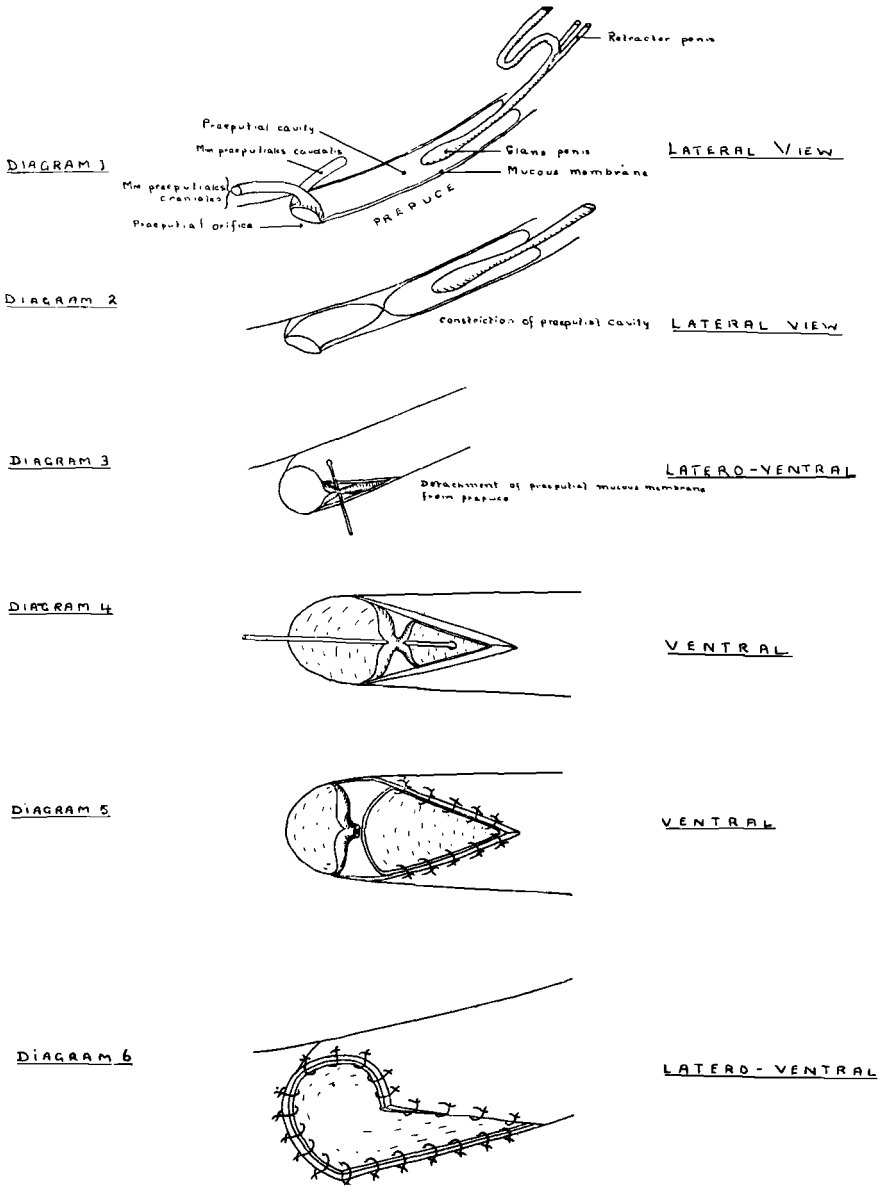
More unusual however is the phimosis of the preputial canal (Diagr. 2). If the constriction is severe, extrusion of the penis may be impossible and erection would only lead to eversion of the mucous membrane anterior to the constriction. Urination may be prolonged and occur more frequently (slow and frequent).

Case: A 15 month old pedigree Friesland bull was purchased on the 10th of June 1942. It served 8 cows normally but became ill on the 10th July. The disease appears to have been Anaplasmosis. On the 9th August the bull had completely recovered and was brought to service. The owner then noticed that although the bull attempted copulation there was only a protrusion of about 4″ of what he thought to be the penis. He assumed that the organ had "broken off."

Examination: On the 13th August examination of the standing animal indicated nothing abnormal. A cow on heat was brought up to it and the bull made several attempts at copulation which failed as only about 4″ of mucous membrane protruded from the preputial orifice. The bull was cast and a rubber tube passed but failed to go further than 4″. Dilatation of the preputial orifice revealed a funnel-like constriction of the canal with a lumen of $\frac{1}{8}$″ in diameter at its lowest point (Diagr. 2). External palpation revealed the constriction to be nodular and the size of a bean. It was decided to perform a radical operation.

Operation: On the 15th August, after 24 hours starvation, the bull (estimated to weigh 850lbs.) received 320 c.c. of a 10% aequeous chloral hydrate solution intravenously and 10 c.c. novocaine (Bayer) 2% epidurally and was cast on its right side. Grass filled bags were pushed under the limbs so as to lift the operation site from the ground. After proper precautions had been taken to ensure an aseptic operation a 4″ incision was made along the midventral line of the prepuce with strong blunt-pointed scissors, extending from the orifice to the constriction. The constricted preputial canal was then dissected from the skin and the entire preputial canal detached by blunt dissection from the surrounding tissues for a length of 3″ anterior as well as posterior to the constriction. Bleeding vessels were picked up by artery forceps. (Diagr. 3).

An artery forceps was applied to either side of the constriction, the canal drawn and a probe passed (Diagr. 4). The canal posterior to the constriction was then cut open with sharp pointed scissors in the midventral line for a length of 3″ and its cut edges stitched to the skin



OPERATION FOR PHIMOSIS (BULL).

with interrupted double catgut sutures. The preputial membrane was severed transversely behind the constriction (Diagr. 5), and the anterior membrane and skin carefully dissected and then removed with strong blunt-pointed scissors. Care was taken to avoid excessive removal of the intertwining fibres of the preputial muscles of the skin.

The free end of the mucous membrane was stitched to the skin with double No. 3 catgut sutures and the operation was complete (Diagr. 6).

The dissected portion measured approximately 5" long consisting anteriorly of the skin which continued as mucous membrane to the constriction which proved to be ¼" long with a lumen of ⅛th of an inch.

Post Operative Treatment. Hot water fomentations were applied 15 - 20 minutes every morning and evening, after which iodoform in olive oil was run into the sheath. An apron was put on to avoid soiling of the wound.

On the 29th August, 14 days after the operation, the bull was brought to a cow on heat to stimulate erection and to prevent constriction of the artificial preputial opening, but service was not allowed. Treatment continued as above with powdering of the wound with iodoform and boracic in equal parts after the fomentation and oil irrigation. He was brought to four more cows on heat at 3 - 4 day intervals and a two day interval, but not allowed to serve.

On the 17th September he was allowed to serve his first cow. There was no difficulty except that retraction was very slow. Treatment now ceased except for powdering the animal with equal parts of iodoform and boracic. Up to 15th October the bull served 10 cows, the periods between service being shortened from 4 to 3 to 2 and finally one day. The last treatment the bull received was on the 26th September after serving the third cow.

On the 15th October the owner reported that service was completely normal, and that the external orifice was large enough to accommodate the penis, and that the operation was entirely successful.

Discussion. It is apparent that the bull must have sustained some abrasion of the mucous membrane which contracted into scar tissue during a prolonged illness, resulting in a severe case of constriction of the preputial canal.

It will be observed that excision of certain lengths of the preputial mucous membrane is feasible, with complete recovery of function.

Note.

The foregoing case report has brought to mind a procedure successfully adopted in a similar case, namely one of abscessation of the prepuce with adhesions to the penis.

The prepuce was opened from the exterior at the site of the adhesions and the penis was dissected loose. A piece of bicycle tubing was then placed over the penis and drawn through the preputial canal to posterior to the wound, where it was fixed by stitches to the outside. The end of the tubing was then cut off leaving about one inch, extruding and was stretched to the preputial ring. This prevented both soiling of the wound with urine and the recurrence of adhesions. After the wound had healed the stitches were snipped and the tubing withdrawn. The procedure was entirely successful.

Richard Clarke,
Onderstepoort.

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

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(Continued from Vol. XIII, No. 4, December, 1942).

V. Treatment of cases of sulphonamide poisoning

The following methods of treatment are indicated in cases of poisoning with sulphonamides:—

(a) Administration of nicotinic acid.

(b) Intravenous administration of isotonic glucose solutions and methylene blue to relieve cyanosis due to methæmoglobinæmia. The average intravenous dose of methylene blue recommended by Jordan for man is 20 mg. or 2 cc. of a 1% solution. It should be pointed out here that glucose-sulphapyridine tablets were found more harmful than sulphapyridine tablets alone; both the free and total sulphapyridine levels in the blood of rats and rabbits are higher with the former tablets than with the latter.

(c) Treatment in renal complications should consist of alkalinization, diuresis, and ureteral cathetherization and irrigations with warm (107°F) distilled water. The bactericidal effect of sulphanilamide is greater in alkaline than in acid urine.

(d) Clonic and tonic convulsions should be treated with small doses of hypnotics, like urethane or nembutal. The use of large doses of these hypnotics is warned against as they are inclined to cause death by increasing the secondary depressant action of sulphonamides. If such cases should occur, picrotoxin should be used to overcome the synergistic effects of the hypnotics and sulphonamides. It is reported that subhypnotic doses of nembutal can completely eliminate restlessness, nausea and vomiting, which follow the use of large doses of sodium sulphapyridine.

(e) Cases of alkalosis and acidosis should be treated accordingly.

VI. Prophylaxis

In the prevention of complications, which may arise in the course of treatment with sulphonamides, the following points are of value:—

(a) It is preferable to commence with small doses, especially in aged patients, and increase these if the patient tolerates them well. Experience appears to indicate the development of hypersensitivity

to the sulphonamides, consequently it is not advisable to administer these drugs uninterruptedly for long periods.

(B) These drugs should be used with the greatest caution in diseases of the blood and liver and when kidney function is in any way impaired. In order to protect the kidneys during sulphonamide administration the daily urinary output should be increased; in man this should be at least 1500 cc.

(C) As many of the sulphonamides are apt to cause acidosis, it is recommended that an alkaline urine be maintained during administration of these drugs. Sodium bicarbonate protects the system against poisoning with sulphonamides, especially as far as kidney damage is concerned.

(D) Lederer and Rosenblatt (1942) warn against the indiscriminate and uncontrolled use of sulphathiazole. During administration of this drug the urine should be regularly examined as crystals of the drug in the urine, hæmaturia and suppression of urinary output are danger signals. A further danger signal is a chill occurring during sulphathiazole administration and which is followed by sustained fever. In these cases the administration should be discontinued without delay. In man concentrations of over 10 mg. of sulphathiazole per 100 cc. of blood are undesirable.

(E) Frankland (1941) warns against the subcutaneous administration of "sulphapyridine soluble" as it results in ulceration of the skin with a persistent sinus. In cases where this drug was injected into the gluteal muscles he has seen foot-drop with varying degrees of anæsthesia as a result of damage to the sciatic nerve. This condition may last for months. Intravenous administration of this drug is recommended in cases of emergency only. There are also reports that intramuscular injection of sodium sulphapyridine resulted in prolonged and permanent damage to the muscles at the site of the injection. Watt and Alexander (1942) report five cases of epilepsy following application of sulphathiazole in proximity to the brain in the frontal region. This epileptogenic property of sulphathiazole has been confirmed in animals, and is apparently not shared by sulphanilamide, sulphapyridine, sulphadiazine and sulphacetamide (= albucid). According to Futch and his co-workers the instillation of 1 cc. of a 5% aqueous solution of sodium sulphathiazole into both nostrils of rabbits three times daily resulted in an early and severe destructive effect on the nasal mucosa.

(F) Nodular rash is the most serious lesion of all cutaneous manifestations in sulphonamide medication. Conjunctivitis is also frequently seen.

(G) From experiments upon animals it appears that high-protein (casein) diets protect against sulphanilamide poisoning and that high fat and high carbohydrate diets increase susceptibility to the

harmful effects of these drugs. There are many references in the literature concerning the protective action of high protein diets against liver poisons. On the other hand there is no doubt that sulphur-containing substances, which on decomposing may liberate sulphuretted hydrogen, facilitate the production of sulphæmoglobinæmia when administered simultaneously with sulphonamides. Such substances are proteins (eggs, etc.), sulphur, sulphites, thiosulphates, etc., and they should therefore be avoided during sulphonamide administration.

(H) Purgatives should not be used during sulphonamide administration, but if evacuation of the bowels is necessary it should be brought about by the use of liquid paraffin and enemata. These steps counteract the effect of cyanosis caused by the development of sulphæmoglobinæmia.

(I) Sulphapyridine appears to inhibit the curative action of nicotinic acid in dogs. In mice sodium acetate reduces the toxicity of sulphanilamide when the two drugs are administered together, but it does not seem to be of any value in preventing chronic toxic effects. On the other hand, ammonium acetate increased the toxicity of sulphanilamide; this is presumably due to the toxicity of the ammonium ion. It is also interesting to note that acetylsulphanilamide was found to be more toxic than free sulphanilamide. The acute toxicity of sulphadiazine in the mouse is reduced by pyridium.

(J) Parenteral administration of Betaxan (vitamin B_1) was most successful in the prevention of paresis of the extremitis of pigeons induced by the feeding of Uleron. It is also reported that calcium rapidly cleared up eruptions of the skin caused by Uleron.

(K) Drugs, which predispose to methæmoglobinæmia should not be administered at the same time as sulphonamides. Such drugs are phenacetin, nitrites, amidopyrine and members of the sulphonal group.

(L) Quinine and sulphonamides should not be administered simultaneously as the former facilitates the absorption of sulphonamides and also their excretion by the kidneys, and consequently aggravates kidney complications. These effects were not noticed when atebrin was used instead of quinine.

VII. Conclusions

(A) Sulphonamides are recognised dangerous drugs and consequently only sold on a physician's prescription.

(B) From the literature it appears that the sulphonamide preparations should be used as follows in the various infections:
  (a) Uleron in heartwater (Rickettsia ruminantium).
  (b) Sulphanilamide in streptococcal and coliform infections.
  (c) Sulphathiazole in staphylococcal infections.

(d) Sulphapyridine in pneumococcal infections.

(e) Either sulphathiazole or sulphapyridine in meningococcal and gonococcal infections.

(f) Sulphadiazine is said to be highly effective in pneumococcic, staphylococcic and streptococcic pneumonia, meningococcic infections, influenzal meningitis, acute infections of the upper respiratory tract including tonsillitis, sinusitis, erysipelas, acute infections of the urinary tract, particularly those associated with bacillus coli and acute gonorrhœal arthritis. Toxic effects from its use are relatively mild and infrequent and it is very well tolerated by man. Consequently its use is indicated in the treatment of chronic bacterial infections where prolonged administration of the drug is required.

(g) The recently introduced " sulphonamide E.O.S." has been used apparently with good results in a variety of infections. It is of low toxicity and should be used in twice the doses that sulphanilamide is.

(h) Sulphapyridine in particular should be used with care as it is inclined to cause gastrointestinal disturbances and urinary calculi. Sulphathiazole also tends to cause urinary calculi, but is much less irritant to the gastric mucosa than sulphapyridine.

(i) Pneumococci which are sulphapyridine-fast are equally resistant to sulphathiazole and sulphamethylthiazole.

(j) Favourable results are reported in the treatment of coccidiosis in cattle with sulphanilamide.

(k) In large animal practice sulphathiazole is considered less toxic than sulphanilamide and it is much more effective in pneumonia and certain other infections.

(l) Sulphamethylthiazole affords only very slight protective action in mice infected with a virulent strain of Salmonella enteritidis.

(m) Several authors report that insufflations of sulphathiazole or sulphanilamide powder into both nostrils yielded negative swabs within five to eight days in diphtheria carriers. Such insufflations, especially of sulphadiazine powder, will probably be very effective in the treatment and prevention of colds and tonsillitis.

(c) Sulphonamides are of great value in the treatment and prevention of infections in wounds, and also in peritoneal infections. It is not advisable to suture wounds after having placed solid sulphonamides in them, as they cause severe inflammation and abscesses. Some sulphonamides also cause severe inflammation of the peritoneum.

(d) The instillation of 1 to 20% aqueous solutions of sulphonamides, preferably sulphadiazine and uleron, may be of great value in the treatment of ophthalmia. One to five per cent. ointments, or

suspensions in cod liver oil, may be more effective, as the drug will remain in the conjunctival sac longer than when it is applied in the form of a solution. Thirty per cent. aqueous solution of sulphacetamide have been used in the treatment of eye diseases in man.

According to Maybaker (S.A.) (Pty.) Ltd., Johannesburg, their preparations sodium sulphathiazole (thiazamide sodium) and Dagenan Sodium (M & B 693 soluble) are unsuitable for application to the eyes as they are too irritant. On the other hand their preparations Sodium Sulphacetamide and Soluseptasine are non-irritant and their solutions may be instilled into the eye.

(E) In a few cases of purulent discharge from the ears the author achieved very good results by daily instillation of a few drops of cod liver oil containing 10% of prontosil powder in suspension. This suspension also yielded excellent results in the treatment of infected wounds.

(F) Sulphanilamide and sulphapyridine have no effect on immune body formation.

(G) The curative effect of sulphanilamide upon streptococcal infections in mice is completely inhibited if it is administered simultaneously with ρ-aminobenzoic acid. There is very sound experimental evidence supporting the theory that the bacteriostatic effects of the sulphonamide drugs are due to the fact that they compete with the essential metabolite, ρ-aminobenzoic acid, for an important site on the bacterial cell. On the other hand there is no evidence, at present, to support the theory that the sulphonamides exert their anti-bacterial effects by interfering with the functioning of the chemically related coenzymes, cozymase and cocarboxylase.

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(*Concluded*).

OBITUARY.

MR. C. R. EDMONDS.

The death occurred at Sharnbrook, Bedfordshire, England, recently of Mr. C. R. Edmonds, at one time Assistant Chief Veterinary Surgeon of the Colony, who some years ago was widely known as "Farmer George." He was about 75 years of age.

Qualifying as a veterinary surgeon in London in 1891, Mr. Edmonds came to Rhodesia and started a private practice in Bulawayo, which he continued until 1899, when he joined the Rhodesian veterinary service. Stationed at Bulawayo, he served under Mr. J. M. Sinclair. When Mr. Sinclair became Chief Veterinary Surgeon in 1904, Mr. Edmonds was appointed Senior Veterinary Surgeon-in-Charge, Matabeleland, and in 1912 he became Assistant Chief Veterinary Surgeon, which post he held until

his retirement in October, 1924, when he left to settle in England, taking up poultry farming.

Mr. Edmonds was the author of the well-known work published in 1921, "Disease of Animals in South Africa," which was re-written in collaboration with Colonel G. K. Walker, of the Indian Veterinary Service, and published in 1929 under the title "Diseases of Animals in Tropical Countries."

For a number of years Mr. Edmonds contributed articles on farming topics to the *Bulawayo Chronicle* under the pen name "Farmer George."

BOOK REVIEW.

Handbook of Practical Bacteriology. Mackie and McCartney.

The sixth edition of this well known and very useful handbook has been brought up to date and much new material has been added, necessitating a considerable increase in size. The first half of the book is devoted entirely to technique and much valuable material has been added, particularly in the section devoted to immunological and serological methods. The second half of the book deals with the pathogenic and commensal micro-organisms including the filterable viruses, and bacteriological diagnosis. An excellent description is given of the technique for the serological classification of the streptococci. Under tuberculosis, the preparation and standardization of the tuberculin is not described but it is mentioned that reference should be made to more detailed works for the necessary information. Under anthrax no reference is made to the more recent work on immunization. The Pasteur vaccine has been given up in most countries in favour of spore vaccines usually with the addition of saponin. The section on the coli-typhoid group is up to date or as nearly so as is possible when fresh information is being obtained at a great pace. A good table is given of the sub-groups of the salmonellas and it is as up to date as possible under the circumstances. Good sections on the practical aspects of the Rickettsia infections and the filterable virusus have been included, and an appendix on the mode of action of the sulphonamide compounds. This latest edition of such a well known text book is very welcome and should prove of great value to working bacteriologists who want the most recent information about technique in bacteriology and immunology.

E.M.R.

"*Handbook of Practical Bacteriology.*" A guide to bacteriological laboratory work. T. J. Mackie and J. E. McCartney Sixth Edition. E. & S. Livingstone, Edinburgh.

THE AETIOLOGY OF EAST COAST FEVER.

W. O. NEITZ.
Onderstepoort.

INTRODUCTION.

The remarkable progress which has been made in the studies of the biological characteristics of protozoa and vira since the days when the aetiology of East Coast fever was first described (Koch, 1897, 1903, and Theiler, 1904) compels one to-day to reconsider very carefully whether *Theileria kochi* (Stephens and Christophers, 1903) or as it is commonly known *Theileria parva* (Theiler, 1904) is the sole cause of this disease. In general reviews on immunity in protozoal diseases by Schilling (1930) and by Kikuth (1933) it is pointed out that the immunity in East Coast fever is sterile, in contra-distinction to that observed in other protozoal diseases, where the immunity develops as a result of a latent infection. The existence of a sterile immunity in East Coast fever, which Du Toit (1928) proved beyond any doubt, has lead to a great deal of speculation as to the manner in which such an immunity is brought about in a protozoal disease. Up to the present, however, no satisfactory explanation has been given. Not only does East Coast fever differ in this respect from other protozoal diseases, but the striking fact has been established that it differs also from the diseases caused by other species of the genus *Theileria,* viz. *Th. dispar, Th. annulata* and *Th. mutans.* The object of this article is to point out several characteristics of this disease which seem to indicate that East Coast fever cannot be considered purely as a protozoal disease. For this reason the aspects dealing with the experimental work on the immunization of cattle and the nature of the immunity in East Coast fever will be considered.

DISCUSSION.

In order to understand the various theories which have been advanced about the aetiology of East Coast fever, it is essential to review very briefly some historical events in the investigation of this disease.

1. THE PROTOZOAL PARASITE AND ITS DEVELOPMENT.

Theileria parva is still accepted to-day as the cause of East Coast fever. The idea of Fülleborn, Ollwig and Martin Mayer, who looked upon the "Koch's bodies" as "inclusion bodies" of a virus, was dropped at the time when K. F. Meyer demonstrated that these bodies gave rise to organisms which were true protozoal parasites. At a

later date, Gonder (1910) studied the life cycle of *Theileria parva* in detail, and clearly proved that the Koch's bodies were a developmental stage in the life cycle of this protozoal parasite. The description of this parasite by Koch, Theiler, Meyer, and Gonder has been the basis for all further investigations of East Coast fever and those of other Theileria infections.

