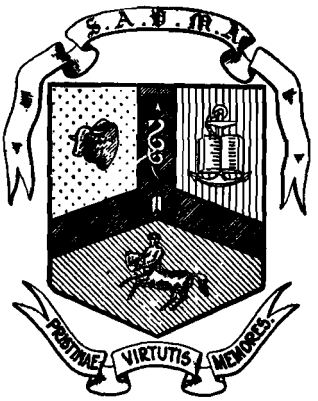
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DIE TYDSKRIF
VAN
DIE SUID-AFRIKAANSE VETERINÊR-MEDIESE VERENIGING

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UNVEILING OF THE THEILER BUST

The frontispiece in this issue is a photo showing Prof. H. O. Mönnig receiving a bust of Arnold Theiler on behalf of the University of Pretoria and the Faculty of Veterinary Science from Coert Steynberg, famed South African sculptor. This occurred subsequent to the unveiling by Dr. Gertrud Theiler.

Some years ago, at an Annual General Meeting of the S.A.V.M.A., it was suggested that persons of exceptional merit in the field of Veterinary Science or who had rendered outstanding services to the veterinary profession should be honoured by means of some memorial or other in the Faculty buildings or grounds. This suggestion was put to Faculty, who accepted it whole-heartedly, and who extended the idea to include events as well. The most obvious event was the founding of Faculty. During deliberations as to the form such a memorial should take, Coert Steynberg donated a bust of Arnold Theiler to the University of Pretoria, who in turn handed it to Faculty. That finalised matters and after an appeal to old students they, as well as students, Faculty members, graduates from other veterinary colleges and even non-veterinarians contributed to erect an imposing pedestal of black granite for the bust in the atrium of the Faculty block.

The unveiling took place on August 26th, during the last Annual Congress held at Onderstepoort. The present Dean, Prof. H. Graf, briefly explained the origin of this memorial, as well as the fact that plans were being made to provide a background of light grey marble flanked by columns in half-relief. Two bronze plates will carry explanatory inscriptions. Prof. P. J. du Toit, past Dean and Director, sketched Theiler's role as teacher and Dean. Mr. Coert Steynberg then handed over the bust. The unveiling and receipt followed: the visible result of the first step to perpetuate Faculty traditions.

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ONTHULLING VAN DIE THEILERBORSBEELD

As titelplaat verskyn in hierdie uitgawe 'n foto van die oorhandiging van 'n borsbeeld van Arnold Theiler deur die gevierde Suid-Afrikaanse beeldhouer, Coert Steynberg, aan prof. dr. H. O. Mönnig, wat dit namens die Universiteit van Pretoria en die Fakulteit Veeartsenykunde in ontvangs neem. Dit het geskied na die onthulling deur dr. Gertrud Theiler.

Reeds enige jare gelede is daar op 'n Algemene Jaarvergadering van die S.A.V.M.V. die gedagte geopper dat persone, wat besonder op veeartsenykundige gebied gepresteer het en buitengewone diens aan die veeartsenykundige beroep gelewer het, deur middel van een of ander gedenkteken in die Fakulteitsgeboue of gronde vereer behoort te word. Die gedagte is aan die Fakulteit van Veeartsenykunde voorgelê, wat dit van ganser harte aanvaar het en uitgebrei het om gebeurtenisse ook te omvat. Die mees voor-die-hand-liggende gebeurtenis was die stigting van die Fakulteit. Tydens beraadslagings oor die vorm van so 'n gedenkteken het Coert Steynberg 'n borsbeeld van Arnold Theiler aan die Universiteit van Pretoria geskenk, wat dit weer aan die Fakulteit bemaak het. Dit het die saak beklink, en na 'n oproep aan alle oud-studente het hulle, sowel as studente, fakulteitslede, graduandi van ander veeartsenykolleges en selfs nie-veeartse hand in die sak gesteek om 'n indrukwekkende swart graniet voetstuk vir die beeld in die atrium voor die auditorium in die Fakulteitsgebou op te rig.

Die onthulling het plaasgevind tydens die laaste Jaarkongres op 26 Augustus te Onderstepoort. Die huidige dekaan, prof. dr. H. Graf, het die ontstaan van hierdie gedenkteken kortliks aan die gehoor verduidelik, asook die feit dat planne beraam is om 'n agtergrond van ligte grys marmer, weerskante afgeskort deur 'n suil in half-relief, later aan te bring. Twee bronsplate sal die verduidelikende inskripsie dra. Prof. dr. P. J. du Toit, oud-dekaan en oud-direkteur, het daarna Theiler se rol as dosent en dekaan geskets. Mnr. Coert Steynberg het die beeld toe oorhandig. Die onthulling en ontvangsname het gevolg: die tasbare resultaat van die eerste stap om fakulteitstradisies vas te lê was daar.

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CHRONIC RESPIRATORY DISEASE: ITS NATURE AND ERADICATION

J. D. W. A. COLES and R. B. CUMMING
Onderstepoort

Introduction

In 1935, Nelson described "coccobacilliform bodies associated with an infectious fowl coryza". This new coryza was noteworthy for having a much longer incubation period than the two days, taken by Haemophilus gallinarum to produce the respiratory symtoms that were then being studied. Although Nelson wrote a short series of excellent articles, little notice was taken of them, and it was only in 1943 that Delaplane and Stuart virtually rediscovered the infection that is today called C.R.D.

These bald statements may suggest that the disease presents no great difficulties to the investigator. Nothing could be further from the truth, and for reasons that will become obvious in this article. Markham and others, who have done so much to solve some of the many riddles associated with the condition, undoubtedly expressed the feelings of all fellow-workers when they wrote, "We believe that the most important lesson to be learned from the present experience is that C.R.D. is even more subtle and complex than we have yet appreciated and that we have no choice but to go about learning how to recognize and eradicate this disease if the industry is to be relieved of its unwelcome burden. The more we learn about it, the more intolerable it becomes". And intolerable it certainly is, for it is most likely costing South African poultry farmers half a million pounds a year. Not only does it attack broilers and laying birds. Few realise how widespread it is in turkeys, where it is known as infectious sinusitis.

The Etiology of C.R.D.

In its simplest form chronic respiratory disease is due to a pleuropneumonia-like organism, or P.P.L.O., called Asterococcus avis or Mycoplasma gallinarum. Different strains vary in virulence and often an infected flock shows no symptoms, or only a little nasal discharge and snicking of a transient nature.

When the Onderstepoort flock of White Leghorns was first submitted to the rapid plate test in 1957, about 80% of the fowls reacted. Yet accurate records kept over a long time, revealed that only one pullet in 200 ever developed signs of any respiratory disease during the first year of production.

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Undoubtedly, other strains exist that are much more pathogenic, and these include the neurotropic ones that produce a sinusitis and aerosacculitis in turkeys only if they do not kill them earlier with encephalitis.

Outside influences may worsen matters. The introduction of infectious bronchitis virus into a susceptible flock harbouring P.P.L.O. will produce the usual I.B. picture, followed within a few days by a typical outbreak of C.R.D. A flare-up can also be expected if the I.B.-virus simply multiplies in birds that are sufficiently immune to prevent the development of bronchitis. Infection with the virus of Crawley and Fahey or even the virus of Newcastle disease may precipitate an outbreak of C.R.D.

Although Escherichia coli will apparently not bring on the disease, it seems certain that particularly the b, c and d types of this bacterium multiply markedly in the respiratory system and become a menace in the presence of P.P.L.O., especially in clinical cases of C.R.D. These coliforms, which enter the body through the respiratory tract, are mainly responsible for the severe lesions of pericarditis and fibrinous perihepatitis that are frequently seen in young birds in bad outbreaks of C.R.D.

In South Africa, where C.R.D. is so rife, there is no reason to suspect the presence of Newcastle disease, infectious bronchitis, infectious laryngotracheitis or the virus of Fahey and Crawley. There is almost certainly another and very important predisposing factor here, but so far it has not been found.

What has been said above about the fowl applies equally to the turkey, but in this bird the viruses of infectious bronchitis and infectious laryngotracheitis play no role.

P.P.L.O. Types

Pleuropneumonia-like organisms are probably bacteria and are the smallest that can be grown, and then only poorly, in the absence of living cells. They vary in size from about 125 to 250 $\mu\mu$, and on solid media the colonies may be as small as 10 to 20 μ in diameter and seldom exceed 600 μ. While many are saprophytes, growing even in sewage, a few are pathogens and include those responsible for contagious pleuropneumonia of cattle, contagious agalactia of sheep and goats, contagious pleuropneumonia of goats and C.R.D. of fowls and turkeys. The significance of several is still uncertain. All are resistant to penicillin, but some are slightly susceptible to the broad-spectrum antibiotics. Giemsa's stain is the best.

There are three important serological types of avian P.P.L.O. and there is no cross-agglutination between them.

1. The pathogenic type. All of these strains produce sinusitis and aerosacculitis in poults, when injected into the infraorbital sinuses and air sacs. They include the neurotropic strains. They tend to colonise poorly on serum enriched agar and after 72

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hours the colonies do not exceed 0.1 mm in diameter. Coccoid forms predominate. They are haemagglutination positive (HA.). Acid is formed without gas in dextrose and maltose, but lactose and mannitol are unaltered. Sucrose is fermented very slowly. So far nobody has proved that C.R.D. can appear in flocks not infected with these so-called pathogenic strains, even though pure cultures of them may fail to evoke visible disease in experimental chickens.

2. The non-pathogenic type. These HA-commensals are readily propagated on artificial media and the colonies often measure 0.4 mm. in diameter within 48 hours. Ring forms are common. This type is non-pathogenic for fowls and turkeys. Many of these strains fail to ferment dextrose, maltose, sucrose, mannitol and lactose. Some produce acid in maltose, dextrose and sucrose, but not in lactose and mannitol. If sucrose is attacked, it is usually in less than 4 hours. Besides inhabiting the respiratory tract, these strains are to be found in the oviduct and elsewhere.

3. The N type from turkeys. This type has been described by Adler and co-workers in California and is widely distributed in turkeys there. It may produce a moderate degree of aerosacculitis in poults, but not sinusitis. So far it has not been described in the fowl. It is not known whether the infection occurs in the Union and whether it is of any real consequence anywhere.

Apparently all these P.P.L.O. may be found in newly laid eggs, in dead embryos, in pipped eggs and in recently hatched chicks and poults. Dead embryos may show aerocystitis and swollen joints as a result of infection with pathogenic strains. It is impossible to predict the percentage of infected eggs laid by any one hen or flock, and there is no doubt that the figure is usually, but certainly not always, much lower than previously supposed. Four or five thousand clean chicks may sometimes be hatched without a break from eggs from a flock full of reactors, admittedly when no symptoms are showing. Van Roekel and others suggest collecting eggs only when the disease is completely dormant, and this practice is followed in South Africa.

The fact that only non-pathogenic P.P.L.O. can often be isolated from asymptomatic reactors, and even frank cases of C.R.D., may be disturbing. Jungherr has found that pathogenic strains may peter out within four weeks and be replaced by vigorously growing non-pathogenic P.P.L.O. Very likely a few slowly multiplying pathogenic organisms persist, but cannot readily be isolated when overwhelmed by the others. It is probable that a flock must be exposed to a pathogenic strain, if several birds are to react well to an antigen of the same pathogenic type.

Pleuropneumonia-like organisms are spread not only by the sale of infected chicks. Over distances of a few yards they may be carried by air currents. And since the nasal and oral secretions are often infected, P.P.L.O. may be easily distributed through the

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feed and drinking water, not to mention the droplets discharged by sneezing.

P.P.L.O. often die within 24 hours at room temperature, and Adler has twice failed to infect poults by putting them into pens vacated the previous day by severely diseased chickens. Nevertheless, while knowledge of C.R.D. remains incomplete, it is perhaps wise to leave premises vacant for one month when eradicating the infection. An added advantage is that Haemophilus gallinarum, Pasteurellas and any viruses that may also be present will succumb within this time.

Some investigators, including Gentry and Kelton, have claimed that P.P.L.O. represent the L-forms of various bacteria. It is doubtful if their views are correct.

Diagnosis and Treatment of C.R.D. and Infectious Sinusitis

The prepatent period of C.R.D. is roughly one to two weeks. It is essentially a disease of chickens five to twelve weeks old, but birds of all ages may sicken. Symptoms are often seen in chicks of only three weeks, on badly infected broiler plants. Generally speaking, adult birds suffer less severely, and the male is the more susceptible sex. The disease spreads slowly and is characterised by nasal discharge, sinusitis, rales, anorexia, poor growth and often gasping, which may be observed only every three to five minutes. The alarming dyspnoea often seen in the early stages usually subsides in a day or two. This remission, due to purely natural causes, is of course, always attributed to any drugs that may have been used. Hatchability and early chick livability may fall by 5%. Egg production may drop by 30%. Though morbidity may exceed 90%, mortality usually varies between 10 and 40%. Egg shell quality is not affected, as it is in Newcastle disease and infectious bronchitis. It usually takes two or three months for an outbreak to run its course in a flock.

Pus, not to be confused with normal mucus, is often present in the nasal cavities, even though the face looks pinched. There may be laryngitis and tracheitis and the air sacs often contain purulent material, sometimes in very large quantities in emaciated birds. The lungs generally show surprisingly little change. A wad of yellow pus may be found behind the gizzard. Fibrinous pericarditis and perihepatitis are common complications. There may be enteritis in birds with a yellowish diarrhoea.

Treatment gives poor results. At considerable cost, the secondary infection may be held temporarily in check, or even reduced appreciably, by feeding antibiotics like oxytetracycline and chlortetracycline at high levels of 100 to 500 grams per ton of mash. Furazolidone and magnamycin may help a little. The injection of drugs is no more successful. Penicillin by itself is disappointing.

In the differential diagnosis it is necessary to consider Newcastle disease, infectious bronchitis, infectious laryngotracheitis,

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fowlpox, aspergillosis, fowl cholera, rickettsial conjunctivitis, avitaminosis A and Haemophilus gallinarum infection. As two or three of these diseases may be found simultaneously in a flock, the diagnosis may be difficult. Fortunately, some of the conditions are not found in the Union.

Infectious sinusitis of turkeys takes 8 to 30 days to develop. Morbidity may reach 50% and mortality 25%. The disease is often to be seen in poults three weeks old. One or both infraorbital sinuses swell up and contain a jelly-like material, which later may harden to some extent. Foam is usually seen in the eyes. If the sinuses are badly swollen, the bird cannot see and eat properly. Aerocystitis may develop and lead to dyspnoea, particularly when the P.P.L.O. infection is complicated by some other viral or bacterial agent. Growth is impaired. Some turkey P.P.L.O. strains are, of course, capable of producing a lethal encephalitis.

Only if the lesions are restricted to the sinuses is treatment likely to avail. Inject into the sinus 100 to 200 milligrams of an antibiotic like erythrocin, oxytetracycline or streptomycin. A second injection may be necessary a week later.

Ornithosis, fowl cholera, aspergillosis, Newcastle disease and avitaminosis A must be taken into account in the differential diagnosis. As yellow maize and lucerne meal often have little or no vitamin A activity mashes should be fortified accordingly. The cost is negligible.

The Eradication of C.R.D. and Infectious Sinusitis

Here it is necessary to bear certain facts in mind, some of which have already been mentioned.

Natural immunity is very poor and it is improbable that a good vaccine will be developed. Many birds remain carriers and may lay infected eggs, though the disease be subdued by feeding or injecting an antibiotic. Naturally, the cost of such medication is usually prohibitive, as it is even when the drug is fed only to broilers for four or five weeks to assist them to retain their weights to marketing age.

The fowl, turkey and duck are all susceptible, but virtually nothing is known about the goose. The pigeon, partridge and pheasant may harbour P.P.L.O., but Crawley believes that they are probably not a menace to the poultry industry in Canada. So far there is nothing to suggest that the position is dissimilar in South Africa.

Because it is the practice here for broiler raisers to buy day-old chicks weekly, and have birds of all ages up to twelve or fourteen weeks on their plants, it is absolutely essential for these to be free of all diseases transmissible through the egg. Otherwise disaster is inevitable, and it has already been shown that the average farmer, who cannot buy clean chicks regularly, is forced out of business by C.R.D. within two years. It is altogether differ-

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ent in North America. There they usually put through three to four batches of several thousands per year, and leave the houses empty for three or four weeks between any two lots. Consequently if C.R.D. does break out, it will spread slowly, and after the affected broilers are marketed the infection will die out — before the new chicks arrive.

Pathogenic P.P.L.O. may exist in a flock without clinical signs of C.R.D. or infectious sinusitis being present, but there can be no evident disease in the absence of P.P.L.O. Therefore, the only safe flock is a P.P.L.O. negative flock, and this fact cannot be over-emphasized.