2. The Method of Immunization and the Nature of Immunity in East Coast fever.

After the recognition of *Th. parva* as the cause of East Coast fever, it naturally followed that methods were looked for which would assist in controlling this disease. With this object in view, Koch (1903) attempted to establish a method by which it would be possible to immunize cattle. For this purpose Koch injected large quantities of blood and of emulsions prepared from the spleens of animals showing good East Coast fever reactions, into immune animals. Once these animals had been hyperimmunized, their serum was utilized for controlling East Coast fever reactions. The remarkable properties which this serum had may be summarized as follows:—

The serum did not cause any systemic disturbances when it was injected into healthy animals. The injection of it into cattle suffering from East Coast fever caused a striking change in the parasites circulating in the blood. The parasites degenerated and in the course of a few days they disappeared. Unfortunately this serum possessed an undesirable property of causing haemolysis of the erythrocytes in sick animals, whereas in healthy animals the phenomenon was not observed. From these observations Koch concluded that the serum of hyperimmunized cattle possessed the property of directly attacking the specific parasites.

The work of Koch was repeated by Theiler (1907), but the protozoacidal and the haemolytic properties of the serum collected from hyperimmunized animals could not be demonstrated. Since it appeared that the method of Koch could not be used for controlling the disease satisfactorily, Theiler (1911) investigated other possibilities of immunizing cattle against East Coast fever.

The artificial transmission of East Coast fever with blood can be achieved only in relatively few cases. The investigations by K. F. Meyer (1909), however, showed that the intravenous injection of an emulsion prepared from the spleen and glands produced symptoms of this disease in a large number of animals. With this method of infection it was shown that there was a striking difference between the naturally acquired and the artificially produced diseases. In the former the course of the disease is fatal in 95% of cases, whereas in the latter more than 50% of the animals which react recover.

This observation naturally prompted further investigations of the

possibility of establishing a practical method for the immunization of cattle. With this object Theiler (1912) conducted experiments at the Onderstepoort laboratory. From the preliminary tests, which were carried out with several hundred animals, it was observed that different types of reactions developed when the animals were injected intravenously with emulsions prepared either from the spleen or the lymphatic glands. These reactions may be briefly summarized as follows:—

a. Typical reactions developed which terminated in death (*Th. parva* and Koch's bodies could be demonstrated).
b. Typical reactions developed from which the animals recovered (*Th .parva* and Koch's bodies could be demonstrated).
c. Mild and irregular reactions developed from which the animals recovered (*Th. parva* and Koch's bodies could be demonstrated).
d. Mild reactions developed from which the animals recovered (*Th. parva* and Koch's bodies could not be demonstrated).
e. In some of the animals no reactions were observed at all.

The animals which survived the artificial infection were subjected to an immunity test. This was done by infesting the animals with known infected ticks. In many instances the immunity test was applied for a second time and in some cases the animals were exposed to a natural infection as well for periods of up to three years.

On analysing the results of the immunization process in 236 cattle which were injected intravenously with spleen and gland emulsions, Theiler (1912) made the following observations:—

1. 59 cattle (25%) contracted East Coast fever and died.
2. 58 cattle (25%) contracted East Coast fever and recovered, of which 3 reacted and died and 7 reacted and recovered when subjected to an immunity test, and 48 proved to be immune.
3. 114 (49·2%) did not show plasma bodies as a result of the injection and when their immunity was tested, 47 contracted East Coast fever and died, 12 contracted East Coast fever and recovered, and 55 proved to be immune.
4. 5 animals (1·8%) died from intercurrent infections. The conclusion one can draw from the above results is that an immunity developed in 83% of animals that had recovered from a typical East Coast fever reaction and 48% of animals in which no *Theileria* could be demonstrated in the peripheral blood nor Koch's bodies in the organs of the body. No comment is made by Theiler as to how an immunity can possibly develop in a protozoal infection without the parasites developing in the animal body.

In addition to the laboratory experiments, observations on the immunization process under field conditions were made by Theiler, Kirkpatrick, Goodall, and Chambers (1912). Similar results were obtained by these workers, who arrived at the following conclusions:—

Approximately 25% of the artificially infected cattle died from East Coast fever, 35% reacted and recovered, whereas the remaining 40% of animals did not show *Theileria* in the blood nor Koch's bodies in the organs. All the animals which survived the artificial infection were exposed to a natural infection for periods of up to three years, and of these no less than 72% were found to be solidly immune. Here again it will be seen that an active immunity had been established in animals in which *Th. parva* and Koch's bodies could not be demonstrated.

The immunization of cattle against East Coast fever was discontinued because it was considered at the time that the systematic dipping of cattle was a more effective method of eradicating this disease. The immunization process always created a large number of reservoirs from which ticks could infect themselves, whereas the systematic dipping destroyed the infected ticks. Once the policy of eradicating the vectors had been decided upon, it followed naturally that attention had to be paid to the fact that animals which had recovered from East Coast fever might act as reservoirs of this disease. It was, however, shown by Du Toit (1928) that cattle which had recovered from East Coast fever did not harbour a latent infection of *Th. parva,* in other words he showed that these animals had a sterile immunity.

The observations by Koch are extremely striking inasmuch as he demonstrated for the first time that in a protozoal disease a serum could be produced with protozoacidal properties. In this respect East Coast fever seems to differ from all other protozoal diseases, since in none of them has such a serum been produced. The demonstration of this property of the serum is sufficient reason to enquire further whether East Coast fever is truly a protozoal disease.

Further evidence which casts doubt on our present conception of the aetiology arises from the well conducted laboratory and field investigations of Theiler (1912). It was mentioned above that an active immunity was observed in 49·2% of animals which had not shown any reaction at all to the *Theileria* parasites. In other words something had taken place in the animal body as the result of the artificial infection which rendered them immune. In all the other diseases caused by protozoa which develop in the blood stream an immunity is only established after the parasites have completed their life cycle in the animal body. In East Coast fever, however, an immunity can be established in two ways. In the first instance in animals which recover from a typical reaction, and in the second instance in animals in which the development of *Th. parva* has not taken place.

The doubt about the conventional accepted conception of the aetiology of East Coast fever becomes even greater if one takes into account the fact that a sterile immunity is produced in a disease supposedly caused by a protozoal parasite. (Du Toit, 1928).

The above statements may be summarized briefly as follows:—

1. In cattle suffering from East Coast fever the protozoal parasites *Th. parva* are invariably found.
2. The serum obtained from hyperimmunized East Coast fever cattle has a protozoacidal action on *Th. parva.*
3. Numerous cases have been observed under laboratory and under field conditions in which an immunity against East Coast fever develops in the absence of the protozoal parasite, *Th. parva.*
4. A sterile immunity is present in cattle which have recovered from East Coast fever.

Analysis of these facts compels one to reconsider one's views on the aetiology of East Coast fever. The only conclusion that seems justified is that this disease is caused by two factors, *Theileria parva* and another disease-producing entity.

The question immediately arises: What is the nature of this second factor? If this disease-producing organism had been a bacterium, it would have been noticed by pathologists examining the sections from organs and by those (Cowdry and Ham, 1938, and Reichenow, 1937) who have studied the life cycle of *Th. parva* in the tick, *Rhipicephalus appendiculatus.* Since no organism has been demonstrated microscopically, it is assumed that the disease-producing organism associated with *Th. parva* is a "virus." Its existence has been shown by the fact that cattle can develop an immunity against East Coast fever in the absence of the protozoal parasite usually associated with this disease.

If one accepts this view about the aetiology of East Coast fever, one is faced with a large number of questions:—

1. In what way will we have to change our views regarding the classification of the *Theileridae?*
2. Is our conception of the aetiology of the diseases which are caused by other species of *Theileria* correct?
3. To what extent will our present methods of control have to be modified?
4. Can the virus factor in East Coast Fever exist in cattle and in the vectors in the absence of *Th. parva?*
5. Can the virus component exist in animals other than cattle? If so, can these animals act as reservoirs?

Before one can answer any of these questions satisfactorily, it is quite clear that a great deal of research work has to be undertaken.

Nevertheless it is advisable that an attempt at answering some of these questions should be made.

In connection with the first question on the classification of the Theileridae, it is essential to mention the conclusions Du Toit (1930) arrived at when he reviewed the biological characteristics of the diseases of cattle caused by the different *Theileria* spp. He raises the question whether the four species of *Theileria* should be regarded as distinct since there is a gradual transition from the most virulent species *Th. parva* to the avirulent one *Th. mutans*. Doubt is expressed whether the four species already mentioned are "good" ones. He states that the difficulty could be overcome either by regarding all the parasites as varieties or strains of one species, *Th. parva*, or by creating new species for each of the unknown types of *Theileria*. Neither of these alternatives is recommended, but rather to follow an intermediate and more conservative course and to accept provisionally the four species named by him. At the same time he points out that the name *Th. annulata* has apparently not always been correctly applied and that *Th. mutans* may prove to be synonymous with *Th. annulata*.

From the above it will be seen that the different names given to the *Theileria* species were based on cross-immunity tests which proved that the *Theileria* diseases were not identical. The production of different reactions in cattle by these parasites was the primary reason why different names were allocated to them. Morphological comparison of the parasites has given very little aid in the classification. A difference which is frequently mentioned is that in *Th. parva* one does not find any multiplication in the erythrocytes, whereas in *Th. mutans* such a development does take place. It is at present questionable whether much significance can be attached to this observation if one considers the morphology of *Leishmania donovani* (Laveran and Mesnil, 1903), a parasite which undergoes such morphological changes when cultivated on artificial media, that the forms have no resemblance whatsoever to the parasites in the body. In this connection it must be mentioned that the natural medium in which *Th. parva* develops differs greatly from that of *Th. mutans*. In East Coast fever it is noticed that an active multiplication of the lymphocytes takes place, whereas in *Th. mutans* infections such an active mitosis does not occur. At present it is not clear whether the change (De Kock and Neitz) * is due to the "virus" or to the protozoal development of the disease. One fact, however, is quite clear, that the proliferation of the lymphocytes commences right from the beginning of the reaction and that the superabundant number of young lymphocytes are an ideal medium for the development of *Th. parva*. In these circumstances one must therefore expect a more active

* An article by de Kock and Neitz on "The Pathology of East Coast Fever" is in course of preparation.

multiplication of *Th. parva* than in an infection of *Th. mutans*, where the proliferation of the lymphocytes is as a rule only slightly higher than under normal conditions.

With reference to the second question, it must be mentioned that in the other *Theileria* diseases one does not meet with a sterile immunity except under certain conditions, in *Th. dispar*. For this reason one is not in the position to draw any conclusions whether the cause of these diseases consists of two factors. An indication that a virus may be associated with these protozoa is given by an observation made by the author. Cattle suffering from Tzaneen disease (*Th. mutans* infection) were infested with known clean *Rh. appendiculatus* larvae. Some of the nymphae which developed from these larvae produced Tzaneen disease when they were allowed to feed on an ox raised under tick-free conditions. The infestation of two sheep with nymphae from the same batch resulted in a febrile reaction. The incubation period of the reaction in the ox was as long as that of the reaction in the sheep (16 days). In the ox, Koch's bodies and *Theileria* parasites could be demonstrated; in the sheep however, no protozoal parasites could be found. The febrile reaction observed in the sheep shows that the nymphae had become infected with some disease producing factor in addition to this *Theileria*. The nature of the infective agent has not been studied further, so that it is not possible to give any indication about its properties. It is believed that this observation as well as those made in East Coast fever will be of great assistance in indicating a correct classification of the *Theileridae*. The doubt Du Toit (1930) expressed as to whether the four species of *Theileria* are "good" ones is fully justified if one takes these new views into consideration. No comments will be made on the remaining three questions. Speculation on these aspects of East Coast fever will in no way improve the control measures which have been practised up to the present date. Careful investigations are essential before any opinion can be expressed on these points.

Conclusions.

1. Attention is drawn to the difficulties connected with and apparent inconsistencies between the various species of *Theileria* and the diseases and the type of immunity they produce.
2. From the experimental work and observations of Koch, Theiler and Du Toit, evidence is advanced to show that the results obtained are not in accordance with our present accepted views on protozoal immunity in general.
3. The suggestion is advanced that East Coast fever is in fact a combined infection of at least two factors:—
   (a) a protozoal parasite *Theileria parva*, and (b) probably a "virus," and that the sterile immunity in this disease is due to the latter.

4. The assumption of such a possibility would facilitate the rational classification of the *Theileridae*.
5. Some of the questions which would immediately arise and require experimental elucidation, should this assumption prove to be correct, are mentioned.
6. No modification in present control measures are envisaged or even worth considering until the validity of this thesis has been thoroughly explored and satisfactorily established.

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A REVIEW OF PSITTACOSIS IN DOMESTIC BIRDS WITH A NOTE ON A CASE OF CONJUNCTIVITIS IN A PIGEON PROBABLY DUE TO PSITTACOSIS.

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Onderstepoort.

With the discovery of psittacosis in domestic pigeons in 1940 by Pinkerton and Swank, and also by Coles, a new chapter was opened on the ecology of the disease. Hitherto only wild birds were held suspect, but the researches ushered in by the above work revealed the widespread nature of psittacosis in pigeons in North America and even in the domestic fowl and doves. Now the virus has been isolated from pigeons in England. Eddie and Francis have also found serological evidence of the infection in domestic turkeys and ducks in Michigan, but no attempt was made to isolate the virus.

While many of these studies on avians were being conducted, investigators such as Francis, Magill, Eaton, Beck, and Pearson were concerned with fairly numerous cases of atypical pneumonia in man in both the eastern and western regions of the United States. It is remarkable how regularly the pigeon types of the psittacotic virus have been isolated from these patients.

The purpose of this article is twofold. Firstly, it is intended to present veterinarians with the salient facts pertaining to natural psittacosis (or ornithosis) in domestic birds, and to the corresponding infection in man. Secondly, reference is made to a case of conjuctivitis in a military pigeon, that was most probably due to psittacosis.

Review of the Relevant Literature.

In 1938 Francis and Magill published a report on an unidentified virus producing acute meningitis and pneumonitis in experimental animals. The virus was invariably isolated from ferrets inoculated intranasally (i.n.) with throat washings from human cases of what resembled epidemic influenza, occurring in California and along the Atlantic seaboard during 1934 and 1936. The agent grew readily on the chorio-allantoic membrane of the chick embryo and was pathogenic for ferrets, mice, monkeys and also, to some extent, for rabbits and guinea pigs. Mice were easily infected, large doses killing in two to four days and small doses in fifteen to twenty-one days, and only rarely did an animal recover. Symptoms were ruffled fur, sticky eyes, and laboured, audible respirations. The mouse lung was characterised by a typical, firm, rubbery, pinkish-grey consolidation, and considerable oedematous fluid sometimes exuded. The intracerebral

(i.c.) inoculation of mice led to paralysis in two to ten days, depending on the dose. A variable disease followed the employment of the intraperitoneal (i.p.) route; the mouse sometimes sickened and the abdomen was swollen and contained some cloudy fluid and much fibrin. Large i.p. doses killed 50% of the mice with paralysis, resembling that evoked by i.c. inoculation. Mice surviving i.n. infection were resistant to reinoculation by the same route. Recovery from an i.p. or subcutaneous (s.c.) infection implied resistance to subsequent i.c. inoculation, regardless of whether or not the initial infection produced symptoms; but these mice were fully susceptible to virus administered i.n. The immunity engendered by one strain of the virus was fully effective against all other strains of what was apparently the same virus. Francis and Magill called the agent the "virus of acute meningo-pneumonitis."

Early in 1939 Coles demonstrated the existence of psittacosis in fancy domestic pigeons in South Africa and a report on the disease appeared in 1940. Infections due to *Salmonella typhimurium, Trichomonas hepatica* and *Haemoprotcus columbae* complicated the picture. The i.p. infection of mice sometimes produced large spleens and occasionally even death, and the demonstration of the L.C.L. bodies in fatal cases was easy. No attempt was made to inject mice and pigeons i.c., as the significance of such a procedure was unknown at the time. The virus could be seen in the lungs of some pigeons.

Pinkerton and Swank in 1940, when working in St. Louis, found psittacosis in young pigeons purchased in South Carolina. The birds were on an experimental diet very low in thiamin. Less than 5% of 400 pigeons presented definite evidence of the disease, but no tests for inapparent infections were made. A fibrinous peritonitis and pericarditis, and sometimes a large haemorrhagic liver, were presumably due to the virus, which was found on the surface of the liver in two pigeons. The i.c. inoculation of mice killed them in two to four days; a chorio-meningo-encephalitis was produced with mononuclear cells predominating in the inflammatory exudate and virus particles plentiful in the mononuclears. Mice did not sicken when injected i.p. or s.c. with large amounts of virus.

In 1941 Meyer made the statement that human infections indistinguishable from clinically typical psittacosis might follow exposure to doves, pigeons and fowls, and he emphasized the value of the complement-fixation test as an aid to diagnosis. After testing the serum of the patient and birds concerned, he concluded that a zoologist in Chicago had contracted psittacosis from some doves, *Streptopelia risoria*. He also described the following cases:—

In Pasadena an old man developed weakness, severe headache, fever and prostration, with marked nausea and anorexia. Pneumonia followed and death occurred on the fifteenth day of his illness, despite sulphapyridine and sulphathiazole therapy. The complement-fixation

test was positive at 1 in 2 on the eighth day, and at 1 in 8 on the twelfth day. Mice inoculated i.p. with lung material were still healthy looking after twenty days. Other mice infected i.n. had focal pneumonic patches in eight to ten days. Mouse passage, using the i.n. route, enhanced the virulence of the strain and massive fatal pneumonias then developed in six to thirteen days. L.C.L. bodies were seen readily in mouse lung smears. The i.c. infection of mice led to a typical chorio-meningo-encephalitis, ending fatally in three to four days. Mice injected i.p. developed large spleens and sometimes marginal hepatic necrosis. Ricebirds, shell parrakeets and doves died in seven to fifteen days after intramuscular (i.m.) infection. The i.c. injection of diluted virus killed pigeons with meningitis in five to seven days. The man undoubtedly acquired the disease from a pigeon owned by his son, whose serum gave a positive complement-fixation test at 1 in 16. The sera of twenty out of thirty surviving pigeons were positive at 1 in 8 to 1 in 256 and the virus was isolated from the spleens and livers of one non-reacting and four reacting birds. Another elderly person in Los Angeles had a history of weakness, fever, cough, leucopenia and irregular hazy infiltrations of the lateral right midlung field. The complement-fixation test was positive at 1 in 256. Recovery was uneventful and no attempt was made to isolate any virus. The son of the patient kept eighty carrier pigeons in the backyard and several had shown loss of weight, diarrhoea and general illhealth. Two of twelve pigeons examined gave reactions at 1 in 128, and three at 1 in 16. An old sick female negative reactor had a large spleen and a virus was isolated that, on passage, produced focal lobular pneumonia in mice. Many of the pigeons had a concurrent S. typhimurium infection. In New York, a mother and her daughter picked up a sick pigeon on the street and nursed it until it died four days later. Within a fortnight both were ill. Chilliness, malaise and a high fever were followed by pneumonia, which did not respond to the use of sulphathiazole. The daughter recovered in three weeks and her mother later. The daughter's serum was positive at 1 in 8 on the twelfth day of illness and the mother's at 1 in 16 on the fifteenth. Twenty of thirty sera from local pigeons were positive at 1 in 4 to 1 in 258. A physician suspected psittacosis in a fifty-four years old woman who farmed poultry in New Jersey. She ran a temperature up to 105°F and had pulmonary consolidation with no cough. Recovery followed soon. Her serum reacted at 1 in 32 on the twelfth day of the illness. She was still positive at 1 in 128 on the 30th and 155th days. Three frozen fowls were shipped to California for examination. One was emaciated and had a slightly enlarged liver and spleen; an organ emulsion was inoculated i.p. into mice and produced rather swollen spleens in the first and second generations. Third passage mice contained much peritoneal exudate and many L.C.L. bodies were visible. This fowl virus was only

moderately infectious for mice and rarely killed, but often, on the twentieth day after i.p. injection, the usual plastic exudate might be seen together with splenic enlargement and marginal liver necrosis. Mice developed extensive lobar pneumonia after i.n. infection. Pigeons, doves and chickens infected i.c. from mice became paralysed, died of meningitis in five to eleven days and numerous parasites were found locally. Pigeons sometimes succumbed in ten to forty-one days after i.m. injection; doves and chickens usually survived this treatment. Thirty sera from the remaining fowls on the farm were negative. However, a psittacotic virus was obtained from the kidney of one fowl by mouse passage. The sera of the father, daughter and eldest son were positive at 1 in 4, and slightly at 1 in 8, but the youngest son's serum was negative. There was little doubt that these people had at least passed through sub-clinical infections. In this same article, Meyer referred to pigeons kept in crowded cages in a damp room in San Francisco. Eight died within a month; they were emaciated and had a fibrinous pericarditis and peritonitis; the spleens were swollen; the livers were enlarged and engorged and occasionally studded with small necroses. Smears of the exudate contained L.C.L. bodies and the sera of the pigeons were positive up to 1 in 128. S. typhimurium was also isolated from the birds. This pigeon strain was pathogenic for mice by the i.n. and i.c. and occasionally by the i.p. routes. When injected i.m., budgerigars sometimes died with typical lesions in seven or eight days. Meyer stated that the virus had been isolated from only 6% of positively reacting pigeons and so antibodies were probably indicative of a healed out infection. However, he recognised that the virus might have been found if other organs had been tested.

In 1941, Eaton, Beck, and Pearson reported on a virus from human cases of atypical pneumonia and compared it with the viruses of meningo-pneumonitis and psittacosis. They isolated the strain from four of six epidemiologically related cases in California. Of the six, three were nurses who contracted the disease from a patient, two dying. The patients first complained of influenza-like symptoms lasting two to six days and accompanied by gastro-intestinal signs and severe headache. Broncho-pneumonia appeared after several days. The temperature was usually high and continuous, and the pulse relatively slow. By the fourth i.n. passage through mice, the animals developed ruffled fur, sticky eyes, bubbling respiration and died within two to three days. Even after repeated i.p. passage through mice, no material ever killed by that route. The results of i.n. inoculation depended on the amount of virus administered. Sometimes only a few small, round, grey foci were seen. In mice dying acutely the lungs were completely consolidated, deep red and very oedematous, thereby suggesting infection with a parrot virus. Most mice succumbed in four to ten days after i.c. infection, with paralysis of varying degree preceding death.

Eaton, Beck, and Pearson compared their virus with that isolated by Francis and Magill. Upon the i.n. inoculation of mice both produced pneumonia without brain involvement and after i.n. injection both led to meningitis and paralysis of the hind legs without pulmonary disturbances. Meningo-pneumonitis virus, however, very often killed mice inoculated i.p. and recovered animals were carriers. The i.p. infection of guinea pigs produced serious illness and often death, but Francis's strain was almost harmless. The i.n. instillation of both strains caused the usual pneumonia in Syrian hamsters. Ricebirds injected i.m. were far more resistant to the Californian virus than to that of meningo-pneumonitis, even though many died. Mice inoculated i.p. with Californian virus subsequently resisted i.c., but not i.n. infection. A small i.n. or i.c. dose did not protect against a large dose given by the same routes, respectively. Although it was difficult to produce cross immunity to Francis' strain with the Californian virus by i.n. inoculation, the converse was not true; the same followed the use of the i.c. and i.p. routes. Antigenically, a relationship existed between the Californian and meningo-pneumonitis viruses, and that from parrots.

In 1942, Pinkerton and Moragues published an article devoted to a comparative study of the viruses of meningo-pneumonitis, psittacosis of pigeon origin and of psittacosis of parrot origin. Pinkerton's pigeon virus failed to evoke apparent illness when mice were inoculated i.p. and meningo-pneumonitis virus did not produce the hepatic necroses characteristic of classical psittacosis. Seven pigeons inoculated i.c. with parrot virus remained apparently normal. Nine pigeons receiving meningo-pneumonitis virus by the same route became ill in three or four days, lost their appetites and had ruffled feathers; the neck was retracted and opisthotonus developed; the stance was uncertain and the gait wobbly. Two of the nine pigeons died on the seventh day and three others were sacrificed on the eighth to tenth days, when moribund. Autopsy revealed meningitis and encephalitis and numerous L.C.L. bodies. Sixteen of eighteen pigeons inoculated i.c. with Pinkerton's virus died between the third and fifteenth days, while only three early deaths, one late death and one case of illness with recovery were seen in thirty-one pigeons injected with parrot virus. Thus the pigeon type of virus shows a significantly greater pathogenicity for pigeons when given i.c. Even pigeons with positive serum reactions will die in four to seven days after i.c. infection with pigeon virus.

Pinkerton and Moragues suggest that little notice should be taken of the slight immunological differences between the pigeon and classical strains, and the virus of meningo-pneumonitis, particularly as a solid immunity, even against homologous viruses in animals inoculated by the i.n. and i.c. routes, is rather uncertain. The same authors drew attention, again, to the fact that human psittacosis of pigeon

origin differs in no way from the disease contracted from parrots.

In 1942 Meyer, Eddie and Yanamura reviewed the subject of psittacosis in pigeons, adding additional information. They believe that all positive serum reactions of pigeons are indicative of exposure to the virus. In this connexion, it is interesting to mention that a lymphogranuloma venereum infection in man will lead to a false positive serum reaction for psittacosis, but positive pigeon serum has no effect whatsoever on the virus of lymphogranuloma. Pigeons with negative sera promptly generate psittacotic antibodies following the i.m. or oral administration of parrakeet or pigeon virus. The virus possesses a highly adapted latent parasitism for pigeons. It is uncertain how the virus escapes from the body of the pigeon, although suspicion is cast on the faeces and urine. Reference to this point will be made in the second half of this article, dealing with conjunctivitis in a pigeon. Infection apparently occurs in the nest or during the youth of the bird. Here it is worth while to note that Meyer found the ovaries of three parrakeets infected, and the yolk of an egg in the oviduct of one bird contained a weak virus. Recovery from this early contact with the virus ensures a life long relative immunity, frequently associated with latency. Crowding of the birds in a poorly-lighted, insanitary environment will easily disturb the balance of the immunity in favour of the virus, and then deaths from psittacosis ensue. Whether the Salmonellosis, which is so often present, helps to break down the resistance of the pigeon is problematical.

Parrakeets and ricebirds died as a result of ingesting and inhaling pigeon virus. Meyer, Eddie and Yanamura failed to produce symptoms in pigeons by feeding them with a pigeon virus, or by inoculating it i.m., but the birds harboured the infection in their organs for many weeks. Only recently-isolated parrot and parrakeet viruses (of avian or human origin) could kill even odd pigeons when injected i.c. Evidence exists that psittacosis may follow the indirect exposure of people to sick pigeons in parks and on plazas. According to these authors Tomlinson determined that the doves, Streptopelia decaocto and S. semitorquata, could become infected spontaneously with the virus of parrot or parrakeet origin.

In a further study by Meyer and Eddie in 1942, it was concluded that the New Jersey fowl virus was of the pigeon type. Its i.m. injection into fowls was followed by a chronic wasting disease, with latency in the spleen, liver and kidneys.

Meyer presented a full-length review on the ecology of psittacosis in 1942 and stated that the ubiquitous pigeon lofts constituted a larger reservoir of psittacotic virus than all the parrakeet aviaries combined. Latent pigeon infections were characterised by enlarged, dark, purplish, mottled or pale spleens and soft, greyish kidneys. Doves were more

susceptible than pigeons to the i.c. inoculation of parrakeet virus. The meningo-pneumonitis virus of Francis and Magill killed doves injected i.c. and ricebirds and parrakeets inoculated i.m. Exposing ten young chicks to contact with sick and latently infected parrakeets yielded two fatal infections—on the fifty-second and sixty-third days of the exposure period—and four latent infections characterised by enlarged spleens and liver necrosis. The virus is often to be isolated from the grossly normal kidneys of pigeons.

Eddie and Francis published a paper in 1942 on the occurrence of psittacosis-like infection in domestic and game birds in Michigan. Of ninety-eight pigeon sera, 61% gave a positive reaction, and the incidence of reactors in the different lots of birds varied from 8·5 to 85%. Sex, age and the size of the pigeon's spleen seemed to make no difference. Virus was recovered from the liver and spleen of one pigeon, and from the ovary and kidneys of another. Twenty-two of 109 domestic turkeys gave a positive complement-fixation reaction when the sera were diluted 1 in 4 to 1 in 16; nine of forty-five domestic fowls reacted at 1 in 2; five of twenty-four domestic ducks were positive at 1 in 2 to 1 in 16. No attempt was made to isolate any virus from the turkeys, fowls and ducks. No trace of infection was discerned in fifty-five wild ducks, twenty-five ringneck pheasants and ten Hungarian partridges.

Finally, in 1943, Andrewes and Mills reported the presence of psittacosis in pigeons in England. They found the disease in an unhealthy-looking batch of birds recently arrived from the United States of America, and in an apparently normal pigeon captured in Westminster, and in some other seemingly healthy subjects from a farm in Berkshire. The Westminster strain took up to fourteen days to kill mice inoculated i.c. All strains failed to produce symptoms when injected i.p. into mice.

Pertinent Points from the Literature.

From this review of the literature on psittacosis in man and in what might be termed domestic birds, a number of important facts emerge. It is most probable that human cases are much more common than is imagined. All instances of atypical pneumonia, particularly those not influenced favourably by the sulphonamides, should be held in suspicion. It must also be remembered that the infection in man may be productive of no symptoms, or at most "influenza." Human to human infections are common, and physicians and attendants should protect the eyes, nose and mouth by donning goggles and masks. The complement-fixation test will reveal a past infection, or a present infection that has been in existence for about a week. The lymphogranuloma venereum virus will upset the serological findings. The best way to demonstrate the virus in man is to inject mice i.p. with the unfiltered, centrifuged sputum mixed with broth.

If the mice fail to die, an emulsion of their spleens should be injected i.p. into more mice after three weeks and from this second lot splenic material should be inoculated i.c. into a third generation of mice after a further period of three weeks. If virus is present this last operation will be productive of the typical chorio-meningitis. Large numbers of pneumococci will kill the test mice and in some instances it has been necessary to use rice birds, when they have been available, in place of mice, for the initial enrichment of the virus.

The disease does not vary in man because the virus happens to be of pigeon instead of psittacine origin. The viruses isolated by Francis and Magill and also by Eaton, Beck and Pearson resembled essentially the pigeon type. This pigeon type readily kills pigeons injected i.c., whereas the psittacine types seldom do this. The classical psittacine strains from Central and North America kill mice inoculated i.p., whereas the Australian strains from parrakeets and cockatoos are far less pathogenic for them by the same route. Many pigeon strains seem to be innocuous for mice when administered i.p., but others will produce fatal infections in a few. Whatever the type of virus, the mouse is more uniformly susceptible to i.c. or i.n., than to i.p. infection.

The disease in pigeons obviously occurs on a very extensive scale. Latent infections are common, irrespective of whether the birds give positive or negative serological reactions. The virus has been isolated from the spleen, liver, kidneys, ovary and lungs. Whenever an outbreak of Salmonellosis is encountered extra caution is needed because of the very frequent association of this disease with psittacosis. Unless the pigeon is acutely ill, no L.C.L. bodies may be visible microscopically. Mice infected i.p. from them also commonly appear to be negative, as the bodies cannot be seen, even in smears from the swollen spleens.

Although the one spontaneous infection of a flock of fowls was due to a pigeon type of virus, it must not be forgotten that chickens can develop the disease after exposure to a psittacine type.

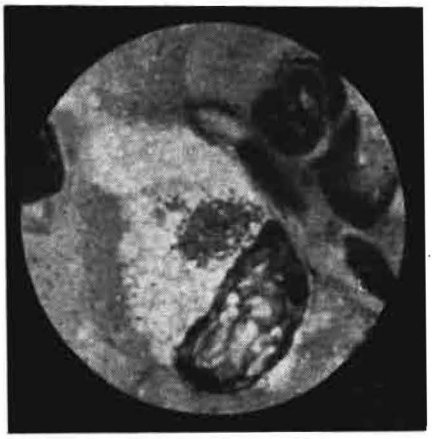
The serological evidence makes it highly probable that domestic turkeys and ducks may play a rôle in the dissemination of psittacosis to man.

Conjunctivitis in a Military Pigeon.

In February 1942 a carrier pigeon was brought in by the officer-in-charge of the pigeon services, who was a fancier before the war. He stated that the bird was suffering from a "cold in the eye," and that such watery eyes were well known in pigeon lofts.

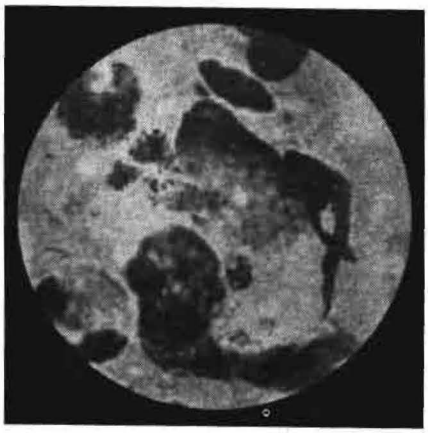
The left eye looked normal. Examination of the right eye revealed that the conjunctiva, particularly of the lower eyelid, was swollen and moist and moderately reddened. There was no discharge, however, and the cornea appeared to be unchanged. The pigeon was

FIG. 1.



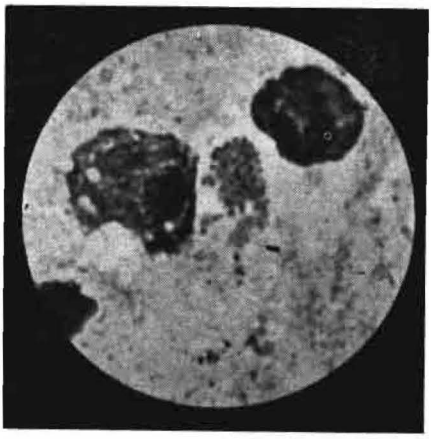
× 1800. Foam cell containing colony of elementary bodies.

FIG. 2.



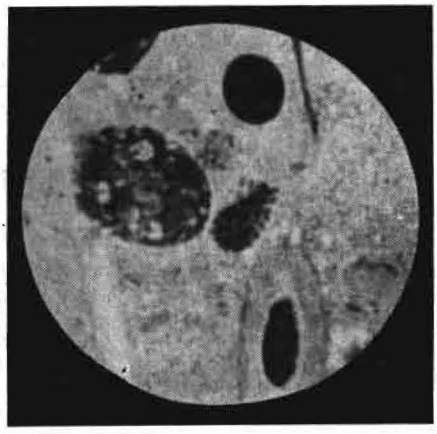
× 1800. Macrophagee with six colonies of elementary bodies. Most of the colonies show disintegration probably caused by the preparation of the smear.

FIG. 3.



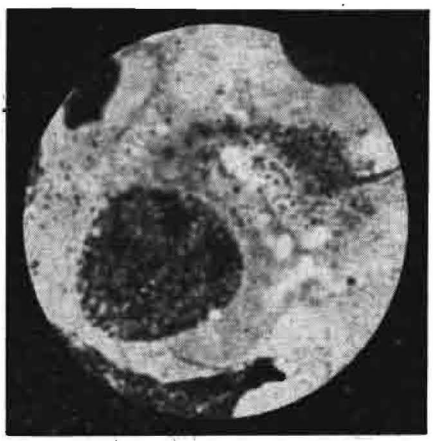
× 1800. Macrophage with a colony of initial bodies.

FIG. 4.



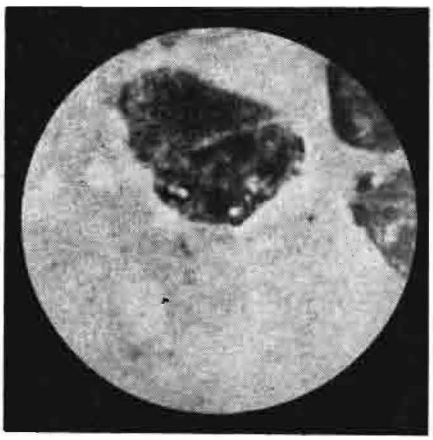
× 1800. Macrophage with a large compact colony and a small loose colony of elementary bodies.

FIG. 5.



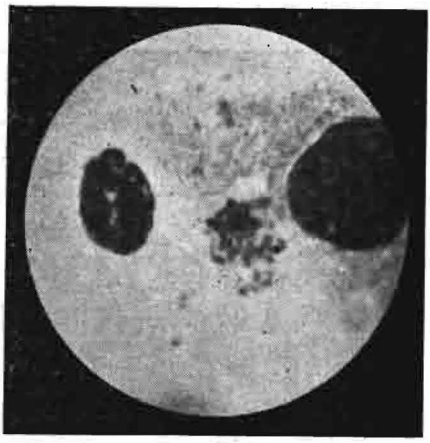
× 1800. Macrophage (foam cell) with a dispersed colony of elementary bodies.

FIG. 6.



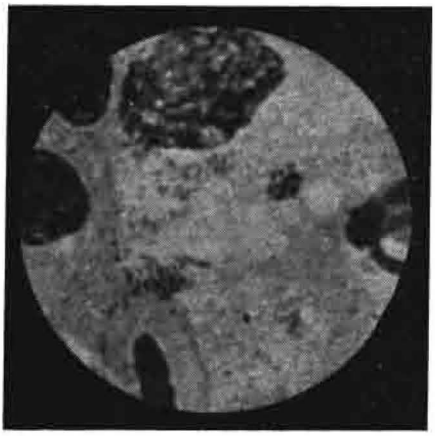
× 1800. Macrophage with a colony of elementary bodies capping its nucleus.

FIG. 7.



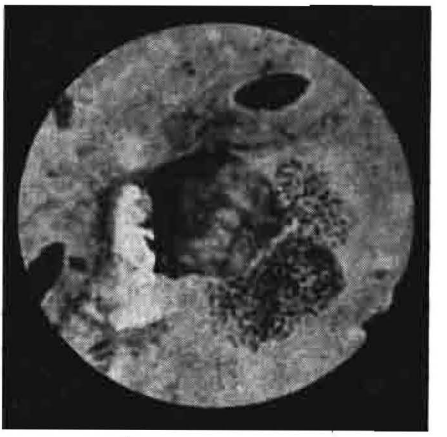
× 1800. A loose colony of initial bodies.

FIG. 8.



× 1800. Foam cell with a dispersed colony of elementary bodies and a small compact colony of bipolar staining initial bodies.

FIG. 9.



× 1800. A macrophage containing two overlapping colonies of elementary bodies.

All the above cells were in the conjunctival scrapings of the military pigeon. The author is indebted to Mr. T. Meyer of Onderstepoort for the preparation of the photomicrographs.

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in good condition and showed no sign of ill-health. Conjunctivitis was diagnosed and smears of conjunctival scrapings were prepared for staining and examination.

The bird was taken back to its loft, as it was desired to retain it. Unfortunately, the military authorities changed their minds and destroyed it before the precise nature of the complaint was ascertained. Thus it was impossible to conduct biological tests.

The smear of the affected eye was stained with Giemsa in the usual manner and a prolonged search for parasites revealed colonies of virus particles indistinguishable from L.C.L. bodies. All the colonies were located in macrophages, particularly the so-called foam cells, which are the cells most favoured by the virus of psittacossis Not a single infection of a conjunctival epithelial cell could be detected, and this most important observation precluded the possibility of the conjunctivitis being due to a new disease of the nature of trachoma or inclusion blennorrhoea. The smear also indicated a leucocytic infiltration of the conjunctiva.

These observations are of considerable practical importance, for psittacosis has never been known to affect the conjunctiva of any bird. If the virus of psittacosis does indeed find the eye a suitable habitat, we can easily explain why the L.C.L. bodies may occur in

the droppings and nasal secretions, while being apparently absent in the internal organs. A conjunctival infection would also constitute a source of danger to human beings, for very obvious reasons.

Summary.

The literature pertaining to psittacosis in domestic birds, such as the pigeon and fowl, has been reviewed. The importance to public health of these new strains of the virus has been emphasized. A case of conjunctivitis in a military pigeon, probably of psittacotic origin, has been described.

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EQUINE ABORTION.

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Onderstepoort. Cape Town.

Although infectious abortion is a well-known disease in Europe and America, it has not been previously described in South Africa. It is true that sporadic cases of abortion have been recorded from time to time, but so far no attempt has been made to study the aetiology.

Kilborne (1893) and Smith (1893) were the first to describe an epizoötic form of abortion in equines and they incriminated an organism of the hog cholera group as an aetiological agent. Later Good and Corbett (1913) and Good and Smith (1914) studied the disease in greater detail. They found that the abortions usually occur about the eighth month and they isolated an organism from the foetus and afterbirth which they called *Bacillus abortivo-equinus* and which they placed in the *enteritidis-cholerae-suis* group. By injecting mares intravenously with cultures of this bacterium they produced abortion in about 10 days' time and recovered the organism from the aborted foetuses. They found S. abortus-equi to be an important cause of joint-ill in foals. Similar results were obtained by workers like Schofield (1914), MacFadyean and Stockman (1917), Miessner and Berge (1917), Krage (1935), Watanabe (1937), and Oiguni and Koihutsu (1938). But this organism may also play an important rôle in the causation of other forms of suppuration and abscessation in the body. Thus Martinaglia (1929) isolated it from horses suffering from tendovaginitis following horse-sickness immunisation, and Fujimura and Hoshi (1936) obtained it from boils on the arm of a veterinarian, who became infected from handling the afterbirth of a mare that had aborted. Oiguni and Koihutsu (1938) described suppurative changes in the thoracic wall and scrotum of equines and traced the cause to abortus-equi.

The disease was also studied by workers like de Jong (1912) and van Heelsbergen (1914) in Holland; Murray (1919) in America; Miessner and Berge (1917), Krage (1935) and Stitz and Görkel (1914) in Germany; Saxer (1938) in Switzerland; Verge (1939) in France; by Fujimuri and Hoshi (1936), Hirato (1936), Watanabe (1937), Oiguni and Koihutsu (1938) in Japan, and by various others. Both von Heelsbergen and Murray showed by means of transmission experiments that abortion could be readily obtained by inoculating pregnant mares with live cultures by the intravenous route, but they

had negative results with mares that received the cultures by the mouth or per vaginam.

Murray (1919) found that the agglutination titre of healthy equines seldom exceeded 1 : 40, while Saxer (1938) regarded a titre of 1 : 100 as positive. But von Heelsbergen (1914), de Jong (1912), Good and Corbett (1913), Verge (1939), Hirato (1936), and other workers consider that the titre of normal mares may be as high as 1 : 200 or 1 : 300.

During recent years Dimock and Edwards (1936), Dimock (1940), Dimock, Edwards and Bruner (1940), Miessner (1938), Hupbauer (1938), and Manninger and Csontos (1941) have shown that serious outbreaks of abortion in mares may occur where the foetal organs and afterbirths were entirely free from cultivable organisms. They carried out several transmission experiments with pregnant guinea-pigs and mares, using both filtered and unfiltered foetal organ suspensions. By means of Seitz filtrates administered either per os or intravenously they succeeded in producing abortions both in guinea-pigs and in mares. The incubation period in the guinea-pig was found to be about 10 to 20 days and in the mare it varied from 20 to 34 days. These authors, therefore, concluded that the causal agent of these abortions was a virus. The affected mares did not appear to suffer appreciably from the abortions and the afterbirths were usually expelled spontaneously. Involution apparently occurred normally and the mare usually took the stallion about 8 days after the abortion. Mares which have aborted once usually carry their foetuses for the full term and deliver healthy foals during subsequent pregnancies. The lesions presented by the foetuses resembled those of a natural case of abortion.

The etiological agent is so highly contagious that as many as 90% of the pregnant mares on an infected farm may abort. Dimock and his co-workers consider that the infection may be conveyed by the stable attendant from an aborted foetus to a healthy mare and they have succeeded in transmitting the disease by stable exposure.

All these workers have found the following lesions in the aborted foetuses, which they regard as characteristic of the disease: Small disseminated necrotic foci in the liver, accumulation of a blood-stained fluid in the thoracic and abdominal cavities, haemorrhages in the spleen and on the epicardium, swelling of the spleen and liver, enlargement and reddening of the colic lymphatic glands and icterus of the subcutaneus, subserous and peri-renal tissues. Moreover, Dimock and his co-workers have demonstrated the existence of intranuclear inclusion bodies in sections made from the liver and lungs and particularly in the bronchioles and other air-tubes. In the liver the inclusion bodies are most easily found near the periphery of the necrotic nodules. It is considered that by finding inclusion bodies a diagnosis

of virus abortion can be made in cases where the pathological anatomy is not quite convincing. Sometimes an affected mare gives birth to a live foal which may die in a day or two showing characteristic lesions of equine abortion.

Prophylaxis.