Although the HI test is probably the best, particularly in the case of turkeys, the tube agglutination test, and the rapid plate test with serum or plasma and stained antigen are very useful. The antigen employed here at present for the plate test is prepared by American Scientific Laboratories.

The test is purely a flock test. In other words, it is employed simply to determine what flocks are infected, and not which individual birds harbour P.P.L.O. Experience has shown that it is impossible to clean up a flock by testing every few weeks and removing reactors, for the organisms are spread much too easily through the air, feed and water and some birds are always in the incubation stage. Besides this, infected birds may react negatively.

When a breeder wishes to clean up, he is told to bring to the laboratory about thirty sexually mature birds, usually culls, selected at random from different houses. From each one about 0.4 ml. blood is run from the wing vein into a glass tube containing one drop of 20% potassium oxalate solution to prevent clotting. The specimens are centrifuged and the plasma is used for the test. All glassware must be scrupulously clean and it is best to do the tests within four or five hours.

In the case of the fowl, plasma (or serum) and antigen are mixed in equal amounts. It may be best to mix three drops of antigen with one drop of turkey plasma. Experience of P.P.L.O. infection in ducks is limited and possibly the HI test should be done. About geese nothing is known. Until more has been learnt about ducks and geese, only fowls and turkeys should be run on P.P.L.O. negative farms.

If the thirty birds are negative, it is a good indication that the others will be the same, unless the P.P.L.O. are just beginning to spread. Where the infection is well established and the birds are on floors, at least 30% will react. The figure is usually 70 to 90%. In batteries, where there is a greater degree of individual isolation, the percentage may be somewhat lower. Agglutination titres also fall steadily during the months following the original exposure to P.P.L.O. Obvious cases of C.R.D. may react negatively, even for a month after the appearance of symptoms. Often only 10 to 20% of sick chickens react to the plate test, when

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brought in by a farmer, and if he is not warned beforehand to expect this, he may lose faith in the test.

It is not uncommon to find what are undoubtedly non-specific reactors. Crawley and Fahey found that a small percentage of adult fowls recently infected with Newcastle disease or infectious bronchitis may have a low HI titre, which vanishes within a month. The plate test not infrequently reveals 4 or 5% of weak or moderately strong reactions in flocks supposed to be clean, particularly when the birds are young. The proper thing to do is to keep flocks with non-specific reactors under observation, and test about 10% of the birds in every house at monthly intervals. If the reactions are really due to pathogenic P.P.L.O., the number of reactors will rise, and at least one third of the agglutinations will be marked and be visible within about thirty seconds. Common-sense and experience are needed for the proper interpretation of P.P.L.O. reactions, as all other biological tests.

If the original thirty birds appear to be negative, an official should inspect the flock and bleed 10 to 15% of the fowls or turkeys over three months old, selected at random in every house and camp. A total of not less than 200 should be bled. If these are negative, it may be presumed that the farm is clean, but during the first year retesting is recommended at intervals of three to six months. Thereafter annual testing should suffice.

If infection exists on a farm, measures to eradicate it must depend on the circumstances. The conditions may be of such a nature that the owner has no option but to depopulate the entire premises for one month, and then restock with day-old chicks from a definitely clean source. At other times simultaneous depopulation and repopulation may be spread over six to twelve months, and the chicks may be bought from a P.P.L.O. negative plant or be hatched in an isolated incubator from eggs laid by presumably infected hens. These, however, must look perfectly healthy. In South Africa considerable success has so far attended the adoption of this last and admittedly risky method, in spite of the fact that the carefully culled breeding birds have not been treated with anti-biotics, and the chicks have not been raised to maturity in small units of 200 to 300. Naturally, a breeder should always try to eliminate the factor of chance by repopulating with chicks known to be clean, for there is ample evidence in the Union that both C.R.D. and infectious sinusitis are often spread through the egg.

The brooder house is left empty for a month before the clean chicks are put into it. From there they are moved to other houses and camps and eventually reach the flock houses or cages, all of which must stand empty for a month before being repopulated. Separate attendants must be used for clean and infected stock and there should also be separate feed and egg rooms. Once farmers see how well the young clean stock is doing they usually hasten the disposal of the remaining infected birds.

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Various Results of Testing

Many veterinarians and poultry breeders are now concerned about the legal and moral issues involved. As a result of a judgment handed down in the Supreme Court in Bloemfontein, in a dispute over the significance of a Salmonella pullorum infection on a farm, it seems that a breeder exposes himself to a claim for damages if he sells hatching eggs or live birds, when knowing full well that C.R.D. exists on his premises. Unless he discloses the true state of affairs on his farm to the potential customer beforehand, he apparently cannot fall back on any general statement, written or otherwise, that he accepts no responsibility for any serious infection present in the chicks on the day of sale.

From the moral point of view, the position is more difficult. The demand for clean chicks far exceeds the supply, and provided that a breeder is actively engaged on cleaning up his plant, and greatly reduces the risk of selling infected chicks by culling his flocks drastically, nothing more can reasonably be expected of him. It is the breeder, who knows his birds are infected and still takes no steps to eradicate the disease who is a menace to the industry. Experience has shown that he cannot even plead that the cost of repopulating his farm is prohibitive.

Progress has been very encouraging. Twenty-five clean farms have been established or found in the Union, and it is known that seven of the farms were repopulated with chicks from infected birds. As sixteen of the farms cleaned up were previously badly diseased, the results have been most gratifying to all concerned. There are only two negative turkey flocks. At present another thirty to forty poultry farms are in the process of being rid of P.P.L.O. and interest in the test is mounting rapidly.

Many wish to know how long a farm will remain clean. There is irrefutable evidence that one farm has been free of P.P.L.O. infection since it was established with day-old chicks seven years ago, and there is no reason why it should not remain so. Furthermore, it has been reported that when a flock is raised free of pathogenic P.P.L.O., it can be assumed that non- pathogenic strains will also be absent.

The occurrence of what seems to be a new respiratory condition on no less than six of the clean farms must be reported. It is seen mainly in chickens six to eight weeks old, which make bubbling noises as they breathe. The morbidity does not exceed one in about 500, and the victims lose no weight and recover in two to three weeks. There are no macroscopic lesions, except occasionally in the air sacs. The birds continue to react negatively to the P.P.L.O. test, and so far attempts to transmit the condition to clean poults and other chickens have failed. One of these very minor outbreaks may be experienced every few months on a farm. Twice a fungus has been demonstrated in an air sac, and further

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study may reveal that these organisms are always responsible. As a precautionary measure, " bubblers " should always be sent to the laboratory for examination.

In conclusion, it appears that if no unforeseen difficulties arise, there is every prospect of virtually ridding the Union of avian respiratory diseases within the next two years. The value to the industry of such an achievement will be immense.

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THE TECHNIQUE OF TOTAL MASTECTOMY IN THE COW

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The operation of total mastectomy in the cow is one which is attended by serious risks if attempted in a haphazard manner. Reports of massive operative haemorrhage and post-operative deaths are common.

It is believed that the technique as presented here carries a minimum of surgical risk.

Indications:

(1) Extreme mammary fibrosis causing udder to hang close to the ground and to impede walking.
(2) Pendulous udder due to weak suspensory ligaments leading to a similar disability.
(3) Chronic progressive mastitis.
(4) Malignant neoplasia.
(5) Preservation of the cow for breeding.

Pre-operative Preparation

The abdominal skin and udder are shaved and food and water withheld 36 hours before operation.

Anaesthesia:

As the operation can take some hours, either nerve block or epidural anaesthesia may be found to wear off too soon. The former is difficult to apply as the udder is supplied by *N. inguinalis* and the skin by *LI, LII* and *LIII*. The visceral vascular dilatation which accompanies spinal anaesthesia may also predispose to shock.

The anaesthetic found satisfactory was intravenous chloral hydrate (4.4 Gm/50Kg) in 10% solution reinforced with Sagatal (M & B). If skilled help is not available the cannula through which the chloral hydrate solution is injected is left *in situ* and connected with an intravenous drip apparatus to dispense 25 to 50% dextrose very slowly. Sagatal, if suitably diluted and held in a graduated bottle connected with the intravenous drip apparatus with a Y tube, can be held in readiness clamped off and an assistant can allow some Sagatal to infuse into the jugular on the surgeon's instructions.

Analeptics should also be accessible at a moment's notice and can be introduced into the bloodstream via the dispensing tube.

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Surgical Anatomy:

The surgical anatomy, the description of which is inadequate in most anatomy books, will be reviewed.

Blood is supplied by *A. pudenda externa,* which emerges from the inguinal canal, dorso-cranially from the *L. inguinalis superficialis.* It varies in diameter in the mature cow from less than 0.5 cm. to 2 cm. *A. perinea* is often absent, but may be single or multiple in the region of the lymph node.

The base of the udder is encircled by a vein giving rise to the *V. abdomina subcutanea* (milk vein) and *V. perinea* running dorsally along the perineum.

The udder is suspended laterally by suspensory ligaments attached to the prepubic and subpubic tendons and inserted ventrally into the median ligaments. These divide the udder into distinct halves making hemimastectomy possible, if indicated. The quarters on the same side, although they do not communicate, are so intimately joined as to defy surgical cleavage.

The operation:

The cow should lie on her side with the lumbar region rotated so as to expose the udder region. The upper hind leg is secured by a Degive's gag and pulled forward and upward.

After suitable draping, an incision is made from the perineum, just dorsal to the *L. inguinalis superficialis* in a wide ventral curve to a point just dorsal to the hind teat and sometimes just cranial to it. All bleeding points are clamped and ligated. The incision is deepened through the fascia. Care is taken to expose and resect the lymph node and to ligate the *V. perinea* and *A. perinea* (if present). A line of cleavage is found against the lateral suspensory ligament and the skin-fascia flap is resected and reflected dorsally, eventually to expose the *A.* and *V. pudenda externa.* This necessitates incision of the suspensory ligament where it joins the abdomen. The bloodvessels are doubly ligated dorsally and singly ventrally and severed between ligatures. This achieves the first and vital objective — interruption of the blood supply on one side.

The skin incision is now continued in a curve to immediately cranial to the udder on the linea alba. The *circulus venosus* is doubly ligated and severed between ligatures, as is the *V. abdomina subcutanea.* This is important as severe retrograde haemorrhage can take place.

This concludes the amputation of one half. The other half is removed similarly after the cow has been turned on to the opposite side.

By suturing the subcutaneous fascia potential space is minimized — sometimes eliminated — and the skin edges are brought together. By stitching loosely they are tailored first and then sutured with strong silk or braided nylon interrupted sutures.

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Sometimes the fascia is not sufficiently loose to allow suturing as described. The skin is then anchored to the subcutis at various places.

The cow can be expected to start feeding soon after the anaesthetic has worn off.

Post-operative treatment is according to general principles.

Discussion:

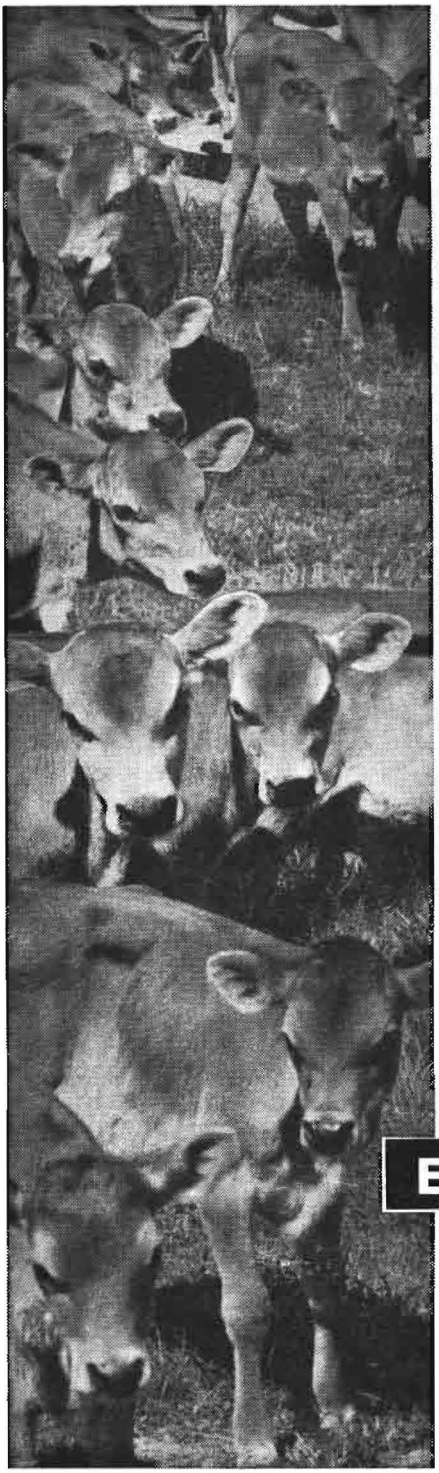
Interruption of the blood supply at as early a stage as possible is imperative. Dangerous haemorrhage and shock are virtually eliminated. The total amount of blood lost will certainly be much less than 500 ml.

As there are tremendous variations in the size of the udder, each case has to be treated on its merits. The weight of the udder varies between a few Kg. to 25 Kg.

The whole pattern of blood supply may be vastly altered by new growths, with the result that unexpected problems might have to be faced.

A cow in calf has been operated upon without abortion taking place as a sequel.

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THE CONTROL OF INTERNAL PARASITES OF SHEEP WITH NEGUVON AND ASUNTOL

A Preliminary Report

S. STAMPA

Introduction:

Chemically Neguvon is Dimethyl-hydroxy-trichloroethylphosphonate and Asuntol is the O, O-Diethylphosphoric ester of 3-chloro-4-methyl-7-oxycoumarine.

Riek and Keith (1958) tested a number of phosphoric ester insecticides for their effectiveness against the common internal parasites of cattle. They found that Neguvon (Bayer L 13/59), reduced the faecal egg counts of *Haemonchus placei, Ostertagia ostertagi, Bunostomum phlebotomum* and *Oesophagostomum radiatum* satisfactorily and of *Cooperia* and *Trichostrongylus spp.* to a lesser extent. In a test with a limited number of animals they found Asuntol (Bayer 21/199) controlled *Cooperia* very well in cattle. Gordon (1958) tested the same materials in sheep and reported a high degree of effectiveness for Neguvon at somewhat high dosage rates against *Haemonchus contortus*, and, when the material was swallowed into the abomasum, against *Trichostrongylus colubriformis*. Neguvon gave variable results against *Oesophagostomum columbianum*. In a test with a few animals he found Asuntol very effective against *Trichostrongylus*, even when administered into the rumen. In a limited number of trials with cattle and sheep Herlich and Porter (1958) found Asuntol very effective against *Haemonchus, Cooperia, Ostertagia, Trichostrongylus, Trichuris* and *Strongyloides*.

McGregor et al (1954), and later others, found Neguvon given per os effective against the larvae of *Hypoderma ssp.* (warbles) migrating in the body tissues of cattle, indicating a systemic effect.

Materials and Methods:

In most trials Neguvon was used as a 10% solution prepared from the technical material or from an 80% soluble powder. In a few trials a 10% suspension, prepared from a 50% wettable powder, was used. Asuntol was used as a 1, 2 or 4% suspension prepared from a 30% wettable powder and in a few experiments as the wettable powder itself. Mixtures used contained Neguvon as a 9.1 or 10% solution and Asuntol as a 0.9 or 1.0% suspension.

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The infestation of sheep before and after dosing was estimated by faecal egg counts using a slight modification of the method described by Laurence et al (1951) and the Gordon-Whitlock method. The same counting method was used in each experiment before and after dosing. The eggs of *Strongyloides, Bunostomum, Marshallagia* and *Nematodirus* were counted individually, but those of *Haemonchus, Ostertagia, Trichostrongylus* and *Oesophagostomum* were counted together, without trying to differentiate between species. The number of eggs to be accounted for by each of these four species was calculated on a percentage basis according to larval identifications from faecal cultures. Frequent post mortem examinations were held in order to check egg counting and larval identification techniques.

Naturally infested sheep usually carrying mixed infestations were used. For one experiment, sheep with a virtually pure *Oesophagostomum* infestation and for another expperiment sheep with virtually pure *Trichostrongylus* and *Nematodirus* infestations were available. The sheep were kept in kraals and fed on dry lucerne in some experiments. In others they were kept on naturally infested pastures. Examinations were done immediately before and 12 to 20 days after dosing. In the kraal experiments, two egg counts at two-day intervals were made before and after dosing, the averages being used to determine the efficacy. In the field experiments only one-egg count was made before and after dosing.