Two aetiological agents of equine abortion have been described and both appear to be very highly contagious under natural conditions. Very stringent sanitary measures should, therefore, be applied for the prevention and control of both conditions in an infected stud. All the affected mares should be moved immediately after the abortion and isolated far away from the healthy ones, and handled by separate attendants. The afterbirth and foetuses should be very carefully disposed of. Attendants who have handled the foetus, afterbirth, or the affected mare should not come in contact with healthy mares. Infected stables should be thoroughly disinfected and not used for pregnant animals during the same season. The remaining pregnant mares should now be divided into small groups corresponding more or less to the stage of pregnancy reached. The advisability of always isolating mares that have reached an advanced stage of pregnancy as a routine measure in each stud is to be recommended.

The vaccination of pregnant mares about 2 to 3 months before parturition is due is recommended. In the case of *abortus-equi* infections, a formolised suspension of the organisms is generally used, and for virus abortion Dimock et al. recommend the use of vaccines prepared from the organs of aborted foetuses.

Although the mare may take the stallion soon after an abortion and deliver a healthy foetus at full term, Dimock and his co-workers consider it inadvisable to have the mare served before 3 to 4 months have elapsed after the abortion.

The evidence provided in the literature shows that there is a marked divergence of opinion among different investigators with regard to the agglutination titre of normal equines for *abortus-equi*. As pointed out above, some workers consider an agglutination titre of 1 : 100 as positive for *abortus-equi* whereas others regard a titre of 1 : 200 to 1 : 300 as normal. It is not clear whether "O," "H," or mixed antigens were used for these tests.

In order to determine the agglutination titre of normal South African animals of different species for *abortus-equi*, one of us (M.W.H.) tested several routine samples of serum from equines, cattle and sheep submitted to Onderstepoort for various purposes. Both "O" and "H" antigens were employed in these tests.

The results of these agglutination tests show that although the "O" titre of a few apparently normal equines may be as high as 1 : 40 the majority have a titre of not more than 1 : 10. Of 128

samples of horse serum examined, from different parts of the country, not one had a titre of more than 1 : 40. A small percentage of bovines and sheep also reacted up to 1 : 40, but the majority did not react at a dilution of 1 : 10. The " H " agglutinations were not appreciably higher than the " O." It was therefore decided to regard an " O " titre of 1 : 50 to be on the border-line; but if the " H " titre of the same animal is 1 : 200 and there is complete sedimentation of the "O" antigen at 1 : 50, the animal is considered to be probably a carrier. Henning and Haig (1939) have shown that for the detection of carriers " O " agglutination alone is of value. In the study reported below very definite " O" reactions were obtained with all the carriers detected, whereas the " H" agglutinations were seldom very marked.

In September 1942 a farmer in the Caledon district submitted for examination two samples of blood taken from two mares that had aborted. The one sample from mare labelled No. 4 was haemolysed but the other which was taken from mare No. 13 gave a strongly positive reaction with *abortus-equi* ("O" = 640 and "H" = 1280). One of us (C.H.F.) was asked to visit the farm and report on the outbreak. According to information obtained, the first abortions occurred in June and July, and during a period of about 10 weeks 27 out of a total of 34 pregnant mares had aborted, and two had given birth to healthy foals. Of the remaining five two mares (labelled Nos. 34 and 35) aborted later and three delivered healthy foals. Most of the abortions occurred between the 7th and 10th months of pregnancy, but three foetuses were dropped between the 10th and 11th month. Apparently the last three mares that were involved were off-colour for a few days prior to the abortions and all three died about 24 hours afterwards. The owner stated that the afterbirths were badly decomposed at the time of abortion and thought that the foetuses had been dead for some days before delivery. The result was that the mares developed septicaemia from which they died. A few of the other mares also had retained afterbirths and some showed a slight vaginal discharge. Apart from unthriftiness and loss of condition the rest of the mares had apparently not suffered much inconvenience as a result of the abortions.

There seems to be no doubt therefore that the cause of the abortions was an infection which was very virulent and highly contagious. Moreover, the conditions under which the mares were kept appeared to be exceedingly favourable for the transmission of the disease. The first abortions occurred in a paddock where the mares were running with a number of pregnant ewes. From there the owner transferred all the mares, the pregnant ones as well as those that had already aborted, to another paddock containing green oats. After a fortnight, however, further abortions occurred so that the owner decided to stable the mares. When the abortions continued in the stable the mares were moved back to the paddock, but still the abortions did

not stop. The owner, by failing to adopt suitable prophylactic and sanitary measures, created conditions which were highly favourable for the dissemination of the contagion. The aborting mares were allowed to run free with the pregnant ones, while the dead foetuses and afterbirths were left undisturbed in the paddocks where the abortions had occurred. The carcases of some of the dead mares and foetuses and some of the afterbirths were partially or wholly devoured by pigs running on the farm without any apparent harm to the latter.

Some of the pregnant ewes that were pastured with the mares aborted about two months before the first mare. In addition two pregnant cows that were placed in the infected paddocks with the mares aborted about two weeks later. The sera of all the ewes and both cows were tested, but all gave negative agglutination tests with *abortus-equi.*

There was no previous history of abortion on this farm nor in the neighbourhood. The infection apparently made its appearance like a bolt from the blue and its source has remained obscure. There is also no record of cases of fistulous withers, poll-evil, joint-ill, or other forms of suppurative disturbances in equines. However, one donkey stallion, Alexander (No. 44), was suffering from a painful swelling of the near testicle. As the serum of this donkey gave a distinctly positive agglutination reaction with *abortus-equi* it is possible that the swelling was the result of an infection with *abortus-equi.* As all the adult equines had been tested for dourine with negative results this disease can be excluded as an etiological agent.

Since the outbreak, all the equines on the farm have been bled from time to time for the agglutination test with *abortus-equi.* The results of these tests showed that a large number of mares that had aborted gave " O " agglutination reactions under 1 : 50. These animals were, therefore, probably free from *abortus-equi* infection. The first test was applied on September 22nd, about 3 months after the abortions had commenced to occur. With this test, mare No. 2 (which had aborted on June 15th, 1942), gave a negative reaction, while mare No. 20 which aborted about 2 weeks later gave a distinctly positive reaction. On the whole, however, the animals reacted very irregularly. An animal which has given a distinctly positive reaction with one test may give a doubtful reaction subsequently, or an animal which has given a doubtful reaction with a previous test may subsequently react positively. A single test, therefore, cannot be relied upon for the detection of all the infected animals; it is only by repeated tests at 2 to 3 months intervals that it is possible to pick out the majority of carriers.

In addition to the adult equines a number of yearlings and foals have also been tested. Of these at least four (Nos. 54, 56, 62 and

68) gave well-marked "O" agglutination reactions. The dam of No. 54 died before the application of the test was started; No. 56 is the foal of a mare which gave three apparently negative and one suspicious reaction; No. 62 is the progeny of a mare which also was found to be negative with three tests and suspicious with the fourth; No. 68 was bought in October 1942. The titre of the donkey stallion (No. 44), which was positive with several tests, and of the Hackney stallion (No. 43), which was either negative or suspicious, rose markedly after inoculation with formolised vaccine.

The unreliability of a single test is most strikingly shown in the case of mares 34 and 35. Both of these gave negative agglutination reactions on September 22nd, before abortion. Mare 35 aborted on September 27th and her serum was tested 12 days later when she gave a distinctly positive reaction ("O" = 320 and "H" = 640). When mare 34 aborted on October 19th, her serum was still undoubtedly negative; but about a week later it had become positive ("O" = 320 and "H" = 640). These are the only animals from which foetal organs and afterbirths were available for bacteriological study. When cultures were made, *abortus-equi* was readily obtained from the foetal organs and afterbirths derived from both mares.

Discussion.

An outbreak of infectious abortion in equines is described. Only two foetuses were available for examination and *abortus-equi* was readily obtained from the internal organs and foetal membranes of both. The sera of the dams of both foetuses were tested a few days to a few weeks before and again after abortion. The titre obtained was so low before abortion, and in the case of one mare 24 hours after premature birth, that the reactions were regarded as negative. The sera of both mares, however, reacted strongly positive about a week after abortion and remained positive for several months. The "O" agglutination reaction of normal equines did not exceed 1 : 40 in any of the animals tested and an "O" titre of 1 : 50 is regarded as probably positive, provided there is complete sedimentation of the "O" antigen. For the detection of carriers, "O" agglutination alone can be relied upon. But as irregular reactions may be obtained with some mares that have aborted, it is recommended that repeated serological tests be performed at intervals of about 2 months. In this way animals that have given an indefinite reaction at a previous test may later be found to be distinctly positive, or they may give a number of reactions which can be regarded as negative. Repeated tests carried out over several months have shown that a number of mares that have aborted may continue to give negative reactions. These mares are being kept under observation and any further information obtained will be published at a later date.

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TRANSFERS OF OFFICERS.

The following movements of officers in the Department of Veterinary Services have occurred since the March issue of the Journal:—

Mr. D. E. Truter appointed G.V.O., Estcourt — 25/3/43.

Mr. T. N. Osborn appointed G.V.O., Dundee — 8/4/43.

Mr. P. L. Uys appointed G.V.O., Dundee — 10/4/43.

Mr. T. N. Osborn transferred from Dundee to Vryheid — 20/4/43.

Mr. J. P. Moll transferred from Umtata to East London — 15/4/43.

Mr. V. Cooper transferred from Johannesburg to Prudential House, Pretoria — 1/5/43.

Mr. J. S. Watt transferred from Pretoria to Johannesburg — 1/5/43.

SALMONELLA ENTERITIDIS, VAR. DUBLIN, INFECTION IN ADULT CATTLE.

G. BISHOP, W. SCHATZ, and A. S. CANHAM.
Allerton Laboratory.

The view is widely held that *S. enteritidis* var. *dublin* assumes its greatest importance in connection with paratyphoid in calves. While one may readily agree with this opinion, it must be pointed out that the significance of this organism in adult cattle has not been sufficiently investigated.

Henning (1939) has comprehensively reviewed the literature on paratyphoid of calves and adult cattle. From this review certain important facts emerge. Children and adults have on more than one occasion become infected after drinking milk from cows that, although apparently clinically healthy, were excreting Gaertner's bacillus in the faeces. It is admitted that the name *B. enteritidis Gaertner* frequently includes a number of closely allied serological types. That members of this group cause disease in both animals and man is recognized. Smith and Scott (1930) studied organisms classed as *B. enteritidis Gaertner* and found they belonged to the *dublin* type. These facts assume great significance in Natal in connection with the recent enquiry that was held into an extensive typhoid outbreak which occurred some months ago in Durban. Karsten (1933) has emphasised the danger arising from cattle that continuously excrete *S. enteritidis var dublin* in their faeces. Henning (in a personal communication) has stated that, although it is undoubtedly possible that chronic carriers exist, workers at Onderstepoort, who are constantly searching for such cases, have up to the present failed to detect such carriers.

That outbreaks of paratyphoid do probably occur in adult cattle in this country is shown by the following cases. On a farm in the Pietermaritzburg district a farmer recently bought seven cows from an outside area. These had not long been on the farm before one of them developed a marked diarrhoea. This became more intense, and, in spite of treatment, the cow wasted away and died. Within a few days a second cow from this group showed the same symptoms and the suspicion of Johne's disease was aroused as the animal remained alive for some time, gradually wasting away. This cow also died in spite of energetic treatment. Post-mortem examination of this case did not confirm the diagnosis of Johne's disease. A third similar case being reported, two of the authors (A.S.C. and W.S.) went to the farm some time after the onset of the disease. As the cow was in extremis,

blood was taken from the jugular vein and the animal was then destroyed. At post mortem a fairly heavy fluke infestation of the liver was seen, together with cavernous angiectasia of this organ, and in addition a few small whitish irregular areas were present on the surface of the liver. The intestinal mucosa was reddened almost throughout its length, some parts more so than others. As there was at the time no suspicion of paratyphoid, no material was taken for pathological examination, nor were cultures made for confirmatory diagnosis. The serum on being tested against *S. enteritidis var. dublin* antigen gave a presumably positive reaction in a 1:80 dilution and a similar reaction in 1:40 with *S. pullorum* antigen. The remaining four cows from this group were then blood-tested against *S. enteritidis var. dublin* antigen. One gave what appeared to be a positive reaction in 1:20, one a trace in 1:20, while the remainder were negative. Control tests were in order. It may be worth mentioning that this farmer had had cases of a similar nature the previous year, and had regularly failed to inoculate his young calves against calf paratyphoid in spite of continual losses from this disease.

In a review by Jansen (1939) of work carried out during 1938 at the Rijks Universiteit at Utrecht, he mentions an acute case of *S. enteritidis var. dublin* infection in a bovine in which the serum gave a positive reaction to *S. pullorum* antigen, and in which the organisms were found in the faeces. He points out the importance of such a finding and draws attention to the possibility that such recovered animals could play an important role in setting up paratyphoid in calves. He goes further, recommending that this disease should be controlled by veterinary regulations and suggesting that where paratyphoid breaks out in calves the blood of adult animals should be tested to detect carriers. He also mentions an acute outbreak of *S. enteritidis var. dublin* on a farm where a number of animals died. Blood was taken from six contacts on this farm and positive agglutinations of the blood with *S. pullorum* were obtained. He deduces that the animals had suffered from paratyphoid. In this last observation he points out that the practising veterinary surgeon has a very useful weapon, in *S. pullorum* antigen, for diagnosing paratyphoid in cattle.

In the review of Henning no mention is made of abortion in adult cattle as a result of infection with *S. enteritidis var. dublin*. *S. abortus bovis* is known to cause abortion in cattle, but this organism has not yet been encountered in South Africa. *S. enteritidis var. dublin* infection in adult cattle generally manifests itself as a septicaemia. Is it not possible that in animals with a latent infection, organisms may at times gain access to the blood and become distributed to various parts of the body without necessarily proving fatal to the host ? Should this be the case, then the gravid uterus of a cow may well become infected as a result of a bacteraemia of this nature.

Olsen (1938), in a review of revelant literature, records several cases of *S. enteritidis var. dublin* infection, causing diarrhoea, abortion, and death in cows and death in foetuses and young calves in Sweden.

Craig (1937) mentions that bacilli of the colon typhoid group have been recovered from the membranes, uterine discharges, and foetus in isolated cases of abortion.

In October, 1941, a fresh 7½ months old foetus was brought to the laboratory for examination. Unfortunately the placenta was not available for study. No thorough examination of the whole foetus was made, but a subcutaneous sero-sanguineous oedema and effusion of red fluid in the peritoneal cavity were noted. The contents of the abomasum were of a mucoid, flocculent nature and the mucosa was slightly reddened. Hanging-drop preparations of the abomasal contents revealed the presence of motile bacilli, but nothing in the nature of Trichomonas, Vibrio or organisms of the Brucella type was observed. These bacilli were Gram negative. Cultures from the contents of the fourth stomach were made on serum-agar and brilliant green-agar. After about fifteen hours' incubation an opaque growth was observed on the media. Smears made from the cultures showed the culture to be pure. The organism produced acid and gas in maltose, glucose, and dulcite, but not in lactose and saccharose. At the same time two guinea-pigs were inoculated subcutaneously with dilutions of abomasal contents and sterile saline in equal parts. Five days after inoculation both guinea-pigs were bled and their sera were found to agglutinate both Pullorum and Gallinarum antigens. Serum from a guinea-pig not inoculated against the same antigens was negative. Seven days from the date of inoculation, both guinea-pigs died. Post-mortem revealed signs of necrosis at the seat of inoculation and definite evidence of septicaemia was observed in both animals. Cultures made from the hearts, spleens and kidneys yielded organisms which were morphologically and serologically identical with those obtained from the foetal stomach. They were Gram-negative bacilli and gave the same fermentation reactions. The cultures from both guinea-pigs were sent to Onderstepoort for identification and were typed as *S. enteritidis var. dublin.*

The original stomach contents (which had been retained in the ice chest for seventeen days) were then transferred to the incubator for a short while. As the bacteria were still actively motile, it was decided to inoculate two more guinea-pigs. One received 2 c.c. of diluted stomach contents intraperitoneally and the other 2 c.c. subcutaneously. The former died two days later from peritonitis and it was again possible to isolate the organism in pure culture from the organs. A week after inoculation, blood was drawn from the heart of the surviving guinea-pig and the serum was found to agglutinate both Pullorum and Gallinarum antigen in dilutions of 1 : 80 and 1 : 20 respectively. This guinea-pig was killed *in extremis* two weeks later and the organ-

isms which were isolated from its organs corresponded in all respects (motility, staining and fermentation reactions) with those which had previously been isolated from the other guinea-pigs and from the foetus. Although one must admit the possibility of natural infection of guinea-pigs with strains of Salmonella, it is rather unlikely that one would encounter, within such a short period, four guinea-pigs all infected with *S. enteritidis var. dublin.*

The owner of the cow which had aborted was asked to submit samples of blood from this particular cow and any others which had aborted. Five samples numbered 1 to 5 were sent to the Laboratory. The first three were from animals which had aborted during the last two or three months of gestation and the remaining two were from heifers with a history of temporary sterility. The cow from which the foetus was obtained (No. 3) was said to be showing a copious vaginal discharge. It is interesting to note that the owner had never used paratyphoid vaccine on his farm and that the animals which had aborted were all primiparae.

The sera from the above cattle were tested against a Dublin and Pullorum antigen with the following results :—

S. enteritidis var. dublin.

No.	1 10	1 20	1 40	1 80	1 160	1 320
1.	+	—	—	—	—	—
2.	—	—	—	—	—	—
3.	++	++	++	+	—	—
4.	++	—	—	—	—	—
5.	+	—	—	—	—	—

Negative Controls: Negative.
Positive Controls : 1 — 40.

S. pullorum antigen.

No.	1 10	1 20	1 40	1 80	1 160	1 320
1.	++	—	—	—	—	—
2.	—	—	—	—	—	—
3.	++	++	+	+	—	—
4.	+	—	—	—	—	—
5.	++	—	—	—	—	—

Negative Controls: Negative.
Positive Controls: 1 — 40.

Sera from these animals were then sent to Onderstepoort and agglutination was found in the following dilutions with 'O' and 'H' Dublin antigens.

No.	'O' antigen	'H' antigen
1.	25	50
2.	0.	0.
3.	50	200
4.	0.	0.
5.	0.	0.

The agglutination test of all five samples for bovine contagious abortion was negative.

Since then many samples of sera from cattle sent to the Laboratory for the contagious abortion test have been utilised for testing for the presence of paratyphoid infection. In one particular group of samples, numbering 41, from the Estcourt district, seven of the samples gave what appeared to be positive reactions to *S. pullorum* antigen in dilutions of 1:20. The Government Veterinary Officer informed us that two of these cows had aborted. As the farmer had not kept detailed notes of all his cases of abortion, it is probable that other animals,

giving apparently positive reactions, may also have aborted. These forty-one samples were all negative for contagious abortion.

When considering an organism as a potential cause of abortion another consideration presents itself, namely the possibility of a prenatal infection. That certain calves may be born infected from carrier mothers seems to be not impossible, in view of the fact that a number of calves develop the typical paratyphoid diarrhoea and die within a week of birth, in spite of having been vaccinated on the fuorth or fifth day.

Since carrier animals tend to lose the infection after a time, we are inclined to suggest that the incidence of paratyphoid abortions, however small, is greatest in primiparae.

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PRESS NOTE.

We have been informed by the Director, Maybaker (S.A.) (Pty., Ltd.), that Dr. Arthur James Ewins, Director for Research for Messrs. May Baker, Ltd., London, has been elected Fellow of the Royal Society. This honour has been conferred upon him in recognition of his achievements as leader of the May and Baker team responsible for the discovery of M. and B. 693 (Sulphapyridine), M. and B. 760 (Sulphathiazole) and the new group of amidine compounds which are being successfully applied to the treatment of various protozoal injections.

TREATMENT OF CANINE COCCIDIOSIS.

B. S. PARKIN.
Onderstepoort.

In the Pretoria district coccidia are occasionally found during the course of the routine examination for worm eggs of the faeces of dogs, which do not show symptoms indicative of coccidiosis. Clinical cases of coccidiosis are apparently of infrequent occurrence in this area. But recently a number of such cases have been encountered and, as these cases have been successfully treated, it is considered advisable to place the results on record, so as to enable veterinarians in the Union and elsewhere to undertake proper trials of the treatment. This is the more advisable because no efficacious therapy has hitherto been introduced. A brief description of the disease will also be included.

In the dog, four species of coccidia have been described. Three of them belong to the genus *Isospora* and one, which is rare, to the genus *Eimeria*. In Isospora there are two spores, each containing four sporozoites, whereas in the *Eimeria* there are four spores, each with two sporozoites. The genera are identified by the development observed in the oöcysts when these are maintained under favourable conditions.

The infective stage of the parasites is the sporulated oöcyst which generally develops outside the animal's body after the oöcyst has been evacuated with the faeces. Dogs are infected by the ingestion of the sporulated oöcyst. The sporozoites are liberated from the cyst into the lumen of the intestine and penetrate into the epithelium, where development continues. Merozoites result which are capable of asexual development. Ultimately some of the merozoites develop into micro- and macro-gametocytes forming the sexual stage and resulting in the production of the oöcysts.

The three species of *Isospora* found in the dog are: *I. bigemina*, which is the smallest, *I. rivolta*, and *I. felis*, which is the largest. The differentiation of the species is based on the size of the oöcysts.

In general the coccidia show a definite host-specificity, but it has been proved that the cat is susceptible to *I. felis* and *I. rivolta* of the dog. The ingestion of small doses of coccidia may result in the development of an immunity, which however is only specific against the particular species of coccidium concerned. The greater resistance to coccidiosis sometimes observed in older animals is probably due to the previous development of immunity by the ingestion of sporulated oöcysts insufficient in number to produce symptoms of the disease.

The sporulated oöcyst, the infective stage, is extremely resistant to the action of chemicals and it is a waste of time to disinfect kennels for the purpose of destroying the oöcysts. They are, however, detrimentally affected by desiccation, putrefaction, and high temperatures. Consequently, dry or moist heat, drying, fermentation of faeces and litter, etc., are utilized in the decontamination of quarters. Measures such as the destruction of infected animals or their isolation, rotation of accommodation, early disposal of faeces etc. are applied in the elimination of the source of infection i.e. the faeces of the animal. The destruction of the source by the sterilization of the infected animal with drugs is a method of control of primary importance, requiring only the discovery of efficacious therapy.

In severe forms of canine coccidiosis the symptoms are marked depression, lassitude and weakness, reduced or capricious appetite, profuse diarrhoea with offensive, watery and at times blood-stained evacuations, and rapid loss of condition.

In all the clinical cases referred to hereafter the occurrence of coccidia in large numbers in the faeces and the above-mentioned symptoms (with the occasional exception of blood in the faeces) were in evidence. The flotation method of examination of the faeces was used. Glycerine was mixed with the faecal emulsion in equal amounts. The preparations thus obtained in 50 per cent. glycerine may be subjected to prolonged examination as they do not dry up. The development of the two spores was frequently seen in such preparations, especially when they were maintained at 44°C.

A very impressive list could be compiled of the drugs which have been used in the treatment of coccidiosis of the dog. Among these there is no drug which has any definite specific action against the parasite. Sulphaguanidine, one of the most recently tried, was ineffective.

In the following description of the trials in therapy, three cases are recorded to illustrate the changes made in the treatment from the first to the third case, when the treatment was considered to be established and highly efficacious.

Case 1. — A six months' old Cocker Spaniel weighing 6.5 Kg. had a history of diarrhoea over the previous two months. It showed marked depression and emaciation and its appetite was capricious. Faecal examination gave a diagnosis of *I. bigemina* very frequent, and *I. felis* rare. On the 1.3.32 it was given an enema of 0.5 g. of sodium sulphanilyl sulphanilate (S.S.S.) and 2 g. kaolin in 50 c.c. of water (i.e. about 7.7 c.c. of 1 per cent. solution of S.S.S. per Kilo) and in addition 0.75 g. of Uleron (4-(4-amino-benzol-sulphonamido)-benzol-sulphon-dimethylamide) *per os.* For the next three days it was given a daily dose of 0.75 g. of Uleron. Within 48 hours the diarrhoea had ceased and a marked general improvement was evident. No oöcysts

were detected in the faeces in the four faecal examinations made between the 7th and the 19th days after the commencement of the treatment.

Case 2. — Faecal examination of a two year old Dobermann-Pinscher of an estimated weight of 15 Kg. showed *I. bigemina* to be very frequent. The dog was in poor condition, very depressed, not feeding, and had diarrhoea. It was given an enema of 1 g. of S.S.S. and 2 g. of kaolin in 100 c.c. of water. This enema was repeated after 24 hours. In addition 1 g. of Uleron per os was given daily for three days and 2 g. of kaolin was fed to the dog daily for 10 days. Improvement was noticeable within 24 hours, and recovery was uneventful. Faecal examinations on the 8th day and after 3 months gave negative results for oöcysts. In this case the S.S.S. was given at the rate of about 6.6 c.c. of a 1 per cent. solution per kilo. (The diagnosis and treatment of this case was carried out by H. O. Mönnig).

Case 3. — The faeces of a four year old Scottish Terrier weighing 9 Kg. showed *I. bigemina* to be very frequent and *I. felis* rare. The dog, while under observation for three days before treatment was instituted, showed inappetence, rapid loss of condition, and diarrhoea. It was given, daily for three days, an enema of 50 c.c. of 2 per cent. solution of S.S.S. (i.e. about 5.5 c.c. of a 2 per cent. solution per kilo.) and on the third day of treatment 0.75 g. Uleron. Faecal examinations made on the three days of treatment were positive, but one made on the second day after cessation of treatment was negative.

Discussion of the cases. The number of enemas given in these cases were: 1, one; 2, two, and 3, three. The oral administration of Uleron was: in case 1 for 4 days, in case 2 for 3 days, and in case 3 on one day. As the faecal examination in all three cases became negative for oöcysts within a few days after the commencement of treatment, it would appear that the efficacy was due to the enemas of S.S.S. and not to the Uleron given *per os.* The inclusion of Uleron may possibly have been a useful adjuvant, but the dose given in case 3 was so small that it is unlikely that the course of the disease was influenced by it.

The earliest negative faecal examination was obtained at the end of the 4th day. It is possible that the first of the enemas given was the one that produced the beneficial results and that the oöcysts found subsequent to the initial treatment were produced before the institution of the treatment and were retained in the intestinal lumen.

Kaolin was originally added in the belief that it would assist in the longer retention of the enema, but in case 3, in which no kaolin was given, the enemas were satisfactorily retained.

The actual bulk of the enema is also probably of importance in the treatment, for it must be sufficiently large to reach the duodenum. In none of the cases was vomiting produced, the largest enema being at

the rate of 7.7 c.c. per kilo. Probably it would be unsatisfactory from the therapeutical point of view for the enema to be so large that vomition or expulsion per anum resulted.

The number of cases treated is insufficient to determine the minimal effective dose of S.S.S. The largest dose of S.S.S. given at one time was 1.1. g. per 10 kilos of live weight and the smallest about 0.6 g. per 10 kilos. Probably the dose could with safety, but unnecessarily, be increased to 2 g. per 10 kilos. The possibility of intoxication from such a dose even if repeated, is small.

The Treatment of Further Cases.

With the information obtained from the above three cases, a routine method of therapy was devised. This routine method has since been employed in the treatment of twelve cases of clinical coccidiosis, with success in all except one. This exception was a case of coccidiosis which was complicated by distemper. The routine treatment is 10 c.c. of a 1 per cent. solution of S.S.S. per kilo as an enema, repeated in 24 hours. In no case have any symptoms of intoxication or vomition been observed, and the retention of the enema has been satisfactory. After the administration of the enema the tail of the dog is held down over the anus for about five minutes to prevent immediate expulsion of the enema, but the dogs did not show a tendency to do this and many dogs have retained the enema for 24 hours, even though rectal tenesmus had often been in evidence prior to treatment.

Definite improvement in the habitus, the appetite and the state of the faeces was apparent, usually within 24 hours and always within 48 hours. Usually by the end of the fourth day the faeces were more or less normal.

In most cases the oöcysts were absent, or present in very small numbers by the sixth or seventh day, the decrease in number being obvious by the fourth day. In several of the cases the opportunity occurred of doing a faecal examination some months after recovery, but in no case were the same oöcysts found. In one case an infection with *I. rivolta* occurred after recovery from *I. bigemina* infection.

One case of coccidiosis in a cat was successfully treated, the dose and amount of the solution being the same, proportionately to weight, as that used in the dog.

Summary.

1. A number of cases of coccidiosis due to parasites of the genus *Isospora* are described.

2. A highly efficacious treatment was found to be the administration as an enema of 10 c.c. of 1 per cent. solution of sodium sulphanilyl sulphanilate per kilo body weight.

Reference.

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THE ASSOCIATION.

COUNCIL MEETING HELD AT POLLEY'S HOTEL, PRETORIA, ON 7TH APRIL, 1943 AT 5.30 P.M.

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

Apologies for absence: Dr. P. S. Snyman and Capt. J. L. Dickson. The latter was represented by Mr. V. Cooper.

Obituary: Referring to the loss at sea of Maj. A. M. Howie the President said that this news was received with deep feelings of regret by every member, and suggested that a letter of sympathy be sent to Mrs. Howie.

(1) *Minutes of meeting held on 22nd September, 1942,* were taken as read and were confirmed.

(2) *Arising from these minutes*:

(a) *Hormone Treatment*: The President stated that the court case which was considered at the last meeting was still proceeding.

(b) *National Health Services Commission*: The Secretary reported that a committee consisting of Drs. du Toit, de Kock, Fourie, Col. v. Heerden and the Secretary had submitted a memorandum on "The role of he veterinarian in a national health service" to the above Commission and that the members of the Committee had also given evidence before the Commission on the 14th December, 1942.

(3) *Student's Loan Fund*: An application from a third year student was considered. Since the rules do not permit the granting of loans to students in the third year, except in exceptional circumstances, it was decided that this application be not granted.

(4) *Standing Committees*:

(a) *Book Fund*: It was decided that the necessary amendment to the Constitution be proposed at the next General Meeting, in order to create a separate Committee to administer the Book Fund, Dr. Thomas and Mr. Haig to form a temporary committee in the interval. Council further resolved to place on record its appreciation of the services rendered to this fund by Dr. Thomas and Messrs. M. H. V. Brown, de Boom and Haig.

(b) *Editorial Committee*: A letter was submitted from a member protesting against the publication of an article. This matter had been satisfactorily dealt with by the Committee concerned and it was decided that no further action be taken.

(5) *General Meeting Resolutions*: The following motions adopted by the General Meeting and referred to Council were considered :—

(a) By Dr. M. W. Henning "That in future no member be required to pay any further subscription after he has been a member of

the Association for 24 years." After discussion, the principle of this resolution was adopted, but Council favoured an extension of the period to 30 years. Dr. Thomas and the Secretary were asked to draft the necessary proposal for the next General Meeting.

(b) by Mr. J. G. Boswell "That a referendum be held as soon as possible on the desirability or otherwise of adopting the courtesy title of Doctor." After full discussion a committee consisting of Mr. J. G. Boswell, Dr. P. S. Snyman, Dr. D. G. Steyn and the Secretary was appointed for the purpose of obtaining the views of members, and forwarding a summary of these with the voting papers to all members.

The meeting adjourned for dinner at 6.50 p.m. and restarted at 8 p.m.

(6) *East Coast Fever*: The President stated that since publication of the news that a large number of cattle were to be slaughtered in Northern Natal he, in his capacity as President of the S.P.C.A., had been inundated with enquiries. Some statements made by him had been taken up by the lay press, and in this manner the Association was also mentioned. His object in putting this question on the agenda was to show that 3-day dipping was successful in eradicating East Coast fever in Southern Natal, and also to ascertain if the slaughter policy is to be applied generally in East Coast fever outbreaks. This would cause a lot of dissatisfaction.

Dr. du Toit explained the method of slaughter, which would not involve any more cruelty than that obtaining in any abattoir. The meat was to be inspected, graded, and used for human consumption. The newspapers responsible for the present agitation have overlooked the fact that the Natal Agricultural Congress, three years ago, passed a unanimous resolution asking the Government to adopt the slaughter policy whenever suitable in East Coast fever outbreaks. By the wholesale adoption of this policy, Portuguese East Africa has wiped out East Coast fever and has never had a recurrence. In reply to the President, Dr. du Toit said that the slaughter policy would not be applied generally, but only where circumstances warranted it and provided the stock owners are agreeable.

Col. van Heerden said that the decision to slaughter was taken in Vryheid in consultation with the farmers only on 29th March, that is, after the agitation had been launched by some sections of the lay press. Slaughtering was entirely on a voluntary basis and this will enable many farmers to obtain something from the wreck, some of whom had already lost practically all their cattle. Col. van Heerden stated that dipping was not always 100% successful and quoted cases of reinfections occurring repeatedly on farms on which the disease was supposed to have been eradicated by dipping.

After further discussion it was unanimously agreed to proceed with the next item on the agenda.

(7) *Post War Employment*: The Secretary submitted two letters from a member suggesting that the Council should take the necessary steps to see that those veterinarians who enlist after qualifying should not be handicapped by the fact that they were not in the Government Service prior to enlistment. It was decided that representations be made requesting the Department to regard the date of enlistment as the date of joining the

Service, for the purpose of determining the length of service, seniority, and salary grade. Drs. Alexander and du Toit were appointed to frame the necessary resolution.

(8) *Veterinary Work by Chemists*: Witwatersrand branch had made a complaint to the effect that veterinary work was still undertaken by chemists in areas served by veterinary pratitioners. It was decided that, pending the receipt of concrete examples, nothing could be done.

(9) *Availability of Drugs*: Some members have complained that they could not get essential drugs because the agents are unable to get import permits. It was agreed that such practitioners be asked to submit their case to the Secretary.

(10) *General*: *Tuberculosis*: The following resolutions which were unanimously passed by a meeting of the Witwatersrand branch were submitted:—

(a) Rand veterinarians are displeased at the Government's present policy with regard to eradication of bovine tuberculosis.

(b) the present scheme is not acceptable to dairymen and is inapplicable under the existing conditions of marketing of milk.

(c) No progress is being made.

(d) The meeting was of the opinion that data is lacking regarding the extent of infection in herds: such information as is available is based upon tests carried out in specialised areas or on specialised herds; and that a comprehensive survey should be carried out at the earliest opportunity.

(e) Many progressive farmers are interested in the eradication of the disease but they are unwilling to take any steps in the matter because, under existing regulations, a preliminary pilot test is not permissible.

(f) The meeting is of the opinion that an opportunity should be offered to farmers to carry out a pilot test without prejudicing their interests in any way. Safeguards may if necessary be introduced to prevent abuse of the pilot test.

It was decided that this matter be placed on the agenda for the next meeting and that a statement from the Director of Veterinary Services be obtained in the meantime.

The meeting concluded at 10.20 p.m.

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

ROLL OF HONOUR.

MAJOR ADRIAN MORRISON HOWIE, O.B.E. (1889 - 1943).

Members of the profession were undoubtedly appalled and shocked to a profound degree, when it was learned that our esteemed colleague had lost his life as a result of enemy action at sea, during February.

The sad news was received, by the writer, with a singular degree of poignancy, as, on the day prior to embarkation, he had had the privilege

of entertaining Adrian Howie, and it seems inconceivable that a life-long friendship should be ended in such a short time.

The late Major Howie qualified in Glasgow in 1910. He spent a year or two in practice, and was then offered a Government appointment in South Africa.

During his official career he had been stationed in Umtata, Lydenburg, Greytown, Estcourt, Cape Town and East London; and whilst it is, perhaps, safe to say that most of his time was devoted to the very complex problems involved in an East Coast fever eradication campaign, his experience was of a very varied nature.

In 1914, at the outbreak of the first Great War, he was among the first to join the newly-formed S.A. Veterinary Corps. He served during the rebellion and in the South West African campaign. At the successful conclusion of the latter, he proceeded with the expeditionary force from South Africa to what was then German East Africa. Again, at the conclusion of this latter campaign he applied for permission from the head of his civil department to volunteer for service in Europe, but this was refused. Howie immediately resigned his civil appointment, and this action, in the writer's opinion, is indicative of his true character, viz., his singleness of purpose to do a job of work which he conceived to be right.

For his military work he was mentioned in dispatches, and was awarded the O.B.E. (Military Division). When the present war broke out, Major Howie was appointed A.D.V.S.

Adrian Howie was a man of the highest character and integrity; genial, deeply sensitive of an injustice, and outspoken to the extent of even being at fault if he considered the cause he was espousing warranted such an attitude of mind.

His benefactions were many, but known to few; the writer is aware of many distressed families he has assisted; and the endowment of a cot at the Children's Hospital, Durban, was sponsored by his good lady and himself.

We extend our heartfelt sympathy to his devoted wife and three daughters, who mourn his loss. *Ave Atque Vale!*

W.A.D.

OBITUARY.

DAVID THOMAS MITCHELL.

It is with deep regret that we have to record the death of David Thomas Mitchell from heart failure at the age of 58. He was born in Northern Ireland on 16th March, 1885 and came to South Africa in 1909 as a Government veterinary officer under the Transvaal Government. A year later he was appointed as a bacteriologist under Dr. Theiler, as he then was, at Onderstepoort. In 1914 he was appointed officer-in-charge at Allerton laboratory, Pietermaritzburg, where he remained until 1918, when he returned to Onderstepoort as Assistant Director under Mr. R. E. Montgomery. In 1920 he was sent to Vryburg as officer-in-charge of the

lamsiekte investigation station at Armoedsvlakte. In 1921 - 1922 he spent a year overseas in post graduate study and on his return was in charge of the laboratory at Allerton until 1928, when he was appointed under the Burmese Government to study anthrax in elephants. He was very successful in producing a vaccine which reduced the mortality from the disease very greatly and earned him the gratitude of the teak companies who presented him with a magnificent piece of silver plate in recognition of his work in Burma, on his retirement in 1939.

After his return to South Africa his services were made use of by the Union Government to superintend the production of rinderpest vaccine for the campaign against the disease in Tanganyika in 1940. His experience of the disease in Burma and his great organizing ability enabled him to make an unqualified success of his side of the campaign. On his return his services were retained to assist in the department for the study of virus diseases at Onderstepoort. He gave up his appointment in February of this year and took up one at the South African Institute for Medical Research at Rietfontein, Johannesburg where he had just commenced to work on typhus vaccine production when he passed away.

To his widow, his two daughters and to his only son, who was a prisoner-of-war in Italy and is being repatriated, we express our deepest sympathy in their sad loss.

E.M.R.

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A RICKETTSIA-LIKE ORGANISM FOUND IN THE BLOOD OF PIGEONS.

A. S. CANHAM,
Allerton Laboratory, Pietermaritzburg.

The intention of this note is to record the finding of bodies resembling rickettsia in smears made from the blood of pigeons.

The best known rickettsia-like organism of birds is Rickettsia psittaci (Lillie). This organism is the cause of the disease psittacosis that affects human beings, parrots, parakeets and a large variety of other birds. It was not widely recognised that birds outside the order Psittaciformes could be a menace in the spread of this disease until in 1933 Meyer and Eddie isolated a strain of the psittacosis virus from apparently healthy canaries (*Serinus canarius*) that were concerned in an outbreak of psittacosis in humans. The same workers identified the virus in a butterfly finch (*Cyanospiza ciris*). Meyer (1935) showed that the Java rice bird (*Padda oryzivora*) could become infected under natural conditions. He was also able to show that the Pekin robin (*Liothrix luteus*) and the bullfinch (*Pyorrhula vulgaris*) were susceptible to psittacosis. Carpano (1936) claimed to have found rickettsia-like bodies in the blood and organs of a bullfinch in Cairo.

According to Donatien and Lestoquard (1937) *R. avium,* the rickettsia of the bullfinch, and *R. psittaci* are found not only in the endothelial cells but also in the mononuclear leucocytes in which they take the form of clusters. They draw attention to the fact that *R. psittaci* is transmissible to the sparrow family, to which finches belong, and suggest that *R. avium* and *R. psittaci* may be one and the same rickettsia. This seems to be borne out by the previously mentioned findings of Meyer and Eddie (1933).

Haagen and Mauer (1938), in Germany, reported the finding of the virus of psittacosis in natural cases of this disease in imported finches and in indigenous siskins (*Spinus spinus*) and coal-tits.. The fulmar petrel (*Fulmarus glacialis*) was shown by these workers, also in 1938, to be responsible for the disease in man in the Faroe Islands.

Coles (1940) described a new rickettsia-like organism found in the epithelium of the conjunctiva of domestic fowls. These birds were the subject of ocular roup and the organism might be compared with *R. conjunctivae,* Coles (1931), and *R. conjunctivae bovis,* Coles (1936).

Pinkerton and Swank (1940) reported on the finding of a virus in pigeons closely related to if not identical with psittacosis virus in

pigeons. They suggest that a thiamin deficiency (*Vitamin B*) might be capable of changing a latent infection with psittacosis virus into an active stage with consequent danger of spread to other birds and man. These investigations are of interest inasmuch as a serially transmissible virus was obtained as a result of inoculating mice intracranially with the exudate or ground-up organs of pigeons showing lesions in the chest or abdominal cavity or in both.

Coles (1940) described psittacosis of the domestic pigeon (*Columba livia* var. *domestica*) from cases investigated by him during 1939 in Johannesburg.

History.

In July, 1939, a pigeon, suspected of having died of paratyphoid, was received from Durban. During the course of the examination of smears made from the blood and various organs, certain bodies were found in a number of monocytes. These smears were sent to Onderstepoort for further examination. A few days later a pigeon just dead from acute pigeon-pox was brought in by the owner from a Pietermaritzburg loft. Smears from this bird also revealed the presence of these monocytic inclusions. Shortly after this, news was received from Onderstepoort that these bodies resembled Rickettsia. In view of the fact that Coles had just confirmed the presence of psittacosis in pigeons in a Johannesburg loft, concern was expressed as to whether these bodies might be connected with psittacosis. It was then decided to examine a large number of smears from pigeons at this Institution and again these bodies were seen fairly frequently.

Occurrence.

The organisms have been found in smears made from the blood, lungs, liver, kidney and spleen of pigeons. They appear to be most common in lung smears and then, in order in diminishing number, in liver, kidney, spleen and blood smears. These findings are in accord with the observations made on the predilection sites of *R. bovis, R. canis* and *R. ovina* by Donatien and Lestoquard (1937). They have described the technique of lung and liver puncture which facilitates the finding of the rickettsia in the live subject.

These organisms are, as a rule, found in the cytoplasm of the monocytes but may occasionally be seen in the cytoplasm of lymphocytes.

Morphology.

The individual organisms are very small bodies varying in size from 0.2μ to 0.8μ and for the most part take the form of coccoid granules. In the majority of cases they are found in circular masses varying in size from 4μ to about 12μ. In the circular clumps individual organisms are packed very closely and are usually surrounded by a

clear halo. This halo appearance was noted by Cowdry (1925), who does not consider it a pathological change. He again remarked on its presence in his work on the etiology of heartwater (1926). Steck (1928) considers that it is probably a staining artefact due to the deprivation of precipitable basic dye by the strongly basophile granules. He compares it with the perinuclear halo in lymphocytes and plasma cells. This does not, however, appear to apply to the organism under description as it is for the most part not markedly basophilic but stains with a light lilac colour. Coles (1931) remarked on the presence of halos in extra and intra-cellular forms of *R. conjuntivae*. Donatien and Lestoquard (1937), in describing the morphology of the rickettsias of the monocytes, make mention of halos. In some of the monocytes the individual organisms are not grouped in circular or oval masses but appear somewhat scattered in the cytoplasm. They vary from 0.8μ to 1μ in size and stain a deep blue colour as compared with the organisms of the compact colonies which take a lilac colour. These large, deep-blue-staining bodies are rather similar to the initial bodies of psittacosis, trachoma and *R. canis* infections. Thus, this rickettsia-like organism of pigeon blood reveals itself in colonies, some simulating initial bodies, and others simulating elementary bodies. As in human trachoma, a few colonies of the pigeon organism appear to contain both elementary and initial bodies.

The number of individual organisms in the compact groups varies from about twenty up to a few hundred, whereas the larger bluish staining organisms are very much fewer. One agranulocyte may harbour up to six colonies. They are always found in the cytoplasm and never invade the nucleus. When the compact circular groups are large they appear to impinge on the nucleus causing a concave depression into which the inclusions fit. May Grünwald-Giemsa stain has been used for identifying these bodies and excellent results have been obtained. The circular compact groups stain a pinkish lilac colour while the isolated larger ungrouped elements take on a bluish tinge. These organisms are Gram negative and are not acid fast. The white cells containing these bodies are not distorted.

Transmission.

Up to the present all attempts to transmit these bodies have been unsuccessful. Thinking that splenectomy might cause the organisms to appear, this operation was performed on pigeons with negative results. It was thought that the common pigeon fly, *Pseudolynchia maura*, might be the transmitting agent but dissection of these flies, followed by the making of smears from various portions of the digestive tract, failed to reveal the presence of any bodies resembling the organisms in question.

Symptoms and Lesions caused by these Cell Inclusions :

No symptoms accompanied the presence of these cell inclusions, and no lesions were discernible on careful post-mortem examination of pigeons in which the bodies were found.

Discussion:

It may now be asked what these bodies represent, and if they are parasites, what their nature may be.