Neguvon reduced the egg counts of *Haemonchus* and *Nematodirus* very satisfactorily when a predose of copper sulphate was given. Its action against *Nematodirus* was much less effective without the predose. Asuntol was efficient against all parasites tested irrespective of predosing. The mixture comprising approximately half the dose of Neguvon and one third of that of Asuntol was equally effective as Asuntol alone except in the case of *Strongyloides*.

Experiment 2.

In a second experiment a mixture of Neguvon and Asuntol in the ratio of 10 to 1 after predosing with copper sulphate, was used throughout. The sheep were weighed individually and grouped so that the dose in mg/Kg was within the ranges indicated in the table for each group. (See Table 2).

Good efficacy against all the species tested for was obtained at a dosage rate of 50—60 mg/Kg or approximately 1.75 gm for an adult sheep. The most resistant parasite was *Strongyloides* which is well known to be very difficult to control. Nevertheless, a reduction of over 60% in the egg count was achieved at this rate of dosing.

Further Observations:

Live adult *Oesophagostomum* and *Trichuris* were found in the droppings from 5 to 36 hours after dosing. It is therefore

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TABLE 1

Experiment 1.

A. With Copper Sulphate Predosing

Dose used	Neguvon 3—4 gm		Asuntol 0.6 gm		Neguvon 2 gm Asuntol 0.2 gm	
	No. sheep	Result	No. sheep	Result	No. sheep	Result
Haemonchus	2	+++	0	—	1	+++
Ostertagia	1	+	1	+++	2	+++
Trichostrong.	2	0	2	++	3	++
Strongyloides	4	+	4	+++	3	+
Nematodirus	9	+++	5	+	3	+++
Oesoph.	3*	+++	0	—	2	+

Experiment 2.

B. Without Copper Sulphate Predosing

Haemonchus	0	—	1	+++	0	—
Ostertagia	3	0	4	++	1	+++
Trichostrong.	2	+	4	+	2	++
Strongyloides	4	0	6	+++	3	0
Nematodirus	5	+	2	+++	4	+
Oesoph.	0	—	4*	+++	1*	++
Marshallagia	0	—	6	++	5	++

0 = 0— 59% reduction in worm egg count after dosing.
+ = 60— 79% " " " " "
++ = 80— 89% " " " " "
+++ = 90—100% " " " " "

* Very slight infection before dosing. Lambs received ½ to ⅓ standard dose.

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TABLE 2

Dose (gm) / Dose (mg/Kg)	1.11 30—40		1.43 40—50		1.75 50—60		2.06 60—70		2.38 70—80	
	No. sh.	Res.	No. sh.	Res.	No. sh.	Res.	No. sh.	Res.	No. sh.	Res.
Haemonchus	1	+++	3	+++	5	+++	4	+++	3	+++
Ostertagia	1	+++	2	+++	3	+++	4	+++	4	+++
Marshallagia	0	—	0	—	2	+++	0	—	0	—
Trichostrong.	1	0	9	++	7	+++	9	+++	6	+++
Strongyloides	0	—	0	—	1	+	0	—	6	+++
Nematodirus	4	0	6	+++	7	+++	3	+++	3	+++
Oesoph.	0	—	1	0	3	++	3	+++	7	+++

(Symbols are as used in the previous table).

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probable that the stimulation of intestinal movement, characteristic of phosphoric esters through their inhibition of cholinesterase, is mainly responsible for the expulsion of these parasites of the large bowel. As sheep show considerable individual differences in the reduction of blood cholinesterase after receiving the same dose, varying efficacy against *Oesophagostomum* is to be expected. In one trial using six sheep, the reduction in blood cholinesterase, as determined by the colorimetric method and the reduction in the output of *Oesophagostomum* eggs were correlated. The dose used was 70 mg/kg of the Neguvon-Asuntol mixture. Two sheep showed only a slight reduction in cholinesterase and the egg output was reduced by 0 and 64% respectively. The remaining four showed a normal reaction and a 75% to 95% reduction in egg counts.

Six sheep infested with *Bunostomum trigonocephalum* were treated with 2.2 gm (60—80 mg/Kg) of the Neguvon-Asuntol mixture, after predosing with copper sulphate, three times at three-weekly intervals. The egg counts, per gm of faeces, before and after treatment, were as follows:

Sheep No.	1	2	3	4	5	6
Before Treatment	5	23	102	82	50	290
After Treatment	0	0	0	0	0	3

Tapeworms of the *Moniezia* species, including the fore-ends, were frequently found in the droppings of sheep 24 hours after being dosed the mixture. Scolices were not found. Three sheep previously infected were kept under observation for 6 weeks. No segments were found in the dropppings of two of them over that period while the third only showed segments three weeks after being dosed.

Larvae of *Oestrus ovis*, up to 6 mm in length were expelled dead from the nostrils of sheep which had received 55 mg/Kg of the Neguvon-Asuntol mixture. At a dosage rate of 88 mg/Kg dead larvae up to 15 mm in length were expeplled.

First and second instar larvae of the blowfly *Chrysomyia chloropyga* were found dead on sheep within 3 hours of dosing the mixture at the rate of 55 mg/Kg. At a 70 mg/Kg level third instar larvae were also killed. Larvae of *Chrysomyia albiceps* withstood even the higher dosing level.

That the Neguvon-Asuntol mixture may be of value in combating *Dictyocaulus filaria* infection is indicated by the following observation. Of 17 sheep all infected with *D. filaria*, as proved by faeces examination, 13 were dosed the mixture (40—55 mg/Kg) seven times at three-weekly intervals. The remaining four were dosed with different vermicides as used routinely by many farmers. All were kept on lungworm infested grazing throughout this period. After treatment no larvae could be found in the faeces of 11 of the 13 sheep treated with Neguvon-Asuntol. The remaining two and all four of the controls were still infested.

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Females of *Ixodes rubicundus* were not affected by doses of 3 gm Neguvon to the adult host sheep.

The finding that these drugs, when given orally, act on parasites not harboured in the digestive tract support the contention of a systemic effect made by McGregor *et al* (1954).

Toxicity — Neguvon alone:

In the Karroo 100 mg/Kg produced no obvious toxic symptoms in any of over 400 sheep dosed. Of a flock of 75 lambs dosed 2.5 gm. (= approximately 110 mg/Kg about 10 reacted but soon recovered. Of 6 sheep dosed 130 mg/Kg, 2 showed severe toxic symptoms. Sheep in the winter rainfall area of the Cape appeared to be more susceptible than those in the Karroo. Of 10 sheep dosed 90 mg/Kg, 7 showed mild and 2 severe toxic symptoms, while of 23 animals dosed 100 mg/Kg, 1 died and 9 others showed severe symptoms. The reason for this difference has not been found. The winter rainfall areas are known to be deficient in certain minerals and trace elements.

Asuntol alone:

The lowest dosage rate to cause death was 17.6 mg/Kg at which level 2 out of 400 dosed sheep succumbed. Of 81 dosed 20 mg/Kg, 4 died and of 6 dosed 27 mg/Kg one died.

Neguvon-Asuntol Mixture (10 *to* 1):

In the Karroo and Orange Free State, a total of 2,152 sheep were dosed at rates from 54 to 90 mg/Kg without producing any deaths or even obvious toxic symptoms. These included sheep on natural grazing as well as animals fed on green and dry lucerne and on prickly pear.

The results of higher doses are given below:

Dosing Rate mg/Kg	No. of sheep dosed	No showing Toxic effects	No. of Deaths
110	50	4	1
120	19	9	1
130	10	1	0
154	5	5	2

The dose found effective against most internal round worms (50—60 mg/Kg) would therefore appear to be well within the safety margin, at least for Karroo sheep.

Angora goats were found to be more susceptible than sheep. Of 72 rams dosed with the mixture at a rate of from 62 to 83 mg/Kg, 53 showed mild and 16 severe toxic symptoms but there were no deaths. Of 5 goats dosed 90 mg/Kg all showed severe symptoms and 3 died. Five sheep dosed at the same rate and at the same time showed no symptoms.

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Sheep in the winter rainfall area of the Cape were found to be also more susceptible to the Neguvon-Asuntol mixture than those in the Karroo. Of 10 sheep dosed 80 mg/Kg, 3 showed mild toxic symptoms while of 24 sheep given 100 mg/Kg, 12 showed toxic symptoms and one died.

Symptoms of Toxicity:

In mild cases there is listlessness, inappetence and frequent stretching. In more severe cases there is also salivation and the animals tend to lie down. Acute poisoning causes prostration with kicking movements and grinding of the teeth. Death occurs between 30 minutes to 4 hours after a fatal dose of Neguvon and from 4 to 72 hours after Asuntol. Atropine should be used as an antidote and heart stimulants with little or no vasoconstrictory effect (e.g. caffeine) are also of benefit.

As the parasympathomimetic effect of these drugs causes increased bronchial secretion they should not be used on animals suffering from bronchitis, pneumonia or pulmonary oedema. Severe liver derangement also renders animals more susceptible to these drugs. It has also been found that animals suffering from severe blowfly strike should only be dosed with extreme caution.

Summary:

1. Neguvon was found to be very effective against *Haemonchus* and *Nematodirus*, possibly also against *Oesophagostomum* as judged by the reduction of eggs voided after a single dose. Asuntol was very effective against *Haemonchus, Ostertagia, Trichostrongylus, Nematodirus, Oesophagostomum* and *Strongyloides*.
2. The effect of Neguvon was greatly enhanced by predosing with copper sulphate but that of Asuntol was not.
3. A mixture of 10 parts Neguvon to 1 part Asuntol when given after copper sulphate, gave similar results to Asuntol alone except that the action against *Strongyloides* was not as satisfactory.
4. Non-intestinal parasites such as *Oestrus ovis, Chrysomyia* larvae and *Dictyocaulus filaria* were noted to be affected by the dosing.
5. An effective dose of the mixture was found to be 50 to 60 mg/Kg body weight. In all, 2,152 sheep in the Karroo were dosed with from 50—90 mg/Kg without producing any visible clinical symptoms. A dose of 110 mg/Kg caused one death in 50 sheep. Higher doses were also tested.
6. Angora goats in the Karroo and sheep in the winter rainfall area were found to be more susceptible than Karroo sheep.

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CLINICAL COMMUNICATIONS

THE TREATMENT OF *FILAROIDES OSLERI* INFESTATION WITH DICTYCIDE

J. E. DORRINGTON
Bellville

In view of the excellent results shown by "*Dictycide*" (I.C.I.) against *Dictyocaulus filaria*, this drug was tried out on six Boxer dogs suffering from *Filaroides osleri*. The dogs all originated from the same kennels and their ages ranged from 4 months to 5 years. Three of the cases were diagnosed by bronchoscopy and the others by the presence of the classical persistent dry cough.

The dose given was 1 gm. per 10 lbs. body weight, repeated for 3 consecutive days where necessary. It was found that by using warm water to make up the solution just before use the pain and discomfort shown immediately after injection was entirely eliminated. No side effects were experienced.

In four cases which were treated at the onset of coughing, the cough disappeared and did not recur after only two doses. Another dog required a second course of treatment three weeks later.

The sixth case was a two-year-old bitch which was very emaciated and had been coughing continuously for the last six months. She was treated on five consecutive days and this was repeated three weeks later. There was marked improvement, the cough disappeared and the bitch returned to show condition. She required no further treatment for the next five months although bronchoscopy after the second course of treatment revealed many eggs in the tracheal debris. The bitch was finally destroyed because of epileptiform fits and post-mortem examination revealed the presence of some ten *F. osleri* nodules. These findings confirm those of Lauder, who reported gross lesions of *F. osleri* in clinically healthy dogs.

Although Dictycide did not produce a complete cure in this case, it would appear to be a valuable drug in relieving the distressing cough and loss of condition.

It is also of interest to note that Lauder has found *F. osleri* in Greyhounds and Poodles. His suggestion that lateral X-ray of the chest is a rapid method of diagnosis is confirmed, as the nodules are visible at the bifurcation of the trachea.

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TETANUS IN CALVES AS A SEQUEL TO ELASTRATION

S. J. VAN RENSBURG
Onderstepoort

Abstract: The death of 14 calves from tetanus following castration by " elastration " is recorded. It is concluded that this complication may assume significant proportions, particularly in warm tropical and sub-tropical climates.

On the 2nd August, 1958, I was requested by a farmer to investigate a fatal illness amongst his calves. The farm was situated some 25 miles south-west of Messina.

Afrikander calves were seen in all stages of typical tetanus. The first noticeable symptom was intermittent prolapse of the membrana nictitans — a constant feature in all the affcted calves. Owing to the early onset of trismus, repeated attempts to suckle were unsuccessful. General hyperaesthesia, clonic muscular spasms and a characteristic stiff gait with abducted legs soon followed. Staring eyes, immobility of ears, extension of head and neck with salivation, and ultimately prostration, were also prominent symptoms.

The owner had " castrated " the calves by means of " Elastration ", the first case of tetanus being noticed 13 days later. By this time, the rubber-ring ligature had caused a deep wound in the scrotum and was completely embedded, owing to considerable inflammatory swelling immediately above the site. The resultant anerobic state, associated with the presence of a fair amount of necrotic tissue due to the thickness of the rings, was ideal for the development of *Clostridium tetani*. The wounds of all the calves showing symptoms were moist and emitted an offensive odour, while those of the unaffected calves were dry and odourless.

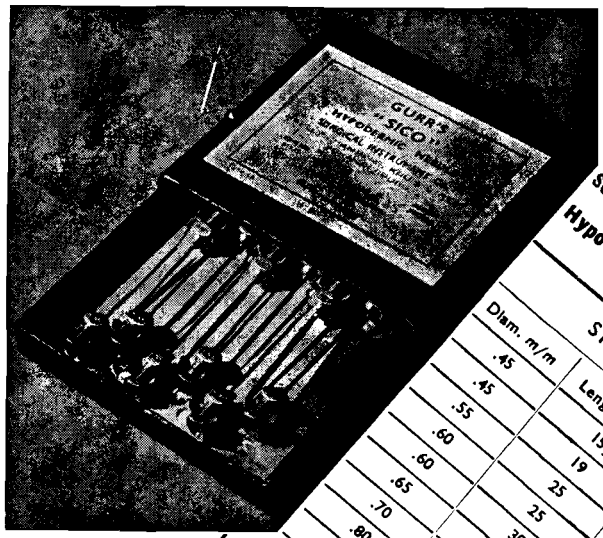
Treatment was applied to five calves, chiefly in the early stages of the disease. The wounds were thoroughly cleaned and wound powders used, while 1.5 million units penicillin and 2-4 mg per Kgm. Chlorpromazine hydrochloride intramuscularly daily were administered. No improvement was observed and all five died within 2—3 days, as did the untreated cases. The dry necrotic scrotums of all the calves were amputated immediately below the rubber-ring ligatures, after which no more cases developed.

Of a total of 42 calves elastrated, 14 (33.3%) succumbed to tetanus, all the cases starting between the 13th and 17th days after elastration. The calves were between 5 and 7 months old; the majority of deaths occurring amongst the older calves.

One six months' old calf, which was not affected by tetanus, had a severely enlarged scrotum which caused it great difficulty in walking. On amputation distal to the ring, one spermatic artery was found to be pulsating.

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RESPIRATORY ACARIASIS OF CANARIES AND GOULDIAN FINCHES

R. B. CUMMING
Onderstepoort

Respiratory acariasis of canaries, due to the mite, *Sternosoma tracheacolum*, was first described by Stephan, Kaschula and Canham in 1950.

This disease now appears to be widespread in the Union, and over the last two years infected birds have been received from George, Tarkastad, Johannesburg, Pretoria and Boksburg North. Further, from letters received, it would appear that the disease is present in practically all the large towns of the Union.

In all cases so far German roller canaries have been involved. Since birds of this breed are kept only for their song, any interference with their breathing and hence singing calls for immediate investigation. It is most unlikely that this breed is more susceptible to the infection than others.

Recently the author diagnosed the presence of this mite in Gouldian finches — *Peophila gouldiae* (Gould). These Gouldian finches were obtained from a successful breeder of German roller canaries. A mild infection of *Sternosoma trachaecolum* was observed in his canaries as well. There appeared to be no direct contact between the canaries and Gouldian finches and it is difficult to explain how the infection of the latter occurred. An excellent description of the disease in canaries can be found in the article by Stephan *et al.* In Gouldian finches it would appear that once established, the disease is far more rapidly fatal. In all cases, once respiratory distress was noticed the disease terminated fatally in about three weeks, unless the bird was treated.

This disease is readily diagnosed in infected birds at night. A typical clicking can be heard, accompanied by laboured breathing and frequent coughing and sneezing. Due to the respiratory distress a bird is unable to sleep in the normal attitude with its head tucked under a wing.