Bedson (1940), who examined the slides showing these bodies, had no hesitation in saying that they were unlike the psittacosis virus. Their staining properties differed from psittacosis particles, while the variations in size, that are a feature of psittacosis virus, are not present in the cell inclusions of the pigeon.

It was suggested that these inclusions resembled the Kurloff bodies in the mononuclear cells of guinea-pig blood. The small Kurloff bodies are compact and homogeneous in appearance, whereas pigeon cell inclusions are made up of a grouping of small separate particles. The large Kurloff bodies have no resemblance at all to the bodies in pigeon blood. Ledingham (1940), has published good plates of these bodies.

Another disease known as "Cell inclusion" or Macfie Disease was first described by Macfie (1914) as having been observed by him in fowls in Nigeria. The following year he identified the same disease in a turkey. Adler (1925) investigated a similar disease in Palestine. Smears made from these cases were sent to Macfie, who expressed the view that the inclusions in the white cells were similar to those seen by himself in Nigeria. Gilbert and Simmins (1931) investigated further cases of cell-inclusion disease in Palestine and came to the conclusion that it was caused by a filterable virus associated with leucocytic inclusions. The same authors (1934) did further work on this disease and from the article one obtains the impression that they suspected some similarity between this disease and fowl plague. Komorov (1934) proved that the virus isolated from cases of "Acute cell inclusion disease" was identical with the virus of fowl plague. Moreover, he showed that cell inclusions identical with those described by Adler (1925) are found in a variety of infections, especially in birds shown to be infected with spirochaetosis. The cell "inclusions" found in cases of spirochaetosis, are, of course, not parasites, but normal specific cell granules of the myelocytes. Finally, he stated that his results did not, however, exclude the possibility of the existence among poultry in Palestine, of a specific virus disease known as "cell inclusion disease," but it seemed clear that no pathognomonic value can be attached to the presence of the "leucocytic inclusions." Coloured plates showing the "cell inclusions" in the Ann. Trop. Med. and Parasit. Vol. VIII, 1914 - 1915, have not the slightest resemblance to the cell inclusions in pigeons' blood.

Donatien and Lestoquard (1937) suggests that Rickettsiosis should

be classified as (a) Local Rickettsioses and (b) General Rickettsioses; with a further sub-division of this second group into (1) Rickettsioses of the endothelial cells and (2) Rickettsioses of the monocytes. This seems a reasonable classification, as *R. ovina* and *R. bovis*, rickettsioses of the monocytes, have been found only in monocytes or free in the blood. *R. canis* has been found in monocytes and free in the blood, but also in neutrophiles. The pigeon organism has been found mainly in the cytoplasm of the monocytes, never free, but occasionally in the cytoplasm of lymphocytes. There appear to be many points of similarity when comparisons are made between *R. canis, ovina, bovis* and the pigeon cell inclusions.

In the case of the "rickettsiosis of the monocytes" the organisms are usually found closely grouped in the form of clusters and they measure from 0.2μ to 0.3μ. Exceptionally these organisms are scattered through the cellular cytoplasm and in these cases are somewhat larger, being from 0.5μ to 0.6μ. The organisms of the pigeon cell inclusions in similar circumstances may be about 0.8μ in size.

In the three "rickettsioses of the monocytes" the number of clusters of organisms vary from a few to a large number and in some cases the whole of the cytoplasm of invaded cells is one mass of organisms. In the case of the pigeon cell inclusions the largest number of clusters that have been seen in the cytoplasm is six, but in no case has the whole cell cytoplasm been occupied by organisms. The predilection sites of *R. canis, bovis, ovina* and the pigeon cell inclusions are similar, as mentioned previously under the occurrence of the organisms. Their staining affinities are similar and none of these organisms invades the nuclei of cells, although they appear to impinge on the nuclei forming smaller or larger concavities in it.

Finally there is an organism described by Tyzzer (1938) *Cytoecetes microti* which is a parasite developing in granulocytes and infective for small rodents. This organism was found in the blood of field voles (*Microtus pennsylvanicus*), and occurs in the form of minute cytoplasmic inclusions, delicate rings and multiplication masses composed of small, more or less rounded elements. It is chiefly confined to polymorphonuclear leucocytes and eosinophiles, but has been seen in basophile leucocytes. Judging from the very good coloured plates accompanying the article on this parasite, there is nothing comparable with the cell inclusions of the pigeon blood cells, quite apart from the fact that *Cytoecetes microti* is found only in granulocytes while the pigeon cell inclusions are found solely in the agranulocytes.

Summary.

An organism which is apparently a new Rickettsia has been described in the blood of pigeons. This has been compared with various other organisms and has most resemblance to those Rickettsiae which

parasitize monocytes. The organism is apparently harmless.

So far all attempts to transmit this organism have failed and the vector has not been discovered.

FIG. I.
White cell showing Rickettsia-like inclusions in cytoplasm.

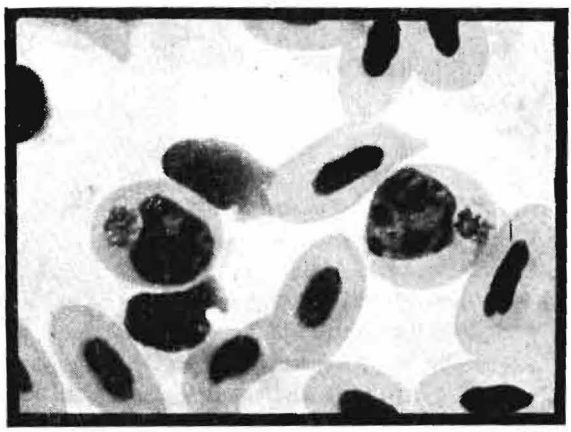

FIG. II.
Two white cells showing Rickettsia-like inclusions in cytoplasm.

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THE ERADICATION OF THE BROWN DOG TICK (*RHIPICEPHALUS SANGUINEUS Ltr.*) FROM A DOG KENNEL.

W. O. NEITZ,
Onderstepoort.

INTRODUCTION.

The common dog tick is found practically all over the world, even in the hottest and driest areas. Under favourable conditions this tick is capable of breeding very rapidly, and the life cycle can be completed within a period of three months. It is a domestic pest usually found in association with dogs. Occasionally the tick may be carried into dwelling houses, where it may become a very serious household pest. Heavy infestation of dogs is common, and human beings may also be attacked. Besides causing loss of blood and severe irritation of the skin, this tick is capable of transmitting a large number of diseases, mentioned in the appended table, to man and animals. From this it is evident that every attempt should be made to eradicate it.

CONTROL.

The control of this tick is by no means simple and in order to carry out a successful eradication, the infested dogs as well as their sleeping places, have to be treated. The various stages of the tick are able to survive for very long periods without feeding, and this naturally adds further to the difficulties of control.

Whilst it is possible to destroy ticks on dogs with arsenical, carbolic and derris dips, these do not prevent the larvae, nymphae and adults from attaching themselves shortly after treatment. This difficulty was experienced in one of the kennels at Onderstepoort. Numerous ticks were hiding in the crevices and the dogs were heavily infested with *Rh. sanguineus,* in spite of the fact that they were being dipped regularly once a week in arsenical, carbolic or derris solutions.

The unsatisfactory condition in the kennels was further aggravated when two dogs which had been infected with *Rickettsia canis* were introduced. In spite of the regular weekly dipping, this disease spread from one kennel to the next, and the danger existed that it would spread to other parts of the station. For this reason serious attempts were made to eradicate the ticks by dipping the dogs regularly and by spraying the kennels. The first attempt was made with the paraffin soap emulsion described by Mönnig (1934). This spray was forced

into the crevices with a hand pump. The results were very disappointing, as only a comparatively small number of the ticks were destroyed. Since very little improvement was noticed after the kennels had been sprayed on three occasions at fortnightly intervals, an attempt was made to destroy the ticks with a 6.6 per cent pyagra solution in paraffin. In order to force this solution into the numerous crevices in the walls and in the woodwork an electric duco spray-pump, as illustrated in the photograph below, was used. (Fig. 1).

After having sprayed two kennels with a surface area of approximately 450 sq. ft., it was found that 2 gallons of the paraffin pyagra

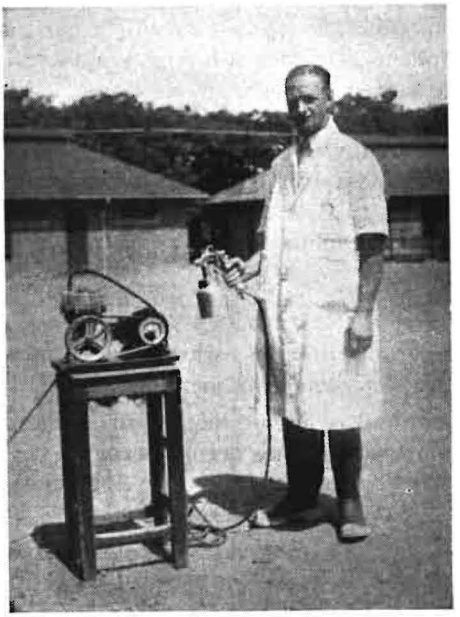

FIG. I.
The electrically operated air compressor and type of spray gun used.

solution were required and that approximately 30 gallons would be necessary to spray all the kennels, which had a total surface area of approximately 6,500 sq. ft. The continuous inspiration of the paraffin fumes by the operator could not be tolerated for any length of time and for this reason it was necessary to find a more suitable vehicle of higher viscosity. For this purpose a mixture of one gallon of paraffin and 4 gallons of old motor oil was used as a solvent for the pyagra. On applying this it was found that far less fumes were created, and that a greater area could be sprayed with a gallon of this pyagra-

oil-paraffin solution. For all the kennels 8 gallons of this solution were necessary.

The kennels were left untreated for a period of three months, when a small number of ticks were again detected on the dogs, which had been dipped regularly at weekly intervals during this period. It must be assumed that they were the progeny of ticks which had escaped destruction and it was realized at this stage that it would be essential to repeat the operation immediately. This was done and since then no further cases of *R. canis* infection in any of the dogs have been observed, in spite of the fact that a dog which harboured a latent infection of this disease was present in one kennel.

Since the second application of this method it has been found that it was sufficient to spray the kennels every six months in order to keep the tick population at a very low level. Complete eradication of the ticks would have been possible if it were not necessary to introduce new dogs from time to time. Their regular re-appearance is a good indication of how rapidly these ticks can re-establish themselves if no counter measures are adopted. The remarkable results which have been obtained by applying this method for a period of two years clearly indicate the value of the method.

Summary.

1. The numerous diseases which the brown dog tick (*Rh. sanguineus*) is able to transmit are mentioned.

2. A practical and economic method of eradicating this tick with a pyagra-oil-paraffin solution has been described.

Acknowledgments.

The author wishes to express his appreciation to Mr. F. B. Boughton for the painstaking way in which he conducted the spraying operation over the entire period of the experiment.

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Parasite.	Disease.	Stage of Tick Capable of Transmitting the Disease.			Occurrence.	Host.
		Larva	*Nympha*	*Imago*		
Anaplasma marginale.	Anaplasmosis Gallsickness.	—	+	—	Many Countries.	Cattle.
Babesia gibsoni	Babesiosis Bilary Fever.	+	+	+	Asia.	Dog and jackal.
Bacterium tularense	Tularaemia.	—	+	+	America, Europe.	Man, rodents, etc.
Dermacentroxenus rickettsi	Rocky Mountain Spotted Fever.	+	+	+	North America.	Man, dog, rodents, etc.
Dermacentroxenus rickettsi var. *brasiliensis.*	São Paulo Typhus.	—	+	+	South America.	Man, dog and rodents.
Dermacentroxenus rickettsi var. *conori*	Fièvre Boutonneuse.	+	+	+	Mediterranean Countries.	Man, dog and rodents
Dermacentroxenus rickettsi var. *pyperi*	Tick-bite Fever.	+	+	+	South and East Africa.	Man, dog and rodents
Hepatozoon canis		+	+	+	Many Countries.	Dog and jackal.
Piroplasma canis	Piroplasmosis. Biliary Fever.	+	+	+	Many Countries.	Dog and fox.
Rickettsia canis	Rickettsiosis.	+ ?	+ ?	+ ?	Africa.	Dog and jackal.
Spirochaeta hispanica	Spirochaetosis. Relapsing Fever.	—	+	+	North Africa.	Man, jackal, fox & moroccan hedgehog

DILATATION OF THE SMALL INTESTINE OF THE FOWL.

J. D. W. A. COLES.
Onderstepoort.

Dilatation of the small intestine of the fowl is a complaint that is not uncommonly seen. The ballooning of the gut, due to *Eimeria necatrix* and *E. maxima* infections, is well known, particularly in the case of young pullets. The ballooning is usually accompanied by haemorrhagic enteritis when *E. necatrix* is responsible, whereas *E. maxima* is productive of only a reddish-tingd inflammatory exudate.

Few are acquainted with the moderate degree of ballooning often associated with a chronic *Eimeria acervulina* infection, which is characterised by the production of an unlimited number of asexual generations of the parasite. When the small intestine of such a fowl is opened the mucosa is seen to be thickened, and it may show small greyish specks, about 1 mm. in diameter, which tend to become elongated in a transverse direction. An intestinal scraping will reveal very large numbers of *E. acervulina* oocysts.

Dilatation of the gut may be the result of a damming-up of ingesta following stenosis. Most of these cases are due to solitary lymphocytomata involving the intestinal wall. Intestinal intussusception is a rare complaint but it, too, may lead to acute dilatation of the bowel anterior to the lesion. Mesenteric hernia and intestinal volvulus are also very unusual causes of dilatation.

A hitherto undescribed form of intestinal dilatation.

The purpose of this paper is to describe yet another, and very characteristic, form of intestinal dilatation, which so far seems to have escaped attention. Thirty-nine cases from the Onderstepoort pedigreed flock have been collected over a period of four years as a result of examining just over 300 adult fowls that have died, or been culled and killed to make room for younger stock. The autopsy records thus reveal an incidence of about 1.3%, a figure that is not altogether negligible.

Among 620 adult hens hatched in 1938 there were 8 cases;
„ 800 „ „ „ „ 1939 „ „ 9 „ ;
„ 950 „ „ „ „ 1940 „ „ 14 „ ;
„ 700 „ „ „ „ 1941 „ „ 8 „ ;

The figures for the various years show no significant differences.

The condition has so far been encountered only in females, but it is most doubtful if this means anything, as comparatively very few males are retained beyond the age of 220 days (the shortest period observed between hatching and the detection of the dilatation).

Considering that few fowls survive or are retained after the age of 900 days, it is obvious from the accompanying table that the disease is essentially one of middle life. Another striking fact is that egg production by the affected birds was exceedingly poor, except in the case of hens E. 36 and E. 951 which were autopsied only after 904 and 1,065 days respectively.

Diagnosis.

The symptomatology is indefinite. Apart from poor egg production, the bird will show nothing more than marked emaciation. It seems certain that the condition is often fatal. Many affected fowls were in reasonably good flesh at the time they were killed.

The diagnosis is easily confirmed after death, when the central portion of the small intestine reveals a sausage-shaped dilatation that is usually 2 to 2.5 cms. in diameter and anything up to 25 cms. in length. The dilatation begins abruptly at its anterior end and ceases just as abruptly at its posterior end. There is no indication of any stenosis being responsible for the complaint. The swelling is generally fluctuating and this, when the gut is opened, is seen to be due to the presence of much fluid and clots of ingesta mixed with catarrhal exudate. The bowel mucosa is thickened and shows a varying degree of inflammation which can be classified as a diphtheritic inflammation in the worst cases.

When the disease terminates fatally the cadaver is grossly emaciated, there is a yellow gelatinous infiltration of the epicardium and sometimes hydropericardium, the heart is about two-thirds its normal size, the liver, spleen and gizzard are atrophied, and the unaffected portions of the small intestine are thin-walled and shrunken and usually empty. In fact it can be said that the fowl starves to death. The disease seems to run a very chronic course, the length of which can only be guessed at from the disappointing failure to lay well.

In one hen there was an accompanying nephrosis. Another had a multiple disseminated necrobiotic hepatitis of bacterial origin, and possibly the infection originated in the gut. Four others suffered from lymphocytomatosis, a proportion no greater than that in the flock as a whole.

The Etiology of the Condition.

The cause of the dilatation remains obscure. Fruitless attempts have been made to correlate the disease with the ordinary common asthenia of unknown etiology, with neurolyphomotosis gallinarum and

with neoplastic conditions. However, there are two facts which, taken together, suggest that inherited factors play a role and that the cock is of greater importance than the hen in transmitting the influence.

Forty-nine cocks were used in the breeding pens during the years under review. Cases of intestinal dilatation appeared amongst the progency of twenty-three and all these males were descended in some way or another from a certain cock 955. There were twenty-six sires that produced unaffected progeny and cock 955 appeared in the pedigrees of twenty, but not of the other six. It is perhaps significant that all six males without cock 955 in their ancestries, should have given rise to apparently resistant birds.

The second interesting point is that two of the twenty-three suspected males produced ten of the thirty-nine cases among 130 of the 1535 pullets concerned. No attempt was made to segregate the progeny of different matings.

The possible importance of the cock also seems to be emphasized by the fact that thirty-seven hens were the dams of the thirty-nine affected birds. In only two instances did two full sisters develop intestinal dilatation. If inherited factors are of importance, the pedigrees reveal that they can be passed from a cock to his daughter and then to her son.

It should be noted that, because of poor egg-production, no hen with a dilated gut ever won a place in a breeding pen, and so all died without issue.

Summary.

The known causes of dilatation of the small intestine of the fowl have been reviewed. Another, and very characteristic form of dilatation has been described. The etiology of this newly-recognised condition has not been elucidated, but there is evidence to suggest that the cock may play an important role in the transmission of a predisposition to the complaint.

TABLE OF CASES 1.

Hen.	Age in days at death.	Eggs.	Remarks.
E. 36	904	211	Emaciated : a 15 cms. dilatation of apparently long standing at centre of small intestine.
E. 219	353	106	Sausage-shaped dilatation at centre of small intestine ; necrosis of affected mucosa.
E. 419	483	124	A 5 cm. dilatation at centre of small intestine ; also lymphoid leucosis of liver.
E. 463	498	71	5 cm. dilatation of gut ; affected intestinal wall thickened, particularly due to complicating chronic localised productive perienteritis.
E. 517	259	11	Emaciated ; ballooning of portion of small intestine ; lymphocytomatosis of myocardium.

TABLE OF CASES I. (Continued).

Hen.	Age in days at death.	Eggs.	Remarks.
E. 951	1065	228	Culled ; killed ; moderate dilatation of middle portion of small intestine.
E. 972	532	113	Dilatation of central part of small intestine.
E. 994	509	92	Middle portion of small intestine dilated.
G. 30	595	132	Emaciation ; hydropericardium ; gelatinous infiltration of myocardium ; 12.5 cm. dilation involving central portion of small intestine.
G. 87	535	118	Culled ; killed ; 15 cm. dilatation at centre of small intestine.
G. 95	514	70	Culled ; killed ; 15 cm. dilatation at centre of small intestine.
G. 136	409	39	Emaciation ; heart, liver and spleen shrunken ; dilatation at centre of small intestine, with necrosis of the bowel wall.
G. 157	371	49	Localised dilatation of small intestine and thickening of the wall due to necrosis of the mucosa of bacterial origin ; also multiple disseminated necrobiotic hepatitis.
G. 162	435	88	Culled ; killed ; 10 cm. dilatation at middle of small intestine.
G. 594	417	41	Dilatation at centre of small intestine ; spleen, liver and gizzard shrunken ; lymphocytomatosis of lungs.
G. 699	420	89	20 cm. Dilatation at middle of small intestine, with a diphtheritic enteritis involving the affected mucosa ; also nephrosis.
G. 909	376	2	Extreme emaciation ; heart, spleen, gizzard and intestines shrunken ; localised dilatation of small intestine ; lymphoid leucosis of liver.
H. 685	386	65	20 cm. Dilatation at middle of small intestine.
H. 740	468	79	Emaciation ; 17 cm. dilatation at centre of small intestine ; atrophy of liver, spleen, heart and gizzard.
H. 742	533	91	Culled ; killed ; 7.5 cms. dilatation at centre of small intestine.
H. 814	436	73	Emaciation ; heart, liver, spleen, gizzard and intestines shrunken ; 7.5 cm. dilatation at centre of small intestine.
H. 886	495	129	Culled ; killed ; 15 cm. dilatation at middle of small intestine.
H. 946	417	55	Emaciation : atrophy of gizzard, spleen, liver and heart ; 13 cm. dilatation in the middle of small intestine.
H. 961	494	84	Culled ; killed ; 12 cm. dilatation in usual place ; full sister of H. 8282.
H. 963	494	45	Culled ; killed ; 22 cm. dilatation of small intestine ; full sister of H. 965.
H. 965	340	7	Emaciation ; shrinkage in size of heart, spleen and liver ; 12 cm. dilatation of small intestine ; full sister of H. 963.
H. 4607	220	0	A 15 cm. segment at centre of small intestine is ballooned and the affected mucosa is necrotic.
H. 8240	430	68	A 15 cm. dilatation in the usual locality; lymphoid leucosis of liver.
H. 8251	494	95	Culled ; killed ; 10 cm. dilatation at centre of small intestine.
H. 8267	487	117	Culled ; killed ; dilatation 15 cm. long in the middle of small intestine.

TABLE OF CASES I. (Continued).

Hen.	Age in days at death.	Eggs.	Remarks.
H. 8282	501	0	Culled ; killed ; dilatation at the usual site was 10 cm. long ; full sister of H. 961.
J. 8429	478	161	15 cm. Segment at centre of the small intestine only moderately distended ; culled and killed.
J. 8520	478	67	12 cm. Dilatation in the usual locality; culled and killed.
J. 8696	464	119	13 cm. Dilatation at the usual site ; culled and killed.
J. 8726	383	105	Ballooning of central portion of small intestine ; culled and killed.
J. 8871	449	92	Culled ; killed ; 25 cm. of small intestine dilated.
J. 8968	441	52	Emaciated ; atrophy of heart, liver, spleen gizzard and intestines ; 3 cm. dilation at centre of small intestine.
J. 9018	463	67	Culled ; killed ; centre of small intestine moderately dilated over a distance of 15 cm.
J. 9026	452	27	Emaciated ; 15 cm. dilatation involving middle of small intestine ; culled and killed.

ERADICATING MEERCATS.

A. D. THOMAS and P. S. SNYMAN.
Onderstepoort. — Bloemfontein.

Past Experience.

Some four years ago, after preliminary research into the life habits of meercats (*Cynictis, Suricate* and *Geosciurus* spp.) we drew up a tentative scheme for the partial eradication of these animals as a means of controlling rabies. It was realised then that total eradication, if not entirely impossible, would cost an amount quite out of proportion to the economic importance of the disease itself. [Snyman and Thomas (1939).]. In a subsequent publication, Snyman (1940) gave details of the work on which these recommendations were based.

In the meantime a gang of eight to twelve natives under the supervision of a European, has been actively engaged in probing and proving the feasibility, or otherwise, of the proposal under conditions actually prevailing in the infected areas. This gang, which may be regarded as the pilot unit in the larger scheme, has not only cleaned up a considerable area of country, but has obtained very useful data and experience which should prove invaluable if eradication measures on a large scale should have to be put into effect in the future.

That rabies is firmly established in the viverrid population of the country can no longer be questioned, and our belief expressed in 1939 that the incidence of the disease seemed proportionate to the density of the carrier population seems well founded — (see maps in articles quoted). Our aim so far has been to utilise this gang for eradicating these carriers in sufficiently large areas around known outbreaks of rabies, in the hope of stamping out the disease at those points. There appears to be a reasonable possibility of achieving this in the Orange Free State, East of the Main North-South railway line, where the meercat population is low and the outbreaks are isolated and scattered. Operations in the more densely populated parts, e.g. at Leeudoornstad, S. Transvaal, show that the infection is practically continuous and that little short of complete and simultaneous eradication in whole districts will be of any avail. In the semi-arid country towards the west, e.g. Hay District, C.P., *Cynictis* becomes very scarce and probably plays a minor role as a carrier. Its eradication under such conditions ceases to be worth while inasmuch as the spotted genet — a non burrowing carnivore — becomes the major carrier in this bush-clad country.

Destruction of Meercats.

The methods employed for the destruction of meercats remain essentially the same as described by Snyman (1940).

The veld is combed systematically in successive trips, by the gassing squad walking in extended formation 20 - 50 yards apart, depending on height of vegetation. In this way the meercats flee to their burrows, which are gassed immediately and closed, and the colony is marked with a stake and number for easy inspection later. Any meercats escaping the effects of the gas are soon located by the fact that they reopen their burrows. Such meercats are mopped up by trapping, which is continued until all holes remain closed, a sure indication that all inhabitants have been accounted for.

To date some 32 separate infected areas involving about 289,000 morgen of ground have been cleaned up in this way. The cost has been worked out, on an average, at about £10 per 1,000 morgen, but this item naturally depends a lot on the nature of the ground covered.

So far, two recurrences of the disease have been detected in the previously treated areas. On seeking the reasons for this failure we found (a) that the trapping had not been thorough enough, especially along the edges, and that re-infiltration of meercats from surrounding untreated veld was rapid ; (b) that treating an area bounded by a line drawn arbitrarily around the original site of the outbreak did not mean that all the infected ground was included unless the circle was made unnecessarily large. Re-infiltration as in (a) above, could therefore take place from an adjoining area still infected.

Re-infiltration of Meercats from the Sides.

For every area treated a sketch map on a fairly large scale is prepared. It shows the farm boundaries, the main topographical features — hills, water-courses, etc., and on it the position of all colonies is marked.

It was discovered in this way that the colonies were distributed mostly along the edges of gullies and water-courses and to a lesser extent in other well-drained localities on high ground. This means that the watershed separating adjacent catchments is more or less a no-man's-land as far as meercats are concerned. Of course the confluence of water-courses forms many open points of contact between the various catchments and there may be other gaps too. However, experience has shown that if such a catchment basin is cleaned thoroughly, re-infiltration over the watershed barrier is limited and slow. Cleaning up at points of confluence and through gaps is carried on for a mile or so beyond, in order to retard re-infiltration somewhat. The value of these simple considerations is well illustrated in some recently cleaned areas in which periodic inspection of the veld reveals that even

12 months after treatment the burrows are still closed and even overgrown with kweek (couch grass). In places done two years ago a few meercats have re-appeared, and after three years most colonies are re-inhabited.

Demarcating the Infected Area.

Early in our work the finding of meercat carcasses and skeletons in or near their holes had evoked the suspicion that the animals might have died of rabies. To-day such carcasses are carefully looked for while combing the veld and gassing colonies, and their position recorded on the map. When fresh enough, brain material from such dead animals is sent up for laboratory examination and several positive cases of rabies have been confirmed in this way.

Plotting the position of these carcasses now serves as a useful guide in determining in which direction and to what extent the cleaning up should be carried out. The idea of course is to continue cleaning beyond the last carcase to the next available topographic barrier.

The rabies mortality in meercats apparently varies within a wide range, but at least two interesting instances are on record in which it must have been high. At Dealesville in 1937 (A.D.T.) and at Ventersvlei in 1940 (P.S.S.) it was noted as very striking that a large proportion of the colonies were uninhabited or abandoned. At the latter place a number of burrows were actually excavated and numerous skeletons of all three species were found. At both these places rabies was diagnosed, but it is merely assumed that this disease was in fact the epizootic responsible for the decimation of the meercats.

Summary.

A short review is given of the work being done and experience gained in eradicating meercats in rabies-infected areas. Of special interest are two observations recently made concerning the advantages of (a) clearing out meercats from topographically enclosed basins and (b) utilising the presence of carcases to indicate the extent of the infected area to be treated.

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HISTORY OF THE S.A.V.C., 1910 - 1939.

(Continued from March, 1943, issue.)

III. Veterinary Considerations.

(i) Preliminary Operations.

According to Smith (1916), at the commencement of hostilities the V.O. (P.F.) was on the sick list and the first step taken by the Defence Force authorities was to instruct the O.C. of the former Transvaal Veterinary Corps (Col. J. Irvine-Smith), then on the Reserve of Officers, to be available to give advice regarding the national emergency. At the outbreak of war the veterinary personnel of the U.D.F. (P.F.) consisted of a V.O., 1 corporal and 1 civilian storeman.

The first step taken by Col. Irvine-Smith (who was appointed D.V.S. as from 1.9.1914) was to engage 7 veterinary practitioners. One (Capt. Bush) was sent to Tempe to relieve the V.O. (P.F.) and the remainder were despatched to Cape Town to join the forces destined for Port Nolloth ("A" force) and Luderitzbucht ("C" force).

Faced with a shortage of horses and mules, difficulty in procuring trained personnel, and lack of mobilisation stores, and of veterinary instruments and drugs, the D.V.S. performed a herculean task in meeting the demands of the Defence authorities. Among the first steps taken was to obtain approval for the gazetting of the veterinary regulations, which were duly published under Govt. Notice No. 1714 of 1914. They were subsequently revised and published under Govt. Notice No. 776 of 1915.

Definite instructions were then received "to form and organise a veterinary service." The base veterinary depot was established at Booysens (Johannesburg) for training purposes, the base veterinary store was set up at Maitland (Cape Town) and altogether 47 veterinary officers and 450 N.C.O.'s and men were recruited. Of the officers, 3 were lieutenant-colonels, 6 were majors and the rest captains.

In seven weeks' time provision from Booysens was made not only for 8 base veterinary hospitals and 15 mobile veterinary sections, but also for "veterinary details for all mounted units, remount and transport depots, shipping details and buying boards.

All appointments, enlistments, records etc., were made and kept at the S.A.V.C. headquarters, Johannesburg, and the demobilisation at the end of the campaign was also handled by this office without a hitch" (*Blenkinsop and Rainey*, p. 386([25]).

([25]) It may be mentioned here that the military authorities received professional assistance from the C.V.D. under Lt.-Col. C. E. Gray. It should be added that V.O.'s. supervised the canning of meat for U.D.F. contracts.

The arrangements made for veterinary hospitals were :— No. 1 Veterinary Hospital was situated at Prieska, No. 3 at De Aar, No. 4 at Luderitzbucht, No. 5 at Parow, near Cape Town, and No. 7 at Luderitzbucht. ([26])

In regard to "B" force, Major J. G. Bush was appointed A.D.V.S. with headquarters at Upington. Bush arrived at his destination at the middle of September, 1914, and "reported that the health of the animals was fairly good" although there were cases of strangles, ophthalmia and sand colic which required "constant veterinary attention" (p. 389).

It had been obvious that there was a rebellious faction and "the situation became distinctly strained. The loyal section consisting of the V.O., transport officer and maxim gun section . . . decided, in order to protect themselves against sudden surprise seizure, that sentries should be posted nightly . . . . and that the maxim guns should be trained on the rebel camp" (p. 389).

When Maritz left for Van Roois Vlei on 2.10.1914, the A.D.V.S. was left in command of the Upington camp. Some days later, on receiving an order from Maritz to despatch to Van Roois Vlei "all horses which could possibly carry a saddle" Bush ignored the instruction and "later on the general officer commanding approved of his action" (p. 389). The rebellion was about to break out.

"C" force, which was based at Luderitzbucht, suffered severely from a water famine and what supply was available was transported either from Cape Town or condensed locally from sea-water. The veterinary hospitals at Luderitzbucht were kept busy with glanders which, however, was eradicated by the employment of the subcutaneous mallein test. "Whale Island was used as an isolation station while the 2nd I.L.H. underwent the test" (p. 389).

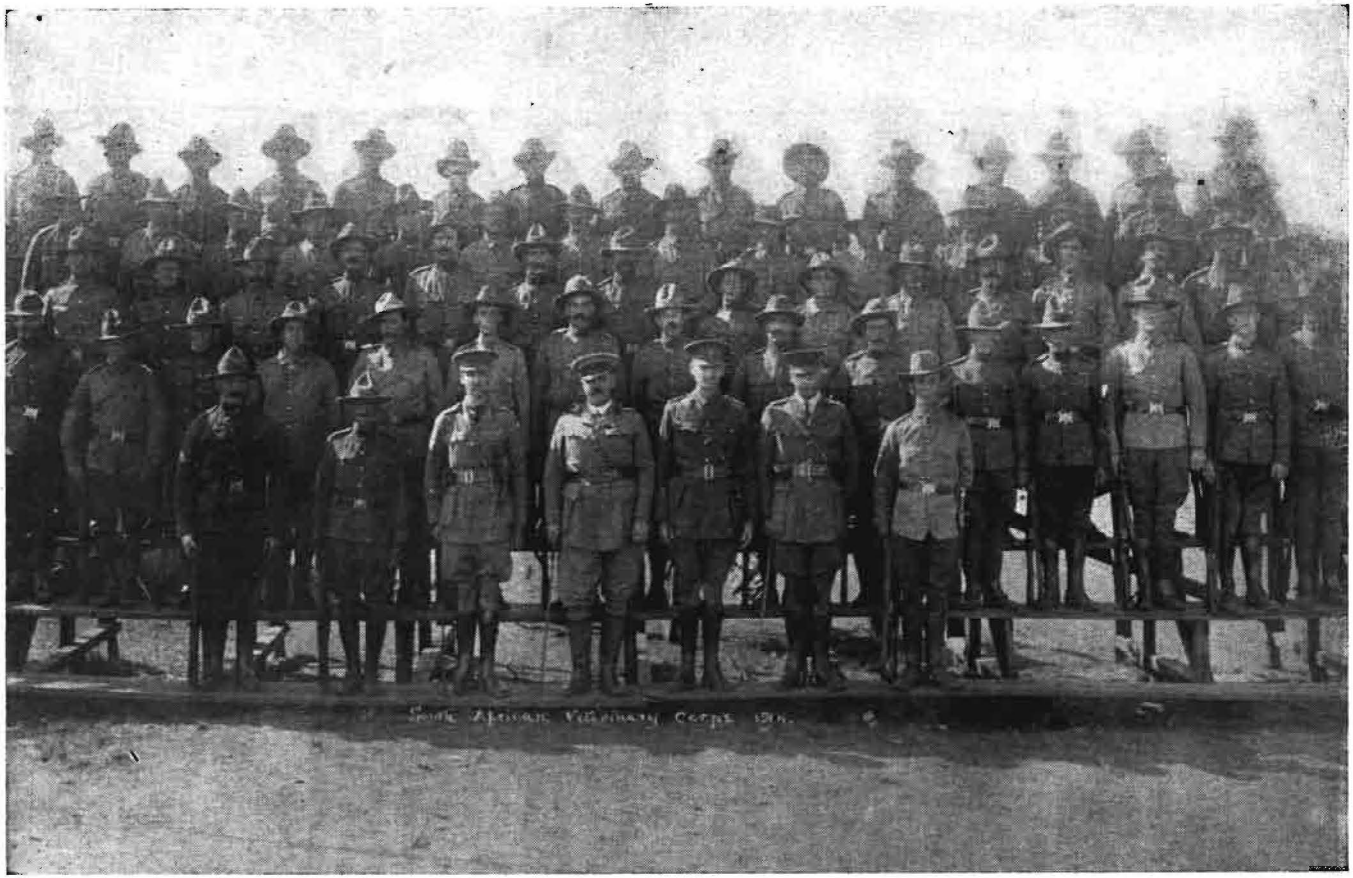
(ii) Rebellion.

With the outbreak of the Rebellion the demand for veterinary services was greater than before, since "numerous commandos and new mounted units were created" (p. 386). Further "a redistribution of mobile veterinary sections and the formation of new ones, as well as the establishment of No. 8 Veterinary Hospital at Pretoria" (p. 387) was arranged.

The position in regard to "B" force which, as shown above, had been precarious became worse. Major Bush, A.D.V.S., had to take command of one of the "defensive positions to the west of the town" (p. 389). Later, however, reinforcements arrived, these including 1st I.L.H., Enslin's Horse, and Natal L.H., as well as the 2nd Permanent battery of Artillery. The horses on arrival were in good condition, except those of the Natal L.H., which showed a high percentage of sore

([26]) As Blenkinsop and Rainey's publication is more accessible than Smith's (1916) roneoed report, subsequent page numbers refer to it.

(Fig. 18).



S.A.V.C., BOOYSENS, 1914.

Capt. W. H. ANDREWS; Lt.-Col. J. I. SMITH; Capt. J. B. COLLYER; Capt. P. R. VILJOEN. (in front).

backs. The donkey transport was in a "wretched condition" chiefly on account of the animals having been purchased too young.

On 25.10.1914 the A.D.V.S. proceeded to Kakamas, where, owing to the shortage of supplies, the horses were fed on wheat with the result that a large number of cases of laminitis and colic occurred.

On 17.11.1914 No. 1 Veterinary Hospital arrived at Upington. "Shortly afterwards a mobile veterinary section was despatched for duty with the brigade under Col. Bouwer, "who was endeavouring to intercept the rebel Kemp before he joined Maritz on the German border. This section did good work at Rooidam and was able to save a considerable number of brokendown and exhausted animals abandoned on the line of march" (p. 390).

On 23.11.1914 "A" Mobile Veterinary Section arrived at Upington. On the completion of the operations against Kemp, the troops returned to Upington. Owing to shortage of rations and overwork their horses were in poor condition. As rebel forces were constantly in the vicinity of the town, grazing was restricted and as the Orange River rose "to an unusual extent" and there was no bridge, the problem of feeding animals became serious. In fact many horses died of debility due to starvation.

On 24.1.1915 Upington was attacked by the enemy, but without success ; and until the rebel forces surrendered on 2.2.1915 the position regarding animal rations remained serious.

(iii) Resumption of Hostilities.

(1) *Southern Force.*

At the time of the general advance in March, 1915 the veterinary arrangements were :—

Headquarters : A.D.V.S. at Upington,

No. 1 Veterinary Hospital, 1 V.O.,

Transport, 1 V.O. and veterinary details, and 4th, 10th and 11th Mounted Brigades, 1 Mobile Veterinary Section each.

The A.D.V.S. left Upington for S.A.V.C. headquarters, on 10.3.1915 and after completing certain routine matters at Kimberley and Standerton returned to Upington "with instructions to join General Smuts' staff as A.D.V.S." with the Southern Army. It will be remembered that this army was formed as a result of the amalgamation of the Southern, Eastern and Central forces at the beginning of April and was disbanded on 5.5.1915.

The A.D.V.S. on arriving at Ukamas in ex-German territory found that the original Southern force was on its way back to Upington. He therefore returned to Upington to make the necessary veterinary arrangements for the oncoming mounted troops, e.g. mallein testing as glanders was prevalent.

Fig. 19.

S.A.V.C. No. 5 SECTION.

BASE VETERINARY HOSPITAL, MAITLAND, 1915.

Sitting: Sergt. A. Gibson; Sergt. W. Papenfus; Q.M.S. W. Rice; S/Sergt. W. Averre; Capt. A. Goddall (O.C.); Capt. McKie; Sergt. P. Johns; Sergt. H. Oldfield; Sergt. Disp. N. Rollett.

Subsequently convalescent depots were opened at Rehoboth, Keetmanshoop and Kume.

The A.D.V.S. after subsequently undertaking certain duties at Kimberley and Cape Town returned to Upington, where he remained from 24.11.1915 to early January, 1916, when the Base Veterinary Hospital (No. 1) was closed.

(2) *Eastern Force.*

The country through which the above force trekked was not only semi-arid (in fact desert in places), but also badly infected with anthrax. Fortunately only a few cases occurred among the transport oxen. Advance veterinary hospitals were established at Kuruman and Boesmansput and other sick animals were evacuated to the Base Veterinary Hospital at Kimberley.

As the force proceeded westwards "E" mobile Veterinary Section (Capt. W. Dykins) brought up the rear and collected casualties. On reaching German territory, rest camps and sick lines were established on farms where grazing and water were available.

The general officer commanding later reported satisfactorily on veterinary services as having been "efficiently carried out" (p. 388).

(3) *Central Force.*

All that is recorded of the veterinary side of the final phase of the operations of the Central force is that sick animals were embarked at Luderitzbucht and forwarded to No. 5 Veterinary Hospital at Parow, near Cape Town.

(4) *Northern Force.*

Accompanying the contingent commanded by Col. Skinner, which occupied Walvis Bay on Christmas Day 1914, were three animal transports with approximately 2,000 animals on board. Blenkinsop and Rainey (p. 395) gives the veterinary arrangements as follows : "One V.O. with a veterinary section on each transport, *viz* S.S. ' Monarch,' S.S. 'Rufidgi' and S.S. 'Glenorky.' Prior to leaving Cape Town the necessary supply drugs, instruments etc. were issued from the base veterinary store and two full sets of veterinary hospital ([27]) equipment and drugs were placed on board as a reserve for future requirements. The casualties on the voyage totalled six which was satisfactory considering that some of the animals were not disembarked for a fortnight, the delay being due to poor off-loading facilities and lack of fresh water ashore. The latter factor contributed largely to the loss of condition of the animals on shore. As soon as they became accustomed to the brack water, condition improved. Most animals were shod in front but not behind.

([27]) A base veterinary hospital was established at Walvis Bay.

As there was no grazing, it was necessary to raise the ration scale of oxen to 30 lbs (? of hay) per day. As facilities for watering improved, so animals were allowed to drink twice a day instead of once as originally.

Altogether 43,000 animals were landed at Walvis Bay and all transports on arrival were visited by an inspecting V.O.

"Generally speaking, the fittings in the transports were good. In some cases the stalls were too short .... and floor battens were omitted in some of the iron decks, necessitating the free use of ashes to give .... a foothold."

"The arrangements at Walvis Bay for off-loading animals were good, although instances of carelessness occurred in the slinging overboard on to the rafts." The transport *British Prince* had "special off-loading gangways which permitted animals to be walked from the ship on to the rafts" (p. 396).

At first animal management on board was not good in spite of the fact that "detailed instructions .... had been issued from the D.V.S's office." Faults which were not speedily combated at the outset were : (a) animals not watered, (b) mangers full of mealies and (c) too many animals on board and gangways packed. Later, however, with supervision, matters improved.

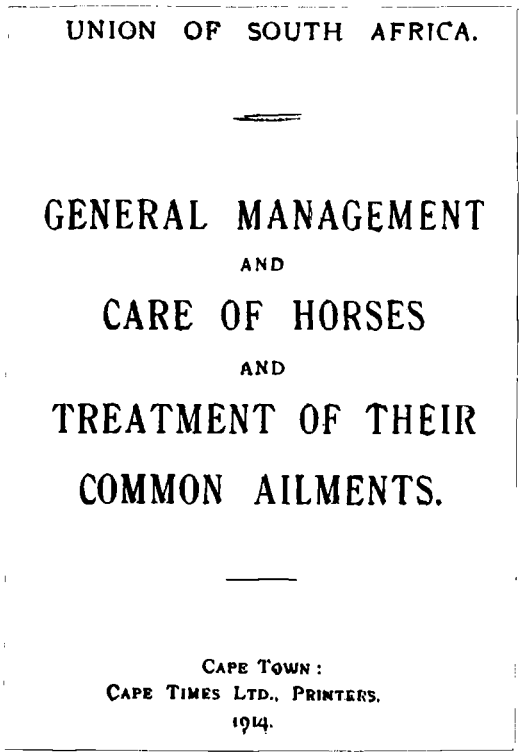
It has been seen how after the first advance from Swakopmund during the period 18.3.1915 - 24.3.15, Botha had been compelled to return his mounted troops to the coast "where they could be fed from the ships." Particular attention was paid to the care and management of the animals so that when the second advance to the railway, between Karibib and Okahandja, took place, during the last week of April and first week of May, 1915, "they were in superb condition. This enabled them successfully to undertake one of the most arduous campaigns on record for animals through desert country. For days they were without food and water, and when this was available, it was only in restricted quantities .... Very few animals were destroyed on trek. Large numbers fell out and were collected and sent back to Karibib. Owing to the difficulties in getting forward supplies of forage in sufficient quantity the animals became extremely emaciated and useless for further service. Eventually supplies of grain . . . arrived, but as there was no grazing or other bulky fodder ... numbers of cases of colic occurred" (p. 399).

Prior to the third advance i.e. to the north (over the period 18.6.1915 to 9.7.1915) "the various mounted brigades were stationed along the railway from Usakos to Windhoek" (p. 399).

The veterinary arrangements were : A.D.V.S., Windhoek ; 1 O.V. with mobile veterinary section at Windhoek and the same at Klein Aukas ; 1 O.V. with mobile veterinary section for each of the mounted

brigades and the infantry brigade and 1 V.O. with the artillery and another with the Imperial L.H.

As has been described, the interval between the second and third advances was used to accumulate supplies, and "some considerable time elapsed before the animals were placed on full rations." In fact, at the time of the advance the majority of the horses and mules were not in good condition, and could not be regarded as fit for the strenuous work in front of them."



UNION OF SOUTH AFRICA.

GENERAL MANAGEMENT

AND

CARE OF HORSES

AND

TREATMENT OF THEIR

COMMON AILMENTS.

CAPE TOWN:
CAPE TIMES LTD., PRINTERS.
1914.

FIG. 20.