Treatment:

Stephan *et al.* suggested the use of B.H.C. preparations. The author has used Malathion, one of the newer acaricides, with promising results. Although belonging to the cholinesterase inhibitor group, Malathion is relatively non-toxic as far as warm blooded animals are concerned. In tables of pesticides in use, drawn up by Ward (1956), Malathion is ranked as one of the least toxic, and safer than B.H.C. preparations.

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The method used is as follows: The birds are placed in a small wooden cage with wire front, over which is spread a towel. Using an ordinary garden duster, 5% Malathion powder is pumped into the cage to create a fairly dense fog. To maintain a good fog it is found necessary to pump into the cage every 60 seconds. The birds are exposed to the Malathion fog for five minutes and the treatment is repeated once weekly for four weeks.

This treatment has proved very successful in both canaries and Gouldian finches. It is advisable to treat all susceptible birds on the premises at the same time, as many canaries appear to be healthy carriers of this mite.

Aerocystitis is a complicating factor in some outbreaks of respiratory acariasis. For this reason it appears advantageous to administer one of the broadspectrum antibiotics in the drinking water during treatment.

Thanks are due to Dr. R. du Toit for identifying the mite.

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THYMOMA IN A SIAMESE CAT

R. K. LOVEDAY
Onderstepoort

History:

The subject, a six-year-old neutered Siamese cat, was X-rayed in September, 1955, following the owner's report of persistent anorexia following a fall from an upstairs balcony some three weeks previously. The radiologist reported a crack fracture of the zygomatic arch on the right side with no separation or displacement, as also a large soft tissue swelling extending forward from the heart into the lower cervical region. " The appearances seem to be confined to the mediastinum, which is enlarged, and when the patient's history is considered, this would appear to be most likely due to an effusion or haemorrhage into the pre-tracheal or pre-vertebral space ".

Appetite returned after eight days and was maintained for some two months. Thereafter anorexia again supervened with a rapid deterioration in condition accompanied by a moist dermatitis which soon spread all over the body. Euthanasia was performed on 9.12.55.

Post-mortem and Pathology:

Extreme cachexia; large mediastinal tumour, approximately 4 ins. x 2 ins. x 1 ins., white in colour, of soft consistency and showing cystic and haemorrhagic areas on section. No metastases were seen.

The pathological report on this tumour reads as follows:

The tumour is well circumscribed.

The pattern varies in different areas:

(a) There are a number of cysts which have a wall of fibrous tissue. Some have no epithelial lining, others are lined by a single layer of flattened cells.

(b) There are a number of masses of eosinophilic material which are surrounded by epithelial cells.

(c) There are masses of spindle-shaped epithelial cells which tend to be arranged in whorls. Small masses of eosinophilic material are surrounded by these cells, which give the appearance of modified Hassal's corpuscles.

(d) There are areas where the growth has the pattern of a basal cell carcinoma in which the stroma is myxomatous, suggestive of adamantinoma.

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(e) There are areas where the tumour cells have a polygonal appearance, and are forming a reticular structure. Between the cells are masses of lymphocytes.
(f) There are areas which consist of sheets of lymphocytes.
(g) No mitoses have been seen.

Conclusion: The tumour has the histological appearance of a thymoma, i.e. carcinoma of thymus".

Literature:

Jackson (1936) describes no less than seven thymomas (three bovine, three ovine and one caprine) and remarks, "the tumours are encapsulated by the mediastinal pleura, but tend to invade the capsule and also their stroma; so that, in spite of their slow growth (mitotic index nil or very low), they are to be regarded as malignant and in their ultimate development are responsible for serious and fatal results". Other reports refer to thymal tumours in a goat (Bos [1946]), in a heifer (Popplow [1950]), and in a two-year-old Persian cat (Locke [1948]).

Cotchin (1951) mentions five tumours in the position of the thymus in dogs and three possible thymal tumours in cats. The same author (1952), reviewing a series of 226 neoplasms in cats, reports on four lymphosarcomas of the thymus and one thymoma in the six endocrine tumours encountered. Of the 184 cats in this series whose age was known, 68.5% were between six and twleve years old.

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A BRIEF NOTE ON MAMMARY SARCOMA IN A COW

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Department of Surgery
Faculty of Veterinary Science, Onderstepoort

SUMMARY

1. A case of sarcoma of the udder in the cow is described.
2. Resulting complications during operation are indicated.

Except for skin tumours, mammary neoplasmata are exceedingly rare in cows in South Africa.

A full-grown Jersey cow was sent to the Faculty hospital by a colleague, who suspected mammary neoplasia.

The cow was in good physical condition and non-lactating. A firm round swelling of the left half of the udder about 15 cm. in diameter could be felt, deep and near the dorsal attachment. Exploratory aseptic puncture drew a fair amount of blood. The supramammary lymphnodes (*Lnn. inguinales superficiales*) were not enlarged clinically. Neoplasia was suspected, but operation was delayed for 10 days for observation. The swelling grew to a diameter of 22 cm. Aseptic puncture again drew a large amount of blood. As the tumour was against the median suspensory ligament spread to the other half of the udder was feared. Total mastectomy was decided upon and performed under general anaesthesia, attempting the technique as described elsewhere in this Journal. The affected side was operated upon first.

It was found that even the smaller cutaneous and subcutaneous bloodvessels were grossly enlarged — necessitating the tying off of several hundred bleeding points. Due to tumour growth, the usual lines of cleavage were not present. When, after considerable difficulty, the *A. pudenda externa* was exposed, it was found to be less than 0.5 cm. in diameter. In fact, the tumour received blood from the large number of enlarged, normally insignificant subcutaneous arteries. Even though each bleeding point was dealt with as quickly as possible, the amount of blood lost gave rise to some anxiety.

The affected half of the udder took nearly 4 hours to separate.

In contrast, the other half with, paradoxically, an *A. pudenda externa* of 1.0 cm. in diameter, was removed in somewhat less than half an hour.

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There was some slight clinical anaemia as a result of operation. Blood transfusion was not found to be essential and the case made a very good recovery.

The histopathological report made by Dr. D. J. Smit, Department of Pathology of this Faculty, was as follows:

" The histopathological picture showed a marked proliferation of spindle-shaped cells with a limited fibril production. Areas of focal necrosis occurred in the centre of the growth where the blood supply was insufficient. The epithelial cells showed a certain amount of hyperkeratosis. One must consider two possibilities in the differential diagnosis. The spindle-shaped cells may be of epithelial origin in which case the diagnosis would be carcinoma. Neoplastic epithelial cells may assume a spindle shape leading to erroneous diagnosis of sarcoma. In the diagnosis of fibro-plastic sarcoma the final criterion remains the presence of fibrils in between the tumour cells and not confined to the stroma. The presence of a limited fibril network in this tumour rules out the possible diagnosis of a carcinoma. This tumour is of special interest in that sarcoma of the bovine udder is of great rarity ".

Acknowledgements

To the Director of Veterinary Services, the author wishes to record his thanks for permission to publish this note in this journal — also to Dr. D. H. G. Irwin, Senior Lecturer, and Mr. D. de Vos, both of this Department, for assistance.

Finally, the author is indebted to Dr. W. J. B. de Villiers, of Klerksdorp, for referring this case to the Faculty Hospital.

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SURGICAL CORRECTION OF PARTIAL PROLAPSE OF THE UTERUS IN THE PREGNANT COW

P. J. KRIEL

The condition where the portio vaginalis uteri protrudes through the lips of the vulva in the pregnant cow, is sometimes encountered. The condition is usually seen in cows that are 7 months or more pregnant.

A few years ago the author was called out to a case like this. The portion protruding was soiled, inflamed and swollen. It was then thoroughly cleaned and pushed back. A pessary was put into the vagina and the vulva sutured. The cow was doing well for a few weeks until the stitches, which were festering, tore right through the lips of the vulva and the condition recurred. New stitches were applied. Not long after that the cow calved and was apparently normal.

At a later date, however, I was called out once more. The cow was pregnant again and the condition had returned. The folds around the os uteri were asymmetrical, very inflamed and had a tumour-like appearance. It was then decided to remove the inflamed portions.

Under epidural anaesthesia, the cervix was drawn out as far as possible. A ligature was put round the cervix as high up as could be reached. The inflamed folds were then cut off and the mucous membrane of the cervical canal was sutured to that of the portio vaginalis. The ligature was removed and the cervix pushed back into the vagina. The cow was seen again the next day. There was no tendency for the prolapse to recur. She calved normally and had no trouble in conceiving again. She has been normal ever since.

Shortly after this a second cow has been treated in the same way. She is in the owner's stable up to this day and has gone through several gestation periods without showing any signs of abnormality.

About seven cows have been treated in this way. All of them were normal directly after the operation but most of them were not seen again afterwards.

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PLATE 1

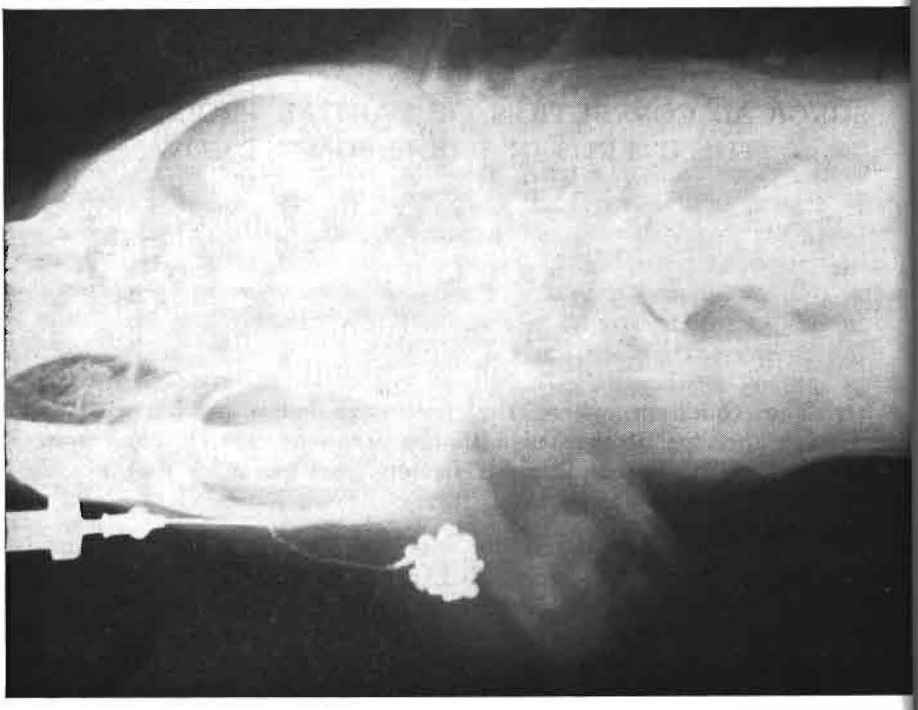
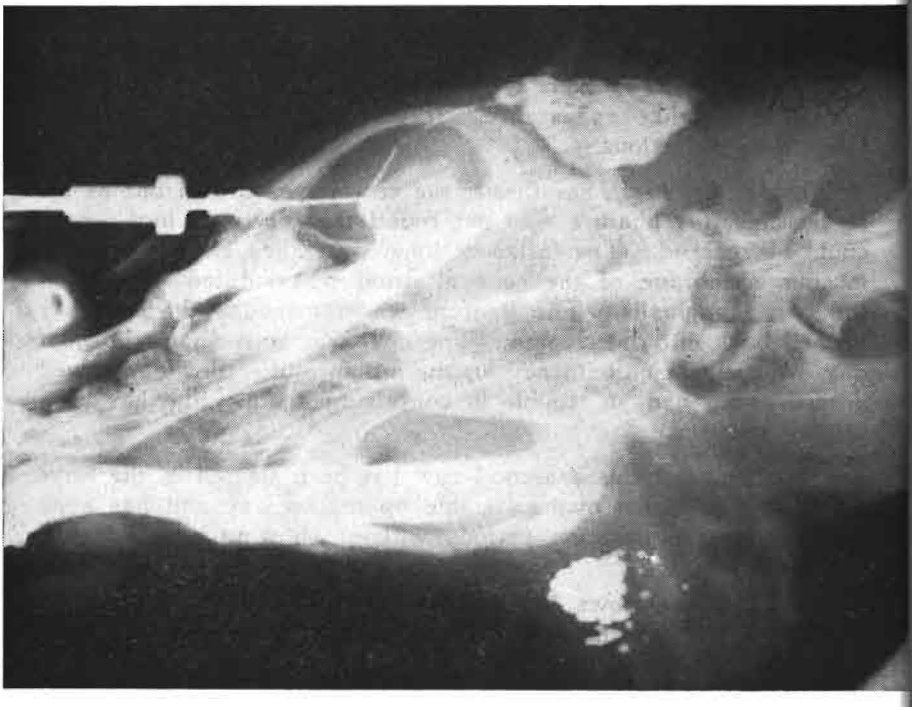


PLATE 2



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OCCLUSION OF THE PAROTID SALIVARY DUCT IN A DOG

D. H. G. IRWIN
and
D. DE VOS
Onderstepoort

The subject was a young adult Boer-bull bitch.

The history was that a swelling had developed on the lateral aspect of the right jaw during the previous 8—10 days.

Examination revealed a mildly tender, fluctuating swelling with a diameter of four cm. on the lateral surface of the vertical ramus of the right mandible. Aseptic needle puncture yielded a colourless slightly tacky fluid, assumed to be saliva. The bevel point of a number 20 B.W.G. hypodermic needle was taken down on an oil-stone. After anaesthetizing the animal the tip of this needle was introduced into the duct via the oral papilla. One ml. 40% Lipiodol was injected into the duct and a sialogram taken (15 ma, 55 KV, 0.25 sec. at 30 ins., using Kodak Ultra speed intensifying screens and Supervedo film). (See Plate 1). Fifteen minutes later a second film, with the same amount of contrast medium injected into the left duct was made (same exposure) as a control (see Plate 2). Plate 1 shows the arrest of the Lipiodol in the right duct. The soft tissue swelling is also clear in this picture. Plate 2 shows the normal parotid sialograph of the left gland and the occlusion on the right side.

Treatment: Using standard surgical technique, the skin over the area was reflected, and the duct demonstrated. The occluding body was a mass of young connective tissue but no foreign body or other aetiological factor was discernible. Owing to the extremely delicate nature of the duct this could not be repaired, and it was decided to create a fistula to the skin, and attempt to lead this to the buccal cavity at a later stage.

Discussion: Possible surgical treatments include:

(i) Extirpation of the parotid gland. This procedure is extremely delicate and fraught with the danger of dividing the internal or external carotid arteries, the internal jugular vein, or branches of the 7th, 9th, 10th, 11th and

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12th nerves. Moreover, one or more of these structures may be embarrassed by contracting cicatrix during healing.

(ii) Destruction of the gland by injection of an irritant. The coincident inflammation and cicatricial contraction may involve the adjacent vessels and nerves.

(iii) Creating a fistula directly to the buccal cavity. This might be possible by using a 1.5 mm. or larger diameter polythene tube. Any tube smaller than this and almost certainly one of a sufficiently small diameter to be threaded through the intact part of the duct, would become blocked.

(iv) The method employed, though crude, has been satisfactory, in as much as the swelling has not returned and the fistula closed spontaneously. Possibly the secreting cells of the gland were destroyed with the back pressure before the surgical interference.

„DICTYCIDE" AS MOONTLIKE BEHANDELING VIR *FILAROIDES OSLERI*

P. H. LE ROUX

Uitstaande sukses is behaal met die middel in 'n geval van *F. osleri* in 'n „Irish Terrier" teef. 'n Kursus van *„Anthiomaline"* het na drie weke geen resultaat gelewer nie.

„Dictycide" is toegedien teen 'n dosismaat van 1 k.s. per 35 pond. Geen skadelike effekte is opgemerk nie, behalwe 'n swaar hoesbui na vier uur. Na drie dae was daar 'n merkbare beterskap en 'n week later was die dier klinies normaal. 'n Verdere toediening van *Dictycide* is gemaak. Sputum is nie weer ondersoek nie.

Aangesien die middel so veilig geblyk het, is die skrywer van mening dat verdere ondersoek i.v.m. die middel die moeite werd sal wees.

Dank gaan aan mnre. I.C.I. vir verskaffing van die middel vir ondersoek.

The writer reports the successful treatment of *Filaroides osleri* infection in a dog with „Dictycide" (I.C.I.) at a dosage rate of 1.0 c.c. per 35 lbs., and suggests that further investigation is warranted.

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APOMORPHINE AS EMETIC IN DOG — CONJUNCTIVAL ROUTE

C. J. MULLER
Onderstepoort

Apomorphine is an alkaloid obtained synthetically from morphine. The central depressant action of morphine is decreased, but not eliminated, while the emetic action is increased in apomorphine.