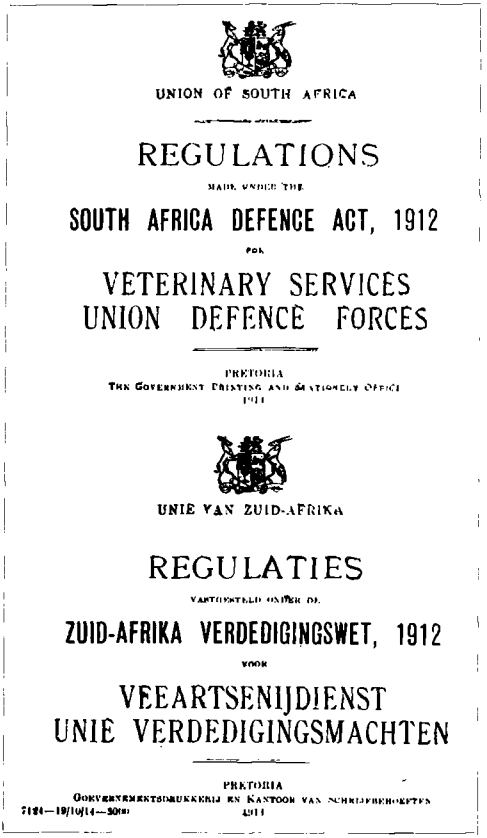
When the third advance took place "approximately 10,000 debilitated animals were left behind, 7,000 of which were in the vicinity of Windhoek. These animals belonged to the *Southern* Force ([28]) and the 3rd Mounted Brigade. The other 3,000 were collected at Karibib and afterwards moved to Klein Aukas near Usakos" (p. 399).

As all supplies coming north were being sent to the troops participating in the final (third) advance, the losses from starvation among

([28]) In view of the fact that the mounted troops of the original Southern Force returned to Upington, it is clear that the animals referred to here belonged to the original Central and Eastern Forces which, together with the Southern Force, constituted the Southern Army disbanded as such early in May.

the 10,000 animals left behind were considerable. In spite of the fact that the A.D.V.S. recommended that the 3,000 animals at Klein Aukas "should be sent back to the coast, where large quantities of grain and forage were available, and eventually embarked and shipped to Cape Town in the empty transports returning there," nothing was done.

The final advance was a remarkably rapid movement, some of the brigades trekking over 40 miles a day. "The transport was left behind in many cases ; and at the time of the surrender of the German forces,



UNION OF SOUTH AFRICA

REGULATIONS

MADE UNDER THE

SOUTH AFRICA DEFENCE ACT, 1912

FOR

VETERINARY SERVICES
UNION DEFENCE FORCES

PRETORIA
THE GOVERNMENT PRINTING AND STATIONERY OFFICE
1914

UNIE VAN ZUID-AFRIKA

REGULATIES

VASTGESTELD ONDER DE

ZUID-AFRIKA VERDEDIGINGSWET, 1912

VOOR

VEEARTSENIJDIENST
UNIE VERDEDIGINGSMACHTEN

PRETORIA
GOEVERNEMENTSDRUKKERIJ EN KANTOOR VAN SCHRIJFBEHOEFTEN
7124—19/10/14—500 1914

FIG. 21.

the horses and transport animals were exhausted. Hundreds of animals dropped out and were left behind on the road" (p. 400).

Most of these were collected later by the mobile veterinary sections and taken to farms where water and grazing were available. Convalescent depots were established at Otavifontein, Kalkfeld, Omaruru, Karibib, Usakos, Okahandja and Windhoek.

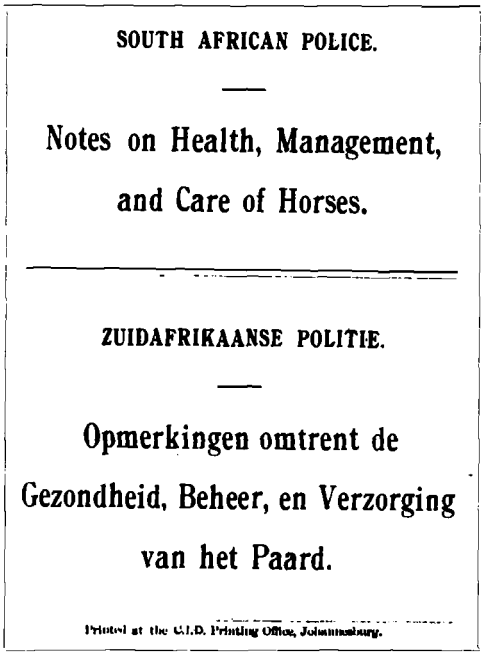
The greatest loss occurred after the surrender when "orders were issued for troops to return to the railway, to leave their animals behind

and proceed back to the Union for demobilisation . . . .

It was a case of the survival of the fittest. Riders left their horses anywhere on the veld and many returned on empty transport wagons" (p. 400).

Here again good work was done by the mobile veterinary sections, but as a result of the difficulties regarding water and grazing, losses were considerable.

As mentioned before, it was the post-campaign period (i.e. up to the end of 1915) that proved so onerous for the Veterinary Service, and some detail will now follow illustrating some of the difficulties encountered.



SOUTH AFRICAN POLICE.

Notes on Health, Management, and Care of Horses.

ZUIDAFRIKAANSE POLITIE.

Opmerkingen omtrent de Gezondheid, Beheer, en Verzorging van het Paard.

Printed at the G.I.D. Printing Office, Johannesburg.

FIG. 22.

(iv) *Post-Campaign Period.*

(1) For veterinary services demobilisation is always a difficult phase ; but in the circumstances, where animals had been cast aside in semi-arid country, it was doubly so.

Blenkinsop and Rainey (p. 401) state that "the transport and remount departments were faced with the problem of having 60,000 more or less debilitated animals on their hands . . . with practically no food with which to feed them. The railways were unable to deal with the congested traffic. All chartered transports with the exception of two were released. The animals were in too debilitated a condition to

trek to the Union, a distance up to 750 miles, part of which was through a waterless desert. Attempts were made to trek the animals from Usakos to Walvis Bay on the coast, where thousands of tons of feed were stored, but the route was strewn with carcases ... The officers in veterinary charge of each depot continued to send urgent telegrams .... (but) it was not until November that anything in the nature of sufficient supplies were available."

The remount authorities endeavoured to distribute the animals over farms "but owing to their weak state they were unable to travel .... in many instances they were reduced to eating their own excreta." During

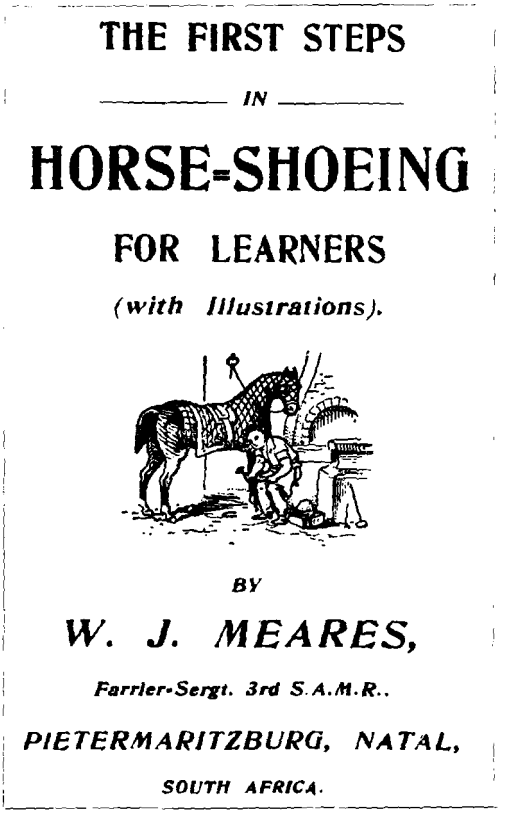


FIG. 23.

the four months following the surrender "several thousand animals died from starvation or were shot for debility" (p. 401). ([29])

([29]) On 21.8.1915 the D.V.S., while on a tour of inspection in the occupied territory, interviewed the military governor at Windhoek and emphasised that a grave crisis would arise if the railway could not furnish animal rations. The needs, however, of the human population were greater, so thousands of animals died of starvation.

The following day the D.V.S. interviewed the special remount officer detailed to deal with the removal of all animals from the Protectorate, but he stated he "did not wish interference from outside sources" (p. 403).

(2) Another problem, but one concerning essentially veterinary services, was the prevalence of communicable disease, especially glanders. Debilitated animals were of course particularly susceptible to the specific maladies, and veterinary officers, not only in the Protectorate (the new status of the ex-German Colony) but in the Union were kept busy for many months, eradicating glanders. Smith records that in South-West Africa "a total of 46,682 animals were tested and retested with mallein ... at twenty-two different centres with the result that 3,313 reactors and 840 clinical cases were destroyed, a large proportion of which were German horses ... The disease also broke out among the transport donkeys numbering about 4,000 at two different centres and about 106 were destroyed" (p. 403). Owing to the acute nature of glanders in donkeys the mallein test was found to be unreliable.

The main centre of glanders infection in the Union was at the Maitland transport and remount camp, where much work was done towards establishing the intrapalpebral mallein test as the routine test in place of the subcutaneous method. Under Capt. Goodall 9,000 animals were tested by this procedure and 864 were destroyed([30]). At Kimberley, army equines were also mallein-tested on a large scale before sale. Other V.O's. used the intrapalpebral test in the field and Howie reported favourably on the method as used at Walvis Bay and Swakopmund.

Other communicable diseases dealt with satisfactorily were mange (1,648 cases at Upington), ringworm, strangles, nasal catarrh and one outbreak of equine influenza, which "occurred in a mounted regiment stationed at Fredericksveld, a German stud farm" (p. 397).

As would be expected, sand colic was the cause of much trouble, as were pneumonia and lameness, the last-mentioned due to the lack of shoeing facilities in some units not provided with shoeing smiths. A case of dourine in a stallion belonging to the German Agricultural Department was detected in 1915.

The S.A.V.C. not only eradicated the communicable diseases referred to above, but also combated scourges such as pleuro-pneumonia in cattle and scab in sheep.

(3) The work of the S.A.V.C. included the care of captured pedigree livestock, particularly Karakul sheep and all veterinary stores, chiefly at Gamans Veterinary Laboratory, prior to handing over to the civil authorities.

([30]) Goodall, in a paper (1915), unfortunately took entire credit for this research. It may be stated that he received much assistance in regard to literature, concentrated mallein and technique from a colleague who had recently been through Sir John MacFadyean's laboratory at the Royal Veterinary College, London.

(4) *Statistics on mortality of livestock* (a) *under veterinary care and* (b) *outside veterinary lines.*

As has been stated previously the number of animals under veterinary care totalled 160,000.

(a) The subjoined table gives details of animals treated by the S.A.V.C. from 4.8.1914 to 31.1.1916 :—

	Admitted.	*Discharged.*	*Dead.*	*Death rate 17 months.*	*Death rate per annum.*
Horses	60,023	50,341	4,611	7.6%	5.3%
Mules	12,168	8,806	983	8.07%	5.6%
Donkeys	2,968	1,830	506	17.04%	12.0%
	75,159	60,977	6,100	8.12%	5.72%

	Deaths outside veterinary lines.	*Deaths inside veterinary lines.*	*Total deaths*
Horses	8,715	4,611	13,326
Mules	2,136	983	3,119
Donkeys	2,452	506	2,958
Oxen	2,559	——	2,559
	15,862	6,100	21,962

The grand total of all details is 21,962 which equals 13.7% for 17 months and corresponds to an annual death rate of 9.09%. Under the circumstances this indicates the efficiency of the veterinary organisation.

(5) *Closing down of depots.* The veterinary units at the following depots were demobilised at the end of August, 1915 :— Tweespruit and Standerton remount farms, Tempe, De Aar, Draghoender, Border, Potsdam, Mellish's farm, Parow, Papkine farm, Boesmansputs, Kuruman, Warrenton, Fourteen Streams, and Kroonstad.

At the same time the base veterinary store at Maitland was transferred to Johannesburg.

On 12.1.1916 a conference was held at Upington regarding the closing down of all remount depots in the Protectorate and the transfer to the civil authorities of transport, remount and veterinary establishments. On the 17th and 19th *id.* the base veterinary hospitals at Upington and Parow were closed.

(v) Veterinary Officers in Campaign.

By studying the Government Gazettes of the period it is possible to trace the transfers of the several V.O.'s. from the pre-Union units

to their final appointments in the S.A.V.C., A.C.F. Space, however, does not permit the inclusion of such data e.g. Capt. S. I. Johnston, who was in the Southern M.R. (Transvaal) before Union, joined the T.V.C. When the A.C.F. was created as from 1.7.1913 he became regimental V.O. to the 10th M.R. (Botha Ruiters); but on the formation of the S.A.V.C. he transferred to that unit.

Fig. 24.

Major RUNCIMAN.

The list given by Blenkinsop and Rainey (p. 46) is that of V.O.'s. who saw service in the Union e.g. during the Rebellion and/or on German territory. The officers in question were :—

D.V.S. — Col. J. Irvine-Smith.* See *Jl. S.A.V.M.A.*, Sept., 1939.

HYGIENE SPECIALIST :— Lt.-Col. C. E. Gray. See *Jl. S.A.V.M.A.*, Sept., 1937.

A.D.V.S. — Lt.-Col. J. G. Bush.* See *Nongquai*, Sept. 1924.
Lt.-Col. G. W. Lee.
Major G. T. Cannon.
Major B. Runciman.

MAJORS: S. T. Amos.
J. B. Collyer — V.O. to the P.F.
W. Jowett.
C. M. Sharpe.

CAPTAINS :

W. H. Andrews.*
C. H. Brogan.
M. Cunningham.
J. W. Crowhurst.
E. T. Clemow.
F. J. Dunning.
J. Donaldson (1877 - 1935).
W. A. Dykins.
S. Elley (1877 - 1941).
J. Forrest.
G. Goodliffe.
A. Goodall (1879 - 1930).
A. F. Harber.
A. M. Howie.
A. Hodder.
J. F. Joyce (1877 - 1926).
S. I. Johnston (1866 - 1929).
E. Kellett (Died 6.9.1932.)
D. B. McCall.*
G. R. McCall (1885 - 1930).
J. McNeil.
J. G. McGregor.
B. A. Myhill.
W. McKie.
J. K. Pilkington (1857 - 1941).
J. Peddie (1870 - 1932).
J. Quinlan.
C. H. Strachan.
F. N. Skues (1866 - 1921).
E. E. Stokes (1867 - 1925).
J. Thompson.
P. R. Viljoen.
C. H. Wadlow.
J. A. Worsley (1866 - 1914).

* Mentioned in despatches.

Actually an unqualified veterinarian, Mr. Heaton of Wynberg, C.P., accompanied the 17th Western Province M.R. to G.S.W. Africa as V.O. He is included in a photograph of the officers of the regiment taken at the time.

Not all the officers mentioned performed full-time service or indeed, wore uniform. Lt.-Col. Gray continued his duties as P.V.O. of the Civil Veterinary Department and Capt. J. Forrest, in addition to his municipal duties at the Maitland Abattoir, was appointed to take charge of the Table Bay Harbour Board Stables.

Capt. Goodliffe, attested as No. 129, sergeant, but was given his commission (as captain) on 28.2.1915.

After the campaign, Capt. Forrest went to England and joined the R.A.V.C. and Capt. Myhill proceeded to France with the S.A. Heavy Artillery.([31])

Not all officers of the S.A.V.C. were called up for active service, e.g. Capt. D. T. Mitchell, who was commissioned on 1.5.1916, could not be spared from his civil veterinary duties.

([31]) It is convenient here to pay a tribute to other S. Africans who proceeded to Great Britain to join the R.A.V.C. They are: F. C. Cavin, J. Keppel, C. H. Cordy, F. C. Simpson and G. H. Melck. B. S. Parkin, who qualified oversea, joined the R.A.V.C. and served in Afghanistan.

(vi) AWARDS.

C.B.E. — Lt.-Col. J. Irvine Smith.
D.S.O. — Major G. W. Lee ; Major A. F. Harber.
M.C. — Capt. P. R. Viljoen.
M.S.M. — Sergeant F. W. Norman.

(vii) CASUALTIES.

Capt. J. A. Worsley died at Prieska on 21.8.1914.

According to appendix 2 of the *Official History of the Great War* the number of persons in the S.A.V.C. who were killed in action or died as the result of active service was 17. For further particulars see p.....

Obituary notices in respect of officers who have died since the First Great War have appeared from time to time in the *Jl. S.A.V.M.A.*

(*To be continued*).

OBITUARY.

COL. GEORGE WILLIAM LEE (1872 - 1943).

Col. Lee was born on 25.9.72 in England and qualified at the Royal (Dick) Veterinary College in 1893. He first came to South Africa as a member of the 2nd English rugby team in 1896 and thereafter took part in the Mashona Rebellion of the same year. On the outbreak of the rinderpest he proceeded to the Victoria Compound, Kimberley early in 1897 in order to study rinderpest immunisation under Professor Koch. He then returned to Salisbury, together with C. E. Gray, Osborne and A. Neill all of whom had received appointments under the Chartered Company. In 1898 he joined the Southern Rhodesian Volunteers and on the outbreak of the Boer War was appointed Veterinary Officer to the transport of the Rhodesian Field Force under General Carrington. On the disbandment of this Force in September 1900 he returned to civil duty for a short time and was the Veterinarian who inspected on board ship at Beira the 1,000 Australian cattle which are believed to have brought East Coast fever into Southern Rhodesia.

From the end of 1900 until the termination of hostilities, LEE was a member of Kitchener's Fighting Scouts under Col. Colenbrander. Later he was attached to the Right Wing and W. C. Lowe (formerly of the Cape C.V.D.) to the Left Wing. For his services in this campaign he received the Queen's and King's medals with six bars. He left the Southern Rhodesian Service in 1902.

He then received an appointment in the Transvaal Repatriation Department and later (as from 30.9.1903) entered the C.V.D. For six months (1904 - 5) Lee was sent on special duty to the Bechuanaland Protectorate in view of the spread of East Coast fever in adjoining territories. On the outbreak of the Great War, when he proceeded on active

service with the S.A.V.C. to German South West Africa, and ended up as A.D.V.S.

During the military occupation of South West Africa (1915 - 1919) Protectorate, Lee was appointed Director of Agriculture and with a small staff of veterinary officers was kept busy controlling epizootics such as glanders, anthrax, scab etc.

In 1920, when a civil administration was established, Lee resigned from the service and took up ranching in South-West Africa in which venture he was eminently successful.

He was an enthusiastic member of the T.V.M.A. In 1920 he resigned from the S.A.V.M.A.

H.H.C.

NOTES.

During the last three months the following veterinarians have paid visits to Onderstepoort during their leave from the British Colonial Service :—

Mr. R. J. Roe, Chief Veterinary Officer, Nicosia, Cyprus.

Mr. G. C. Brander, District Veterinary Officer, Malakal, Southern Soudan.

Mr. R. R. Temple, District Veterinary Officer, Iringa, Tanganyika.

The following candidates were successful in the final B.V.Sc. Examination in June : W. A. de Waal, C. E. Isaacs, A. J. Louw, S. A. R. Stephan, U. A. von Bäckstrom and D. M. Walters. Messrs. de Waal, Louw and Stephan have been appointed in the Division of Veterinary Services and have been posted to Umtata, Ixopo and Vryheid respectively. Messrs. von Bäckstrom and Walters have joined the Bechuanaland Administration.

The pup kindly donated by Mr. Hans Theiler to be disposed of on behalf of the Benevolent Fund realised £12.18.0 The winner was Mr. A. C. Kirkpatrick.

The receipt of a donation of £5.5.0 towards the Benevolent Fund from Col. C. M. Sharpe is also gratefully acknowledged.

Dr. C. C. Wessels has now recovered from his long and serious illness and commenced duty at Onderstepooort in August.

Mr. D. E. Faulkner has been appointed Principal Veterinary Officer, Swaziland, vice Mr. W. A. Elder who has retired.

MOVEMENTS OF OFFICERS.

Mr. P. L. Uys appointed government veterinary officer, Dundee, 14.4.43.

Mr. S. A. R. Stephan appointed government veterinary officer, Vryheid, 21.7.43.

Mr. W. A. de Waal appointed government veterinary officer, Umtata, 21.7.43.

Mr. A. J. Louw appointed government veterinary officer, Ixopo, 29.7.43.

Dr. C. C. Wessels appointed veterinary research officer, Onderstepoort, 1.9.43.

Mr. F. E. Cavanagh resigned to take up private practice with Mr. S. T. Amos in Durban, 30.4.43.

SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION.

BALANCE SHEET AS AT 31st MARCH, 1943.

LIABILITIES.

Benevolent Fund	£588	17	3			
Plus Subscriptions	83	5	0			
,, Profit on Group Endowment	28	7	4			
,, Interest	29	17	3			
	730	6	10			
Less Assistance Payments	48	0	0			
				£682	6	10
Prize Fund	162	17	1			
Plus Profit Book Fund	26	5	6			
,, Interest	6	12	4			
				195	14	11
Natal Branch	10	2	0			
Plus Subscriptions	0	10	0			
				10	12	0
General Fund	1,952	1	9			
Plus Profit, 1942-43	129	3	8			
				2,081	5	5
				£2,969	19	2

ASSETS.

Investments plus accrued interest:								
Government 3½% Local Regd. Stock	£405	0	0					
Government 3¼% Local Regd. Stock	202	14	2					
Union Loan Certificates	1,735	15	0					
United Building Society	101	6	8					
Standard Building Society	22	5	0					
				£2,467	0	10		
Student Advances				214	9	6		
Group Endowment Insurance Surp. A/c.				4	1	4		
Subscriptions due	198	8	6					
Less paid in advance	5	6	6					
				193	2	0		
Cash in hand	15	16	5					
Cash at Bank	75	9	1					
				91	5	6		
				£2,969	19	2		

SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION.

INCOME AND EXPENDITURE ACCOUNT FOR THE YEAR ENDING 31st MARCH, 1943.

EXPENDITURE.				INCOME.			
To. Interest Book Fund			£6 12 4	By Subscriptions accrued			£309 9 0
,, Interest Benevolent Fund			11 17 6	,, Interest accrued			71 16 8
,, Stationery			8 10 0				
,, Clerical Assistance and Typing			48 0 0				
,, Expenses General Meeting			2 18 10				
,, Wreath			1 1 0				
,, Bank Charges			4 6 4				
,, Printing and distributing Journal	£233 17 11						
Less Adverts. and Subscriptions	86 1 5						
			147 16 6				
,, Miscellaneous Expenses			20 19 6				
,, Balance Profit transferred to General Fund			129 3 8				
			£381 5 8				£381 5 8