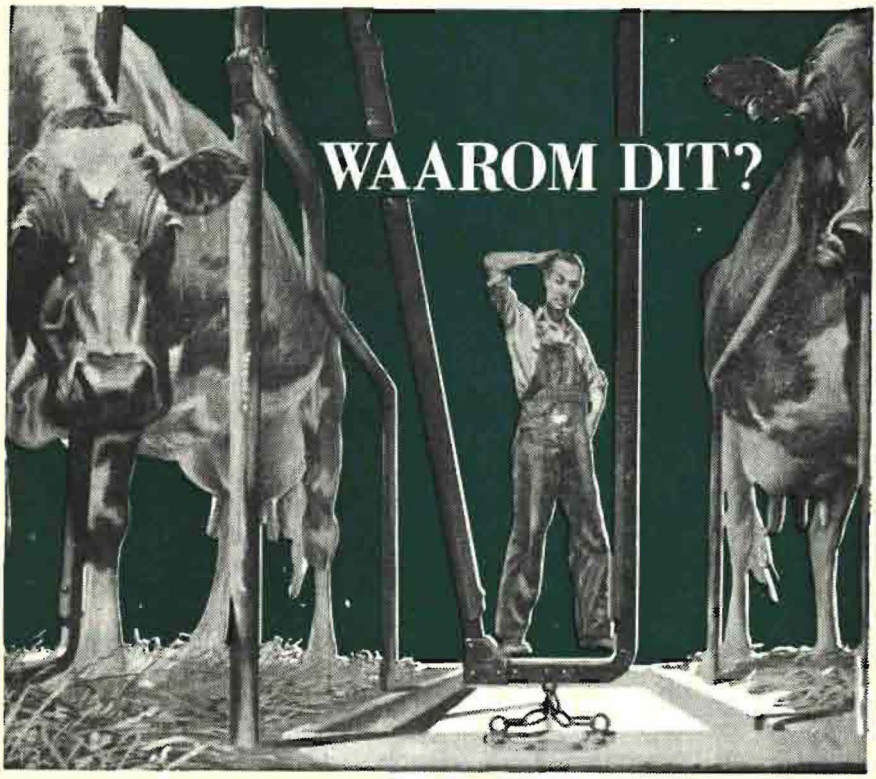
Apomorphine can be administered by injection or by dropping the required dosage, i.e. 1/40—1/20 grain (in tablet form) into the conjunctival sac. The drug is absorbed through the conjunctival membrane and acts directly on the emetic centre, producing vomition within 3—8 minutes. The advantages of the conjunctival route are obvious — eliminating preparation of a site for injection and no sterile equipment is required. The drug produces a slight irritation of the conjunctival membrane which shows up as increased lachrymation, but it disappears after one hour.

It is not advisable to use apomorphine on dogs that have been sedated or anaesthetized because under these circumstances the emetic centre is not at all or only slightly responsive to stimulation by apomorphine, and in fact the drug then produces further depression of the C.N.S. without vomition.

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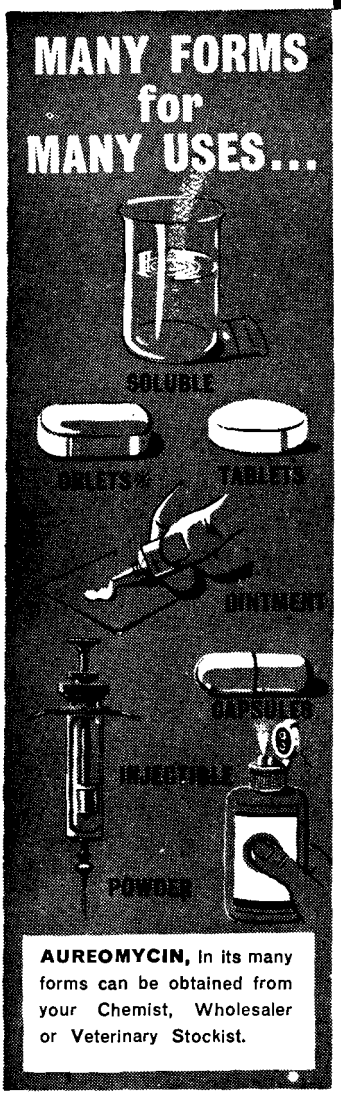
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BOOK REVIEWS

EVOLUTION OF THE VETERINARY ART. A Narrative Account to 1850 — by J. F. Smithcors, D.V.M., Ph.D. Illustrated by Mary Ellen Cross. Numerous figures and plates. Pp. xvii x 408. Veterinary Medicine Publishing Co., Kansas City, Missouri. Price not stated.

Smithcors presents here a scholarly work on veterinary history from prehistoric times until 1850. In a subject such as this it would be easy for the author to lose himself in a maze of detail — yet this book is both systematic and lucid and a pleasure to read.

It will be worthwhile to repeat some information given.

The principal veterinary record of ancient Egypt is the papyrus of Kahun (ca 1900 B.C.) discovered in 1889 A.D. It is suggested that the profession was taught in the temple of Memphis in 3000 B.C.

The Code of Hammurabi (ca 2250 B.C.) of Babylonia was a legal document regulating the practice of veterinary and human medicine.

In ancient India (1800—1200 B.C.) the Vedas were an ethical guide to the practice of human and animal medicine.

According to the Vedic interpretation human medicine in a sense originated from veterinary medicine. Sakhotra was a famous Indian veterinarian who flourished about 2350 B.C.

During the reign of King Asoka of India, hundreds of veterinary hospitals were established. Medical relief was ordered for all animals and birds (and also man), the only exception being criminals and trappers. The following translation from Sanskrit is still true: "The fool who does not know the different parts of a horse's body in detail, dos not know the signs, bad or good of horses, nor the treatment of their diseases." Indian veterinary medicine included all species.

In China veterinary treatises were written 2500 B.C. on disease of horses and buffalo.

In A.D. 598 a Korean veterinary priest taught the profession to a member of the Imperial Japanese Court, who in turn established veterinary schools in Korea.

Early Greek writings (like those of Aristotle) referred to veterinary medicine. This author for instance, described oophorectomy of sows. Greek veterinary writings were not very extensive as there were no epizootics affecting the country during those times.

Of the numerous interesting references to the veterinary medicine of Rome let us refer to one or two only. The Georgics of Virgil (30 B.C.) contained impoprtant veterinary writings. An erudite veterinarian of the period was Columella who wrote about obstetrics and sterility of ruminants and many other subjects.

A veterinary classic and one of the prized literary works of all time, the Hippiatrika, dates from the Byzantine. It represented works of 156 veterinarians, brought together in the 10th century A.D. by order of Constantine Porphygenitus.

With the Arab invasion the Hippiatrika was translated into Arabian and the Court Veterinarian to the Sultan of Egypt Abou Bekr wrote a 1,000 page treatise.

During the Renaissance the profession was one of the last to be resurrected, although it presented highlights then and later. Carlo Ruini, a Senator of Bologne, wrote "Anatomie de Cavallo," a treatise on the anatomy of the horse in no way inferior to that of the famous human anatomist, Vesalius, of 50 years earlier. Michael Harward, a scholarly Irish veterinarian of the 17th century,

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was a pioneer in intestinal surgery. His technique was far advanced beyond that of any veterinary or human surgeon of his time.

In this book by Smithcors of which the above represent only odd extracts, the thread of history can be followed up to a century ago. He has ranged wide for his material.

The reviewer, however, wishes to suggest that interesting additional information might be found in publications from central European countries. The book might perhaps also have given a little more of the early Spanish veterinarians like Salamiellas, who is very briefly referred to. This is, however, unimportant criticism compared with the general excellence of the book. It is hoped that it will find a place on the shelf of every veterinarian who esteems his profession.

C.F.B.H.

VETERINARY ANAESTHESIA by Professor J. G. Wright. Pp. xiii x 317. 4th Ed., 1957. Bailliere, Tindall & Cox, London. Price: 30s.

This book has won for itself a very wide acceptance and its value is well established. This fourth edition has been augmented to bring it in line with recent developments in Anaesthesiology.

Wright states that the position of chlorpromazine as an anaesthetic potentiator or tranquillizer is not preperly assessed. He refers to the intravenous use in horses. The reviewer would hesitate to employ this drug in such a manner in the horse as there may be unfortunate side reactions bordering on mania. There is much less risk of this when the drug is used subcutaneously or intramuscularly. In cattle sudden death may occur with intravenous use as reported by a colleague.

Anavenol K, especially if used for long operations, may cause fatal pulmonary oedema in ruminants. This fact could be included in the book with advantage.

Resuscitative measures during anaesthetic collapse could perhaps have been elaborated to a greater extent, as well as cardiac massage and intracardiac injections through a thoracotomy opening in small animals.

Finally, a chapter on physiology of respiration and circulation would not be out of place. This is knowledge which has a very intimate relation with anaesthesiology.

The above suggestions do not in any way indicate that the book is anything but a valuable addition to the library of the clinician. C.F.B.H.

VETERINARY OPHTHALMOLOGY, by R. H. Smythe, M.R.C.V.S. Examiner in Surgery to the Royal College of Veterinary Surgeons. Numerous figures and 30 plates. Pp vi x 379. 2nd Ed., 1958. Baillliere, Tindall & Cox, London. Price: 40s.

The first edition was reviewed in this Journal (Dec., 1956). The fact that a second edition was called for in two years testifies to the fact that this book filled a keenly felt need. This need was created by the practice of making this interesting subject a small and often obscure part of a work on surgery, with the result that the material suffered from incompleteness and other deficiencies.

To those unfamiliar with this work a brief description of contents would be desirable. The first 88 pages are given to a description of anatomy — a detailed, clear exposition. A description of animal vision occupies twenty pages and the optical system and accommodation of animals more than thirty pages. These lengthy descriptions, before clinical ophthalmology is considered, are amply justified by the interesting and important information supplied.

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The clinical ophthalmology occupies more than two hundred pages. The subject is dealt with systematically. It is inevitable that clinicians will disagree with each other on some points. The reviewer feel that the technique described for the removal of corneal dermoid is open to criticism. Perhaps the use of cortisone and allied compounds in ophthalmology could have been given in greater detail.

Smythe refers to infectious keratitis of cattle and sheep as it occurs in India, China, Canada, U.S.A., etc., and claimed to be caused by Moraxella (H) bovis. There is no reference to the very prevalent condition in South Africa which is clinically identical with the above, but which is caused, we believe, by a Rickettsia. These remarks, however, do not alter the fact that the book under review is the best work on veterinary ophthalmology with which the reviewer is familiar. C.F.B.H.

NEWSOM'S "SHEEP DISEASES." Second Edition. By Hadleigh Marsh.

The Williams and Wilkins Company, Baltimore, 1958.

In reading through this book one is impressed by the completeness of the information on sheep diseases.

Although it has been written primarily with the conditions affecting sheep in the United States in mind, it provides useful information on certain diseases found in other parts of the world.

In my opiinon Newsom's "Sheep Diseases" will be a most useful addition to the bookshelf of the private practitioner who has no time to search for the information which is systematically presented in this book.

One would, however, like to see a more modern approach to the treatment of conditions affecting the fore-stomachs. Tartar emetic is prescribed as a ruminal stimulant, while no mention is made of prostigmine, the dosing of acetic and propionic acid, the feeding of readily fermentable carbohydrates, e.g. molasses, and the dosing of ruminal contents of healthy sheep. Intussusception, which is fairly common among sheep, is not described.

The book is printed on high quality paper, attractively bound, and is remarkably free from typographical errors. B.C.J.

ANNUAL ROYAL COLLEGE OF VETERINARY SURGEONS' DAY

We are informed that the above celebration will take place in London on Tuesday the 2nd June, 1959.

The day commences with a full Choral Service and Commemoration of Benefactors in the Queen's Chapel of the Savoy followed by a luncheon in the Savoy Hotel. The Annual General Meeting of the Veterinary profession in the United Kingdom with presentation of awards, is to be held in the afternoon. There will also be an exhibition relating to the foundation of the Royal Veterinary College of London in 1791.

The President, Professor T. J. Bosworth, has indicated that all visiting veterinarians and their wives will be welcome to any or all of the above celebrations if space permits. The price for the luncheon will be £3 per person.

Early application to attend should be made to: The Registrar, The Royal College of Veterinary Surgeons, 9 and 10, Red Lion Square, London, W.C. 1.

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Diagnose
Diagnosis } DEMODEX

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Treatment } DEMODEL
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AGRICURA LABORATORIA BEPERK/LIMITED
SILVERTON

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OBITUARIES

IAN MOWAT

Dr. Mowat, after qualifying in 1942, enlisted in the Union Defence Forces in the Hygiene Section and was shortly afterwards commissioned in the South African Veterinary Corps. After the war, he was appointed as an Assistant Veterinarian in the Municipal Service in Johannesburg, in July, 1946. In 1948, he was appointed as a Senior Veterinary Officer and placed in charge of all the clinical work associated with the City Council of Johannesburg's transport animals, zoo animals and farming enterprises. Ian Mowat loved open air life, and the appointment he held suited his temperament admirably. He avoided office and routine work, and was happiest in the "sick lines" or on the farms. He was an excellent judge of both cattle and horses and was a very active member of the Executive Committee of the Society for the Prevention of Cruelty to Animals.

Dr. Mowat was a "good mixer", and made innumerable friends, both in and outside his work. His premature death on the 23rd of November, 1958, came as a great shock to all who knew him, though his health had shown signs of deterioration in recent months. Dr. Mowat leaves a wife and three children. To them we extend the heartfelt sympathy of all his colleagues. M.C.R.

GEORGE WATT

Dr. George Watt, M.R.C.V.S., whose death, after a lingering illness, occurred on February 19th, 1959, graduated at the Glasgow Veterinary College in 1928. He came to Florida in 1929 and commenced practice where he remained until his death. George Watt was a keen bowler and frequently represented his Club in tournaments; he was also known as a very fine shot. He leaves a widow, Iris, and a married daughter; his brother, James S. Watt, is in S.W. Africa. To all of the family, the profession offers expressions of sympathy in their bereavement. J.G.B.

B. RUNCIMAN

We regret to learn that Dr. Ben Runciman died recently in Kenya. He graduated from the North Edinburgh Veterinary College in 1900. He was the son of a veterinarian and had two brothers in the profession. He came to South Africa during the Anglo Boer War. He then took up private practice in Johannesburg where he built up a successful connection in racing circles and held appointments with the various racing clubs on the Rand. He had an extensive knowledge of the thoroughbred horse. For a time he practised in Epsom, England, but later returned to Johannesburg where he remained until his retirement a few years ago.

A.C.K.

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COUNCIL MATTERS

The following resolutions have been adopted by Council and are published for the information of all members.

USE OF ANAESTHETICS IN SURGERY

This meeting resolves that it is unethical for a Veterinary Surgeon to castrate or spay any adult animal (i.e. any animal having reached sexual maturity) without the use of a suitable anaesthetic; likewise it is unethical to perform any major operation on such animal without adequate or suitable anaesthetic. — (Council Meeting of 24.11.1958).

ADVERTISEMENT OF PROFESSIONAL POSTS

Members are requested not to apply for posts unless they are advertised through the South African Veterinary Medical Association either in its journal or by circular.

Members are further requested to notify the Secretary of any professional posts being advertised *only* in the lay press. — (Council Meeting of 24.11.1958)

INTERNATIONAL CATALOGUE OF VETERINARY FILMS

The Permanent Committee for the International Veterinary Congresses hopes to make available, early in 1959, its definitive catalogue of veterinary films.

The price will be about 10s. per copy, and those interested are asked to communicate with Dr. R. A. Alexander, Director of Veterinary Services, Onderstepoort, indicating the number of copies they are likely to require. No money should be sent at this stage.

THE STUDENTS' LIBRARY ONDERSTEPOORT

Colleagues will be interested to learn that the Students' Library at the hostel has been taken over by the University of Pretoria, and is to be greatly enlarged and reorganised. It will be housed in the common room of the old hostel building.

The University has made available a very handsome sum of money for the purchase of technical books and periodicals, and will undertake the organisation and supervision of the administration of the library.

In accordance with the wishes expressed at the last Annual General Meeting of the Association, all books belonging to the Association, and all journals received in exchange for our own, will be presented to the University for use in the Students' Library. The journals will also be bound by the University.

There is no doubt that the presence of a good technical library in the hostel will prove a major factor in the intellectual development and education of our students.

The facilities of the library will also be available to members of the Association.

The recreational library will also be housed in the same room.

Members are asked to remember the students' library when looking through their bookshelves. Recreational reading of all types will be very welcome and especially books of general interest, e.g. travel, hobbies, etc. On the more veterinary side books of historical interest would make valuable additions to the library.

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WARNING NOTICE

The Council of the South African Veterinary Medical Association, supported by the Annual General Meeting of the Association held at Onderstepoort on the 28th of August, 1958, has decided that it is unethical for any registered veterinarian who is employed by any animal welfare society, co-operative society or any other employer who is not himself a registred veterinarian, to allow any fees collected in return for professional services rendered to pass to the employer.

In such cases the registered veterinarian is enabling an unregistered person (the employer) to render professional veterinary services through himself, i.e. he is "farming out" his professional status. According to legal advice it is immaterial whether payment is voluntary or not.

Warning is therefore hereby given that in future any registered veterinarian who permits such action shall be deemed to have "permitted himself to be exploited in a manner detrimental to the public or professional interest" and shall be liable to disciplinary action. It must be fully realized that the responsibility in this connection lies with the veterinarian not the employer.

WAARSKUWENDE KENNISGEWING

Die Raad van die Suid-Afrikaanse Veterinêr-Mediese Vereniging, ondersteun deur die Algemene Jaarvergadering van die Vereniging, gehou op 28 Augustus 1958, te Onderstepoort, het besluit dat dit oneties is vir enige geregistreerde veearts in diens van enige dierewelsynsvereniging, koöperatiewe vereniging of enige werkgewer wat nie self 'n geregistreerde veearts is nie, om toe te laat dat enige gelde ingesamel in ruil vir gelewerde professionele diens aan die werkgewer oorgedra word.

In sulke gevalle laat die geregistreerde veearts 'n ongeregistreerde persoon (die werkgewer) toe om deur hom (die veearts) professionele veeartsenykundige diens te lewer, m.a.w. hy „huur sy beroepstatus uit." Volgens wetsadvies is dit nie tersake of betaling ongevraag geskied al dan nie.

Alle veeartse word hiermee gewaarsku dat dit enigeen wat in die toekoms sodanige gebeure toelaat, toegereken sal word dat hy „homself toegelaat het om uitgebuit te word op 'n wyse wat in openbare of professionele belang skadelik is," en hom aan dissiplinêre stappe blootstel. Dit moet terdeë besef word dat verantwoordelikheid in hierdie opsig by die veearts lê en nie by die werkgewer nie.

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SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION

PROCEEDINGS OF THE 53RD ANNUAL CONGRESS HELD AT ONDERSTEPOORT ON THE 26TH, 27TH AND 28TH OF AUGUST,

The following members registered at the Meeting: Drs. A. J. Louw, P. J. Uys, W. J. Wheeler, J. Bisschop, B. Osrin, H. Graf, L. Stonier, N. Barrie, H. P. A. de Boom, R. Clark, G. L. Muller, R. B. Cumming, J. D. Coles, R. T. Bangay, M. Meeser, T. W. Naude, J. M. M. Brown, J. L. Dickson, H. Kleeberg, A. van der Heever, J. E. Robinson, P. S. Snyman, D. C. L. Wacher, R. du Toit, L. T. Edwards, G. Theiler, F. W. Langbridge, S. L. Snyders, J. Boswell, K. Schulz, A. M. Diesel, K. M. van Heerden, J. W. Groenewald, J. M. Erasmus, L. W. van der Heever, C. D. Meredith, Z. Kempster, G. J. H. Stevens, L. Stewart, C. M. Breytenbach, P. Bisschop, L. Blomfield, H. Dames, S. W. J. van Rensburg, A. B. la Grange, C. F. B. Hofmeyr, I. van Schalkwyk, M. de Lange, S. B. Friedman, R. C. Tustin, L. Abrams, J. D. Smit, T. Adelaar, D. Irwin, J. Huyser, P. Anderson, H. G. Wallace, P. Thorold, T. Smuts, W. Eschenburg, A. M. Day, K. E. Weiss, N. F. Viljoen, W. B. Allchurch, I. S. Canham, Prof. D. G. Steyn, W. P. van Aardt, N. T. van der Linde, Jac. Louw, R. K. Loveday, A. Tarr, J. R. Scheuder, H. P. Steyn, P. D. de Wet, A. D. Thomas, P J. Fourie, M. S. Reichert, W. H. Buhr, T. A. T. Louw, P. R. Mansveldt, J. W. Pols, J. Zwarenstein, G. C. van Drimmelen, M. Bergh, W. J. Ryksen, G. C. Dent, J. M. W. le Roux, E. M. Robinson, C. H. Flight, W. C. Viljoen, C. M. J. Meldal-Johnson, M. M. Greathead, D. le Roux, P. B. Botha, R. J. Thomas, W. D. Malherbe, A. A. Albertyn, B. J. Brummer, K. van der Walt, J. A. de Kock, D. E. Truter, B. C Jansen, E. J. Pullinger, M. C. Robinson, D. L. McWhirter, P. J. Schutte, I. S. McFarlane, P. N. Collier, J. J. Oosthuizen, W. du T. Malan, J. W. van Niekerk, S. Solomon, G. E. Thompson, W. E. J. Warnes, A. M. Lubbe, P. H. le Roux, C. L. Craig, A. J. D. Williamson, D. Daly, G. F. J. van Rensburg, H. J. J. Terblanche, C. J. Muller, P. H. Brown, J. J. Hamman, M. Hearn, M. Keep, R. Alexander, W. Liebenguth, F. van der Riet, J. G. van der Wath, J. F. W. Grosskopf, M. W. Henning, P. G. Howell, D. J. Thornton, P. J. Meara, P. L. Louw, A. C. Kirkpatrick, R. K. Reinecke, O. T. de Villiers, J. W. A. Brookes, H. S. Purchase, J. Grobler, A. R. J. McGregor, S A. R. Stephan, F. J. Veldman, L. A. de Jager, J. H. Mason, H. Palmhert, L. L. Hansmeyer, J. van Niekerk, G. D. Sutton, J. S. van Heerden.

Apologies for absence were received from: The Director of Veterinary Services, Mocambique, The Director of Veterinary Services, Northern Rhodesia, The Director of Veteinary Services, Kenya, The President C.S.I.R., Dr. K. Unsworth, Drs. du Pre le Roux, Abreu, de la Harpe, Thorogood, A. J. du Plessis, J. E. Dorrington, J. A. Thorburn, R. S. Garraway, G. Martinaglia, J. Frean, L. G. Steel and J. G. Keppel.

TUESDAY, AUGUST 26th:

At 9 a.m. the President, Dr. P. S. Snyman called upon the Hon. Secretary to read the notice convening the meeting. The printed program, giving details of the Congress had been forwarded to all members.

The following motion, proposed by Dr. Graf was unanimously adopted, and the meeting stood for a few moments as a mark of sympathy: „Hierdie samekoms van die Suid-Afrikaanse Veterinêr-Mediese Vereniging treur oor die afsterwe van ons Eerste Minister, Sy Edele mnr. J. G. Strijdom en dra ons diepste meegevoel oor aan die treurende familie."

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At the President's invitation Dr. R. A. Alexander thereupon welcomed the many distinguished guests, visitors and delegates to the Congress and expressed the hope that they would find the proceedings enjoyable.

The President thanked Dr. Alexander for his address and also for his hospitality in allowing the Congress to be held at Onderstepoort.

Mr. J. G. Grobler, President of the South African Agricultural Union was introduced by the President, and in opening the Congress made mention of the importance of veterinary science to the Agricultural industry, where enormous losses from animal disease occurred — in the year ended 31.8.1954, European owned cattle and sheep to the value of nine million pounds were lost.

Organised agriculture wished to see greatly expanded research facilities and staff and also favoured the establishment of diagnostic centres in all important stock regions as well as mobile units for on the spot investigations of new diseases.

The Agricultural Union was in favour of an independent Agricultural Research Council, with a status similar to but not falling under the C.S.I.R. and the speaker hoped that the profession would support this idea.

Mr. Grobler also mentioned the shortage of veterinarians in the country areas and the difficulties attending rural practise. His Union was in favour of short courses for farmers to enable them to help themselves. In conclusion, he mentioned that the main aim of both organisations was to bring the knowledge and services of the profession to as wide a range of farmers as possible, at a cost which farmers can afford, and that in furthering this common aim all avenues must be exploited and explored. Mr. Grobler then declared the Congress formally opened.

The President warmly thanked Mr. Grobler for his inspiring address.

PRESIDENTIAL ADDRESS:

Dr. Snyman delivered a most absorbing address on the growth of veterinary science in South Africa as he had experienced it, and made particular mention of three important phases, viz., the founding and expansion of the Faculty, the union of the research and field divisions in 1926, and legislation which had affected the profession. The faculty had developed in a most striking manner during the last 38 years, when one compared the first lecture in 1920 in a room marked "Ladies' Restroom," to the present magnificent faculty buildings! In conclusion he expressed the hope that the present harmonious co-operation between the various branches of the profession would always be maintained.

OPENING OF TRADE EXHIBITION:

Dr. H. P. Steyn, the Vice-President, wlecomed the trade representatives in a short address and officially opened the Trade Exhibition. Mr. Mayne, President of the Medical Exhibitors Association, replied to the address and presented a cheque for £100 as a donation to the Benevolent Fund from his Association. The President thanked Mr. Mayne for this most generous gift.

UNVEILING OF BUST OF SIR ARNOLD THEILER:

Dr. H. Graf, Dean of the Faculty of Veterinary Science, in opening the proceedings thanked the Association for the opportunity afforded in holding the unveiling ceremony during the proceedings of the Annual Congress, and welcomed the guests, amongst whom were representatives of the University of Pretoria, the Departments of Agriculture, Education and Public Works, also Mr. and Mrs. Coert Steynberg, whose magnificent gift of the bust had made the ceremony possible.

Dr. P. J. du Toit was then introduced by Dr. Graf, and recalled meeting Sir Arnold Theiler and Dr. Green at Armoedsvlakte on the 11th December, 1919, when Sir Arnold was busy with the plans for a curriculum for the veterinary faculty to be founded at Onderstepoort. In paying tribute to Sir Arnold he said, "We must be eternally grateful not only for what Theiler

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himself did for South Africa, but also for what he taught his students and his colleagues; for the inspiration he gave them and left behind him when he passed on."

Mr. Coert Steynberg described his pleasure in being asked to create the bust in London in 1934, and of the further honour he had received in being commissioned to make the granite statue of Sir Arnold at Onderstepoort. He then asked Professor Mönnig to accept the bust for the Faculty, not only as a token of his appreciation of Onderstepoort for what it had meant to him, but more as a proof of his highest regard towards a great man who had done so much for South Africa.

The bust was then unveiled by Dr. Gertrude Theiler, Sir Arnold's daughter, who in a short speech mentioned the fulfilment of her father's life ambition to be a teacher, when he was asked to organise the veterinary faculty.

In accepting the bust on behalf of the University of Pretoria, Dr. H. O. Mönnig warmly thanked Mr. Steynberg for his generosity which was very greatly appreciated. He stressed the great value to everyone of Sir Theiler's example in carrying out his duties to himself, to the community in which he lived and to the branch of science he represented, and expressed the hope that we would always try to emulate this example.

Dr. Graf then brought the ceremony to a close and voiced his conviction that the memorial which had been erected would serve as an inspiration to the students each time they passed it.

After tea had been taken a start was made with the scientific programme, the papers to be delivered having all been issued in galley-proof form to members.

Drs. D. H. G. Irwin and P. J. Schutte introduced their papers on blood and fluid replacement in veterinary practice and were called upon to answer numerous questions. Dr. Kleeberg mentioned the use of plasma in preference to whole blood as being much safer, and Dr. Ryksen described his experience in practice.

At 2 p.m. the paper on Infectious Infertility in Sheep was introduced by Professor S. W. J. van Rensburg, and was followed by a good discussion and a practical demonstration of the diagnostic methods usde.

Thereafter Dr. la Grange introduced his paper on the relationship between conception figures and the time of insemination in cattle. The discussion which followed served to emphasize the role of ovulatory failure and delayed ovulation in the conception results obtained by A.I.

In the evening a large audience attended the showing of films at the Technical College on the treatment of dystokia (posterior and anterior presentation-) and the role of cortico-steroids in clinical medicine.

WEDNESDAY — AUGUST 27th:

At 8.30 a.m. the President invited Dr. J. H. Hofmeyr of the Division of Animal Husbandry to introduce his paper on "Certain Aspects of Pig Progeny Testing," and thereafter the other two papers on Swine Health Control and Management and Diseases of the Sow and Litter were introduced by Drs. Loveday and Abrams respectively. (The discussion which followed has already been published in the Journal).

Professor J. D. W. A. Coles next spoke on the joint paper by himself and Dr. R. B. Cumming on "The Significance and Control of Chronic Respiratory Disease in Poultry." The discussion on this important paper had to be curtailed by the President due to lack of time.

After tea Dr. P. J. Meara illustrated the introduction to his paper on "Antibiotic Contamination of Milk as a Veterinary and Public Health Problem," with a number of graphs, and drew attention to the need for further investigation of the degree of contamination of South African milk supplies.

Various speakers who participated in the subsequent discussion emphasised the role the profession must play in this matter, and the need to bring the question to the notice of the authorities concerned.

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This paper was followed by a most interesting film on B.H.C. poisoning, and Mr. P. M. Bekker thereafter briefly discussed his paper on the spray-dipping of sheep and the shortcomings of the method.

At 2 p.m. Dr. H. P. Steyn proposed the adoption of the New Constitution in the place of Dr. A. M. Diesel who could not be present. This motion was seconded by Dr. R. du Toit. Before the discussion commenced the President drew the attention of members to Rule 12 governing the conduct of Debate.

Dr. Steyn explained that the draft constitution was the result of requests from branches throughout the country and had the approval of Council.

The meeting then went into a detailed discussion of the draft as circulated to all members and approved of the entire draft except for making changes enumerated below:

Articles of Association: Clause 7 was agreed to on the understanding that should the annual subscription at any future date be amended then this Clause 7 would have to be amended simultaneously.

Clause 8: The word "shall" in the second paragraph was changed to "may."

Clause 10: The word "shall" in the second paragraph was changed to "may."

Clause 11: The word "shall" in the second paragraph was changed to "may."

Dr. Mason pointed out that one cannot speak of "non-veterinarians" as in the second paragraph, and it was accordingly agreed to chage this read "accorded to persons who are *not* veterinarians."

Clause 16, was agreed to after Professor van Rensburg had explained that the clause had nothing to do with the Veterinary Board but pertained only to membership of the Association.

Clause 21: The last part of the clause was changed to read "his privileges of membership may thereafter be suspended until such payment be made."

Clause 24: Mr. Wegger, the consultant, explained that the whole of this clause was laid down in the Companies Act and that the word "agreed" just before paragraph (a) should be followed by a semi-colon and not by a full-stop. The meeting thereupon agreed to the Clause with the semi-colon as specified above.

Clause 29 it was pointed out was statutory and was thereupon accepted.

Clause 35: A proposal to alter this clause to allow voting by proxy was made by Dr. Ryksen, and seconded by Dr. Irwin, but the proposal was lost.

Clause 36(i) was amended by the addition of the following to the end of the sentence, "and any other member of the Association as is considered desirable by Council."

Clause 36(vii): An objection to this clause by Dr. L. W. van den Heever was recorded, after the President had ruled that the clause could not be changed as a proposal to amend the preceding paragraph (No. 36(vii)) had already been lost.

Clauses 37 and 38: On the proposal of Dr. M. Bergh, seconded by Capt. J. L. Dickson, the meeting voted to delete both clauses and to revert to the method of electing a President and Vice-President as laid down in the old constitution. It also authorised the President, together with Mr. Wegger and the Committee concerned to put the the wishes of the meeting into the proposed draft and to consider any consequential amendments which might be necessary.

Clause 44: Amended to read, "A member shall be responsible to Council."

Clause45: Agreed to delete the words "a majority of" in the 11th line from the bottom of the clause.

In reply to a question from Dr. Steyn, Mr. Wegger explained that Sections 15 and 64 of the Companies Act provided for the method by which the constitution might be modified and altered, and these could be quoted in the constitution if required. He also explained that by-laws were purely for the internal management of the Association and were normally prepared and adopted

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by Council. A formal vote of thanks to Mr. Wegger was minuted, amidst acclamation following a proposal by Dr. Alexander.

The President expressed the Association's thanks to Drs. Steyn, Diesel, van Heerden and all past secretaries who had served on the Committee.

THURSDAY — AUGUST 28th:

A large number of members filled the auditorium when Dr. van der Linde rose at 8.30 a.m. to deliver the paper on Snotsiekte for Dr. D. J. Louw who could not be present. The paper was followed by an excellent discussion, and was illustrated by a number of interesting colour slides.

At 9 o'clock the meeting listened to a tape recording explaining the aims and objects of the Professional Provident Society and thereafter the many questions of members were replied to by Drs. Glatt and Fraser and Mr. Kohler of the Society, who had kindly consented to attend the meeting. Dr. Steyn then thanked these gentlemen for having taken the trouble to come over and explain the privileges of the Society.

BUSINESS MEETING:

(i) *The Minutes of the 52nd Annual General Meeting* having been published in the Journal of the Association were taken as read, and their adoption moved, seconded and agreed to.

(ii) Matters arising from the Minutes.

(a) Employment of Veterinarians by Co-operative Societies: Dr. Steyn explained to the Meeting that Council representatives had met with members of the S.A. Agricultural Union to explain the complaints raised against the old methods of employment. The Agricultural Union had accepted the basis fully, but had made the not unreasonable representation that in certain circumstances, there was still place for the full-time employment of veterinarians. There was no objections whatsoever to this, where the veterinarian works only for the members of the Co-operative and does the work free of charge. Council was busy drawing up a draft contract for the guidance of members and Co-operatives. On the motion of Professor Clark it was agreed to leave the matter in the hands of the General Purposes Committee of the Council.

(ii) (a) *Employment of Veterinarians by Animal Welfare Societies.* Dr. Steyn reported that on Council's instructions the General Purposes Committee had investigated the whole matter. Speaker and the Hon. Secretary had discussed the matter with the legal adviser and had put the following points to him: (a) Has our Association the right to tell veterinarians working for animal welfare organisations that they must not do clinical work for which fees are charged and paid into the welfare organisation's account, and (b) whether animal care societies, which are welfare organisations, have the right to supply services on which they make a profit?

The legal adviser had given it as his opinion that it was definitely unethical for colleagues to work for welfare organisations under the present circumstances, which constituted a typical case of "farming out." Under present legislation it was competent for this practice to be stopped. After the Veterinary Board had published a warning notice that from a certain date the misuse of veterinary services would no longer be allowed, the Board could take disciplinary action against members who persisted in this practice. Nothing could be done against the welfare organisations concerned. The matter would be reported to the next Council meeting, and the Annual General Meeting was not required to take any action. It was further explained by Dr. Steyn that the above ruling meant that veterinarians working for animal welfare societies would be restricted to doing only welfare work and would not allow these societies to collect fees for clinical work done.

(ii) (c) *Adoption of Draft Schedule of Fees.* After a short discussion the draft was adopted on the resolution of Dr. Wacher seconded by Dr. Tarr.

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(ii) (d) *Resolutions*:

(i) Resolution regarding part-time employment of veterinarians by the State. Council had decided to await developments in view of the possible recommendations of the departmental committee of enquiry into bovine tuberculosis.

(ii) Resolution regarding control of Chronic Respiratory Disease — Council had referred this to the Director of Veterinary Services who replied that the problem was receiving proper and direct attention.

(iii) Resolution regarding improvement of remuneration of State Veterinarians. Council had decided on the advice of the Director of Veterinary Services that nothing could be done about this. It was acknowledged that the remuneration of state-employed veterinarians has been improved at the present time.

(iv) Resolution regarding the control of Faculty by the Department of Agriculture. Council had decided to leave this matter in abeyance.

On the motion of Professor Henning seconded by Professor Bisschop, the meeting decided to instruct Council to reconsider this matter.

(iii) MEMBERSHIP:

(a) Election of New Members: The Hon. Secretary stated that applications for membership had been received from the following veterinarians. The applications had been considered by Council which now recommend them to the meeting for approval.

R. J. Redelinghuis, F. de St. van der Riet, H. J. Venter, R. J. H. Howell, R. J. Thomas, S. K. Bakker, W. G. Barnard, W. Eschenburg, A. Vogelnest, A. M. Day, J. W. Roussouw, B. A. Matson, D. Venter, J. W. van Niekerk, I C. Horak, C. J. Mare, S. J. van Rensburg, Miss L. van der Merwe, L. W. Langlands, R. Worthington, D. J. Jarvie, C. H. B. Marlow, L. Black and G. C. Steyn.

With the exception of R. J. Thomas (M.R.C.V.S.) all the other applicants were graduates of the local faculty.

Unanimously approved.

After discussion the meeting also decided to approve the membership of J. G. Pienaar, subject to his making the necessary application.

(b) Resignations: The Hon. Secretary indicated that there were no resignations. The Hon. Treasurer then asked that the name of Dr. C. E. Isaacs, now resident in New Zealand, be removed from the list of members as being four years in arrears with subscriptions, and that the subscriptions be written off as a bad debt. Agreed.

Another member now resident in New York had paid her subscriptions and asked for membership to be continued.

The meeting also decided on the recommendation of the Hon. Treasurer to allow the name of a member to remain on the membership list pending a further attempt to recover outstanding subscriptions.

The Hon. Secretary announced that the following had become Life Members of the Association. Drs. W. Allchurch, P. L. le Roux, N. F. Viljoen, R. du Toit, W. J. Ryksen, M. Sterne, L. Stonier, G. Watt, J. S. Watt and J. Bekker.

(iv) CONSTITUTION OF COUNCIL:

The Chairman announced the constitution of Council:

President: Dr. H. P. Steyn.
Vice-President: Dr. R. du Toit.
Hon. Secretary: Dr. S. van Heerden.
Hon. Treasurer: Dr. G. D. Sutton.
Members 1958-1960: Drs. P. S. Snyman, M. de Lange, A. F. Tarr and and M. C. Lambrechts.
Members 1957-1959: Drs. M. W. Henning, C. F. B. Hofmeyr, J. W. Pols and L. W. van den Heever

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As Dr. du Toit had ceased to be an ordinary member of Council on his elevation to the Vice-Presidency, the member with the next highest number of votes in the election, in terms of accepted practice, viz., Dr. L. W. van den Heever, comes into Council for the remaining year of Dr. R. du Toit's term of office.

After thanking Council for its support during his term of office, Dr. Snyman inducted the new President. Dr. Steyn pledged himself to serve the Association to the very best of his ability.

The President thereupon announced that Drs. H. P. Steyn and S. W. J. van Rensburg had been elected to the Veterinary Board as the Association's representatives.

(v) REPORTS:

(a) Balance Sheet and Auditor's Report. These had been circulated to all members and were explained by the Hon. Treasurer. There was very little excess of income over expenditure. Council had decided to limit the credit allowed to any student to £25, and if an account was unpaid within 90 days no further credit would be allowed. The expense involved in printing the Journal was high and Dr. du Toit suggested it might be worthwhile to investigate the cost of printing in Holland. Dr. Henning suggested the possibility of obtaining assistance in this matter from the Department of Education. Adoption was then moved, seconded and accepted.

(b) Standing Committees: These reports had been circulated, and after Professor Clark had appealed to members for books for the Students' Library, these reports were adopted by the Meeting.

(vi) GENERAL:

Dr. Kleeberg proposed the use of the word "Dierarts" instead of "Veearts" for the profession. The present name had been approved some years ago when the matter had then been discussed.

Dr. MacFarlane proposed that the holding of Congresses elsewhere than at Onderstepoort be considered. The President explained that the matter had already been investigated by Council and found to be impractical, but the resolution would be noted and Council would send out a circular during the year to ascertain members' views.

Dr. Uys appealed to members to make available photographs of interesting cases for a permanent collection which could also be used for the training of students.

The Hon. Secretary drew members' attention to the Third World Congress on Fertility and Sterility and stated that application forms could be obtained from him.

Dr. Meredith drew attention to the fact that the Association's Journal was widely read overseas, where Afrikaans articles could not always be understood. The President pointed out that at a National Congress it was desirable to use Afrikaans as often as possible in order to help in extending its vocabulary of technical terms, but a brief summary of what had been said could be given in English for the benefit of visitors. The same applied to articles in the Journal.

Dr. la Grange enquired regarding the request of the A.I. Co-ops. to Council for a form to be drawn up for veterinary examination for sterility. The President indicated that this matter was still being finalised by Council.

(vii) RESOLUTIONS:

1. Moved by Dr. Diesel: "That the Veterinary Board be asked to insert into the Code this particular requirement: 'This meeting resolves that it is unethical for a veterinary surgeon to castrate or spay any adult animal, that is any animal having reached sexual maturity, without the use of a suitable anaesthetic. Likewise, it is unethical to perform any major operation on such animal without adequate or suitable anaesthetic." Seconded and agreed.

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2. Moved by Dr. van den Heever: "This 53rd Annual General Meeting of the S.A. Veterinary Medical Association is most concerned about the possible harmful effects of antibiotic contaminated milk. These include effects on the human consumers, the manufacture of dairy products and the susceptibility of bacteria to such antibiotics."

Council is therefore urged to approach the Departments of Health and Agriculture with a view to instituting an immediate investigation into the degree of such contamination of South African mlk supplies, its effects and to consider methods of control should this be deemed necessary.

3. A motion by Professor Henning in regard to the establishment of diagnostic centres was discussed but obtained no seconder.

4. Proposed by Professor Clark: "That this meeting welcomes the assurance of the Director of Veterinary Services that the policy of regional diagnostic centres is going to be implemented and the Association gives him the assurance that this policy has the wholehearted support of the profession." Seconded by Dr. Diesel — Agreed.

5. Voorgestel deur Professor van der Walt: "Hierdie vergadering versoek die bestuur van die Vereniging om 'n permanente komitee aan te stel met die volgende doelstellings:—

(i) Om in gevalle waar veeartse deeltydse werk en besoldiging van liggame of individue aanneem, die onderhandeling van hierdie kontrakte ten behoewe van die betrokke veearts te onderneem en deur te voer.

(ii) Om die gebruik te propageer by veeartse wat nuwe betrekkings of praktyk aanvaar, om dit te doen slegs na raadpleging met hierdie komitee."

Sekondeer Dr. Irwin — Aangeneem.

6. Voorgestel deur Dr. la Grange: "Hierdie vergadering versoek ons geëerde Raad om die wenslikheid van korrektiewe operasies op potensiële stamboekkalwers met erflikheidsgebreke te ondersoek en op ons volgende Algemene Vergadering verslag te doen. Korrektiewe chirurgiese behandelinge vir erflike gebreke hou volgens hierdie Kongres potensiële bedreigings in vir ons land se veestapels, veral waar K.I. toegepas word."

Gesekondeer en aangeneem.

7. Proposed by Dr. A. J. Louw: "This meeting of the South African Veterinary Medical Association be requested to take immediate steps for the necessary alteration of the Constitution of the Association, as provided for in the said Constitution, in order to enable Council to appoint a full-time Secretary/Treasurer who will also be Public Relaions Officer for the Profession."

Seconded by Professor Bisschop. — Agreed.

(viii) ADJOURNMENT:

The President expressed the meeting's thanks to Mesdames Glover & Klerck for their assistance and to the ladies responsible for the floral decoration of the platform and round the Theiler bust.

At 2 p.m. a numbre of members and guests visited the C.S.I.R. on an organised tour.

Several members participated in a competition at the Pretoria Country Club for the P. J. du Toit Golf Trophy, others participated in a bowls tournament for the R.A. Alexander Bowls Trophy.

At 6 p.m. members and guests gathered for refreshments and dancing at the Pretoria Country Club, where a most enjoyable evening was spent.

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THE SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION

MINUTES OF COUNCIL MEETING HELD ON MONDAY THE 24TH OF NOVEMBER, 1958, AT THE MEAT BOARD BUILDING, PRETORIA

PRESENT:

Dr. H. P. Steyn (President), Drs. P. S. Snyman, P. J. du Toit, A. C. Kirkpatrick, E. M. Robinson, A. M. Diesel, M. C. Lambrechts, C. F. B. Hofmeyr, J. W. Pols, S. W. J. van Rensburg, A. F. Tarr, R. A. Alexander, L. W. van den Heever, M. de Lange, G. D. Sutton (Hon. Treasurer), S. van Heerden (Hon. Secretary) and R. K. Loveday (co-opted).

Apologies for absence were received from Drs. R. du Toit and H. H. Curson.

1. MATTERS ARISING FROM THE MINUTES:

(a) Letter from the Director of Veterinary Services regarding the sale of Distemper Vaccine, read. The Director of Veterinary Services was prepared to give serious consideration to any valid reasons which might be advanced as to why the vaccine should only be administered by veterinarians. To date no single valid reason had been advanced why this should be done. — Noted.

(b) Letter from Dr. A. D. Thomas read, wherein it was pointed out that Council had previously agreed that all monies accruing from this source (trading in books, etc.), should be accounted for separately, and at no time should they be merged into or utilised for general revenue, benevolent or purposes other than those stated in the letter. On the President's suggestion it was agreed to refer this matter to the Finance Committee for consideration and report back to Council.

2. MATTERS ARISING FROM 1958 ANNUAL GENERAL MEETING:

(a) Motion of condolence at the death of the Prime Minister. The motion had been communicated to the Office of the Prime Minister and an acknowledgement received.

(b) Resolution regarding full-time Secretary and Public Relations Officer. Agreed to refer this matter to the Finance Committee.

(c) Resolution regarding Antibiotics in Milk. Council agreed to delegate Dr. van den Heever to discuss this matter with the Secretary for Public Health, also that the motion should be communicated to the Minister of Agriculture (Economics and Markets) together with a copy of the summary of Dr. Meara's paper.

(d) Employment Committee. Agreed to refer this resolution to the General Purposes Committee.

(e) Castration or spaying of sexually mature animals without suitable anaesthetic. Agreed to publish this resolution in the Journal and also to refer it to the Veterinary Board.

(f) Surgical correction of hereditary defects in potential stud animals. It was agreed that Drs. van Rensburg and le Grange would prepare an informative article on this subject for the Journal.

3. NEW CONSTITUTION:

Dr. Diesel explained that the finalisation of the new constitution in terms of the decision of the Annual General Meeting had been delayed by the illness of Mr. Wegger. The matters to be completed (Election of President and

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Vice-President by members; explanatory paragraph re alterations to the constitution and the by-laws) would be dealt with by Mr. Wegger as soon as possible, and would be reported back to the next Council meeting.

4. CONTRACTS BETWEEN MEMBERS AND NON-VETERINARIANS:

Dr. Diesel explained that the model contract (appendix B) before the meeting was drawn up from the Moorreesburg and Senekal contracts for submission to the law advisers. It was agreed, after discussion, that Clause 7(i) (calculation of net income) would, if possible, be assessed on a cash received basis. Clause 6(d) was to be amended so as to debit all expenses allowed by the Receiver of Revenue. Clause 5 required consideration as to the period which must elapse before the contract could be revised by either party, as also the use of the word "indienstreding" in the same clause. It was further agreed to refer the model contract back to the General Purposes Committee for revision and submission to the law adviser, and that Dr. Hofmeyr would accompany the Secretary when interviewing the law adviser on this matter.

5. MEMBERSHIP:

(a) Applications for membership by—

(i) David Gordon Cloe, 190, Ridge Road, Central Durban.
Proposed by L. R. Morford, seconded by V. J. E. Amos.

(ii) J. G. Pienaar, Bray, P.O. Kirstonia, via Vryburg.
Proposed by J. L. Dickson, seconded by W. C. Viljoen.

(iii) W. H. Chase, P.O. Box 251, Mafeking.
Proposed by A. C. Kirkpatrick, seconded by P. J. du Toit.

Were approved by Council for submission to the next Annual General Meeting.

(b) The Hon. Secretary informed Council of the death of a member, Dr. I. Mowat, of Johannesburg, and the meeting stood as a mark of respect. It was agreed that Council's sympathy be conveyed to the widow.

6. ELECTION OF STANDING SUB-COMMITTEES:

The following Committees were elected:—

(i) General Purposes: Dr. R. du Toit (ex officio) Convenor; Members: Drs. Clark, Hofmeyr, Wheeler and Lambrechts.

(ii) Finance: Dr. S. W. J. van Rensburg, Convenor; Members: Drs. R. Alexander, M. C. Lambrechts.

(iii) Editorial: Dr. E. M. Robinson's letter of resignation was read and accepted with regret by Council. The President thanked Dr. Robinson on behalf of Council and the profession for his valuable services as Editor.

Professor R. Clark was proposed as the new Editor and Drs. Diesel, Robinson and de Boom as members of the Committee.

(iv) Library: Professor R. Clark, Convenor; Members: Drs. T. Alexander and R. du Toit.

(v) Disciplinary and Fees Adjudication: Dr. P. S. Snyman, Convenor; Members: Drs. A. F. Tarr, C. F. B. Hofmeyr, J. W. Pols and L. W. van den Heever

(vi) Book Fund: Dr. G. D. Sutton, Convenor; Members: Drs. J. W. Pols and W. D. Malherbe.

7. CIRCULAR RE APPLYING FOR PROFESSIONAL POSTS ADVERTISED IN LAY PRESS ONLY:

The Hon. Secretary pointed out that the Medical Profession applied only for posts advertised in the professional press.

After discussion it was agreed, on the motion of Dr. Alexander, seconded by Dr. Tarr, to notify members by a notice in the Journal, that they should not apply for posts not advertised in the professional press, and that members

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should also notify the Secretary of any posts being advertised only in the lay press.

At the suggestion of Dr. Hofmeyr it was also agreed to add this information to the letter sent to new graduates and to forward a copy of the Journal notice to the Veterinary Board.

8. CONDITIONS OF ENPLOYMENT OF VETERINARIANS BY ANIMAL WELFARE ORGANISATIONS:

After discussion of the legal opinion, it was decided that a warning notice would be sent to all members in regard to "farming out" of their services to an animal welfare organisation, which would become illegal after a certain date.

Also agreed to forward to the Veterinary Board a copy of the warning notice, together with a copy of the legal opinion and the 1957 Annual General Meeting resolution on this matter.

9. SUGGESTED TRANSFER OF VETERINARY RESEARCH TO C.S.I.R. BY THE DU TOIT COMMISSION:

After Dr. du Toit had explained what had led up to the Commission's recommendation, it was decided, after a full discussion, on the motion of Dr. Hofmeyr, seconded by Dr. Lambrechts, to appoint a small committee of 4 members to study the implications of the Report's recommendations, and to make recommendations to Council.

The Committee elected was Dr. Steyn (Convenor), Drs. Alexander and Lambrechts and Jansen. At the suggestion of Dr. Alexander, it was agreed that Dr. Le Clus, the Director of the S.A.U. should be co-opted to this committee when required.

10. MEMORANDUM OF PUBLIC HEALTH GROUP TO BE SUBMITTED TO BORCKENHAGEN COMMITTEE:

Dr. van den Heever explained that the Public Health Group had been unable to agree on a memorandum, and also outlined for the information of Council, the purpose of the Borckenhagen Committee and the position of the profession in non-personal health services.

After discussion Council agreed to a suggestion by Dr. van Rensburg that the Public Health Group should meet with the General Purposes Committee to consider this matter.

11. FINANCE:

After it had been pointed out that numerous advertisements in the Journal were being lost and not being renewed, Council agreed to instruct the Edito ial Committee to appoint one of its members specially to handle this matter.

12. CORRESPONDENCE:

(i) Letter from Secretary of Faculty thanking the Association for its kind co-operation and hospitality regarding the unveiling of the Theiler bust at the recent Congress, noted.

(ii) Letter from Dr. P. J. Meara pointing out that the Commissioner of Inland Revenue has recently approved the principle that all medical practitioners in full-time empoyment may deduct from taxable income subscriptions to the Medical Association and to medical journals, for purposes of assessing taxable income, and enquiring whether a similar concession could not be obtained for members of the S.A.V.M.A. in full-time employmepnt.

Council agreed to take the matter up with the Commissioner and to advise Dr. Meara accordingly.

(iii) Professional Provident Society's letter detailing two new benefits, viz. Group Life Assurance and Hospitalisation benefits. Agreed to publish this in the Journal.

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(iv) International Veterinary Congress enquiry regarding representative for coming year. Have been notified that Dr. R. A. Alexander has been re-elected.

(v) University of Pretoria advising the creation of an Agricura Bursary of £300 p.a. from 1959, and that the S.A.V.M.A. is entitled to nominate one member on the selection committee. Agreed to write thanking Agricura for bursary and also write to Registrar, University of Pretoria noting bursary with pleasure.

13. GENERAL:

Dr. Lambrechts expressed his appreciation of the honour accorded him in representing the S.A.V.M.A. at the recent B.V.A. Congress, and felt that the Association could usefully adopt some of the methods used in organising this Congress.

The meeting closed at 6 p.m. with a vote of thanks to the Chair.

BOERE-BYSTANDSWET

PERSVERKLARING

As gevolg van 'n sameloop van omstandighede gebore uit die abnormale toestande van die Tweede Wêreldoorlog met sy nasleep en ongunstige klimaatstoestande oor 'n aantal onlangse jare, bevind 'n groot aantal boere in verskeie dele van die Unie hulle vandag in 'n moeilike finansiële toestand.

Die Regering besef dat die boer deur omstandighede wat gedurende die jongste oorlog ontstaan het op aanmoediging van die owerheid sekere produksierigtings ingeslaan het teneinde nypende voedseltekorte te bowe te kom. Hierdie produksierigtings het meganisasie op 'n groot skaal genoodsaak en alhoewel dit meegewerk het om die voedseltekorte uit te wis, het dit enersyds vir baie boere 'n abnormale skuldelas meegebring, en andersyds die uitputting van landbougronde tot gevolg gehad. Vir 'n groot aantal het dit 'n vry ernstige finansiële toestand geskep wat deur seisoensterugslae verskerp is. Daarom is dit dat die Regering besluit het om boere in sommige gevalle te help met die konsolidasie van hulle skulde, of waar boere te ver agter geraak het weereens finansieel gesond te maak onder die Boere-Bystandswet.

Die hulp wat hierdeur beoog word sal beperk wees tot verdienstelike *bona fide* boere wat nie in staat is om hulle self te help nie, of wat nie deur die gewone kanale gehelp kan word nie, en dit moet op 'n streng ekonomiese grondslag berus. Hierdie hulp kan slegs daarin slaag om gesonde toestande in die landboubedryf te skep indien applikante onderneem, waar nodig, hulle boerderye so te rasionaliseer of te hervorm dat hulle voortaan ekonomies selfstandig sal wees. Hierdie hervorming sal gelyktydig deurgevoer moet word in samewerking met die Departement van Landbou-tegniese Dienste wat boere sal bystaan met die beplanning van 'n gesonde en aangepaste boerderystelsel vir elke ekologiese streek. Waar die voorsiening van vee of ander boerderybenodigdhede noodsaaklik is om die doel te bereik sal hulp daarvoor ook beskikbaar wees.

Hierdie bystandsplan beoog suiwer en alleen die bevordering van 'n gesonde landbou-ekonomie waar soiets bewerkstellig kan word deur finansiële steun aan boere wat daardeur weer hulle boerdery op 'n gesonde ekonomiese grondslag kan plaas.

Die bystandsplan sal enersyds deur die Landbank en andersyds deur die Boere-Bystandsraad behartig word. Die Landbank raad het onderneem om sy beleid ten opsigte van die betaling van drukkende skulde en die voorsiening van bedryfskapitaal te verslap met ingang vanaf 1 Januarie 1959. Hy sal dus bereid wees om boere in dié opsig by te staan waar dit moontlik is binne die

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perke van sy tans bestaande bevoegdhede. En hierdie bevoegdhede sal by wetgewing uitgebrei word.

Konsolidasie van skulde kan egter die perke van die Landbank se bevoegdheid oortref en in daardie gevalle sal applikante hulle tot die Boere-Bystandsraad moet wend. Omdat die Boere-Bystandsraad slegs finansiële bystand kan verleen tot die mate van die redelike waarde van die applikant se totale bates volg dit dat die prosedure van die Boere-Bystandswet by die finansiële suiwering van die applikant, in sommige gevalle altans, eers toegepas sal moet word voor en aleer die appplikant gerehabiliteer kan word.

Dit is besluit dat met ingang vanaf 1 Januarie 1959 die Boere-Bystandsraad weereens finansiële hulp aan boere sal verleen. Die hulp wat verleen word sal egter beperk wees tot eise wat ten tye van hierdie aankondiging reeds bestaan het. Met die oog daarop word die toepassing van moratoria onder die Boere-Bystandswet dadelik gestaak alhoewel bestaande beskermings geldig sal bly. Hersiening in sommige gevalle is egter moontlik.

Hierdie bystandskema sal van beperkte duur wees en boere wat daarvoor in aanmerking wil kom, sal voor 30 Junie 1959 aansoek moet doen. Laat aansoeke sal onder geen omstandighede oorweeg kan word nie.

Volledige besonderhede van die beoogde hulp onder die Boere-Bystandswet sal na inwerkingtreding van die skema van alle Landdroste op die platteland verkry kan word.

Vir hulp deur die Landbank moet daar op die gewone wyse aansoek gedoen word.

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OUTBREAKS OF SCHEDULED DISEASES IN THE UNION OF SOUTH AFRICA DURING THE PERIOD 1.6.58—30.8.58

DISEASE	*CAPE*	*NATAL*	*O.F.S.*	*TRANSVAAL*
Anthrax	2 East London 1 Bellville 1 Gordonia	5 L. Umfolosi 1 Klipriver 1 Ndwedwe	2 Frankfort 2 Heilbron 1 Winburg 3 Bloemfontein 1 Bethelehem 1 Kroonstad 1 Theunissen	1 Randfontein 1 Mafeking
Foot and Mouth Disease	1 Vryburg		1 Boshoff	1 Barberton
Fowl Typhoid	1 De Aar			
Johne's Disease	1 Stutterheim 1 Uady Grey			1 Belfast
Lumpy Skin Disease	9 George 2 Mossel Bay 1 Riversdale 3 Ngqeleni 1 Cradock 1 Elliotdale 1 Mqanduli 1 Tsolo 2 Umtata 1 Indwe	1 Utrecht 1 Harding	1 Hoopstad 1 Vrededorp	1 Vanderbijlpark 3 Ermelo 1 Letaba 1 Carolina 1 Randfontein 2 Bronkhorstspruit 1 Delmas 1 Rustenburg 5 Heidelberg 1 Vereeniging 1 Nigel 1 Potchefstroom 2 Zoutpansberg 1 Ventersdorp 1 Standerton 1 Pretoria
Mange (Goat)	1 Elliotdale 1 Peddie 4 Glen Grey 1 King William's Town 1 Colesberg 3 Butterworth 2 Nqamakwe 1 Moanduli	1 Inanda 1 Camperdown 1 Umlazi		1 Barberton
Mange (Porcine)	1 Bellville		3 Warrenton	1 Klerksdorp Rustenburg
Mange (Bovine)				1 Lydenburg 1 Rustenburg
Mange (Equine)	3 Keiskamahoek 2 Libode 2 Nqamakwe 2 Tsomo 5 St. Marks 2 Mt. Fletcher 3 King William's Town 1 Elliotdale 1 Engcobo 2 Kentani 1 Glen Grey 2 Umtata	3 Lionsriver 1 Utrecht	1 Harrismith	1 Rustenburg
Psittacose				1 Pretoria
Rabies	1 Vryburg 1 Maraisburg 1 Colesberg 1 Steynsburg		2 Boshoff 1 Heilbron 1 Wesselsbron 1 Thabanchu 1 Welkom 1 Bloemfontein	1 Pietersburg 1 Sibasa
Scab Sheep				3 Mafeking
Swine Erysipeles	1 Kokstad	1 Lionsriver		
Trypanosomiasis (Dourine)	2 Gordonia			
Tuberclosis (Bovine)	1 Riversdale 1 Somerset West 1 Springbok 1 Paarl 1 Bedford 1 Bathurst	2 Pietermaritzburg 1 Camperdown 2 Durban 1 Inanda 1 Lower Tugela 4 Umzinto 1 Vryheid 2 Poulpietersburg 1 New Hanover	1 Bultfontein 1 Ventersburg 1 Bloemfontein	1 Ermelo 2 Bronkhorstspruit 1 Potgietersrust 1 Warmbad
Tuberculosis (Avian)	1 Caledon			

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OUTBREAKS OF SCHEDULED DISEASES IN THE UNION OF SOUTH AFRICA DURING THE PERIOD 31.8.58—30.11.58

DISEASE	*CAPE*	*NATAL*	*O.F.S.*	*TRANSVAAL*
Anthrax	1 Kimberley 1 Matatiele	1 Eshowe 1 Estcourt 1 Hlabisa 1 Newcastle 1 Umvoti	5 Bloemfontein 1 Heilbron 1 Vrede 1 Senekal	1 Johannesburg 2 Klerksdorp
Epididymitis and Vaginitis				2 Rustenburg
Foot and Mouth Disease	1 Vryburg			
Fowl Cholera				1 Pretoria
Johne's Disease	1 Cathcart			1 Ermelo
Lumpy Skin Disease	1 De Aar 1 George 1 Ngqueleni	1 Ixopo 2 Ngotshe	1 Bultfontein 3 Bethlehem 1 Hoopstad 1 Vredefort 1 Wesselsbron	12 Ermelo 1 Carolin 1 Heidelberg 2 Lydenburg 4 Potchefstroom 1 Pretoria 1 Vereeniging 2 Zoutpansberg
Rabies	1 Colesberg 1 Maraisburg 3 Steynsburg 1 Venterstad 3 Vryburg		1 Boshof 1 Ficksburg 3 Kroonstad 1 Senekal 1 Viljoenskroon 2 Thaba Nchu	3 Letaba
Sheep Scab	4 Mafeking			
Trypanosomiasis (Nagana)		1 Hlabisa		
Mange (Equine)	1 Flagstaff 1 Kentani 2 Mt. Fletcher 1 Qumbu 7 St. Marks 1 Tsolo 1 Tsomo 1 Umzimkulu 1 Umtata 1 Victoria East	4 Ingwavuma 2 Nongoma 1 Umlazi		
Mange (Goat)	3 Glne Grey 1 Kentani 2 Komgha 2 King William's Town 4 Middeldrift 1 Mqanduli 1 Nqamakwe 1 St. Marks 2 Tsomo			
Mange (Porcine)	2 Warrenton 1 Hanover 1 Mqanduli 1 Elliotdale			1 Rustenburg
Tuberculosis (Bovine)	2 Albany 1 Komgha 1 Malmesbury 1 Oudtshoorn 2 Paarl 1 Piquetburg 1 Stellenbosch 1 Worcester	1 Babanango 4 Camperdown 1 Dundee 1 Inanda 2 Paulpietersburg 1 Pietermaritzburg 2 Polela 3 Pinetown 1 Port Shepstone	1 Odendaalsrus 1 Senekal 1 Zastron	1 Boksburg 1 Bronkhorstspruit 1 Delmas 1 Heidelberg 4 Klerksdorp 1 Letaba 1 Potchefstroom 1 Potgietersrust 1 Prteoria 1 Ventersdorp 1 Randfontein 1 Waterberg 1 Zoutpansberg
Tuberculosis (Avian)	1 Elliot		1 Senekal	
Tuberculosis (Porcine)	1 Worcester			

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OUTBREAKS OF SCHEDULED DISEASES IN THE UNION OF SOUTH AFRICA DURING THE UERIOD 1.12.58—28.2.59

DISEASE	*CAPE*	*NATAL*	*O.F.S.*	*TRANSVAAL*
Anthrax	1 Bellville 2 East London 1 Mafeking 1 Stutterheim 1 Victoria East 1 Yynberg	1 Bergville 1 Harding 1 Ubombo	1 Bloemfontein 1 Heilbron 2 Wesselsbron	1 Bronkhorstspruit 1 Klerksdorp 2 Lichtenburg 1 Potchefstroom 1 Standerton 1 Wolmaransstad 1 Ventersdorp
Epididymitis				
Epizootic Lymphangitis	1 King William's Town			
Foot and Mouth Disease				5 Barberton 21 Pilgrims Rest
Fowl Typhoid	1 Albany			1 Brakpan
Lumpy Skin Disease	6 Elliot 1 Engcobo 3 Indwe 3 Kentani 1 Kimberley 6 Maclear 1 Ngquelent 1 Qumbu 1 Riversdale 1 Somerset West 1 Tsolo 3 Umtata 1 Vryburg 1 Willowvale	1 Bergville 2 Dundee 5 Eshowe 1 Inanda 1 Ixopo 2 Klipriver 1 Mahlabatini 2 Newcastle 2 Nongoma 1 Umfolozi 3 Umvoti 3 Utretcht	13 Bethlehem 1 Boshof 1 Ficksburg 6 Frankfort 3 Harrismith 1 Hoopstad 6 Heilbron 4 Jacobsdal 15 Kroonstad 4 Ladybrand 5 Lindley 12 Reitz 1 Senekal 1 Thaba 'Nchu 1 Ventersburg 2 Viljoenskroon 1 Vrede 1 Winburg	1 Bronkhorstspruit 3 Carolina 2 Delmas 1 Delareyville 9 Ermelo 3 Germiston 8 Heidelberg 15 Johannesburg 7 Krugersdorp 1 Middelburg 20 Potchefstroom 2 Pietersburg 3 Piet Retief 1 Potgietersrus 6 Pretoria 3 Randfontein 2 Rustenburg 1 Schweizer Reneke 2 Standerton 3 Ventersdorp 2 Witbank 11 Zoutpansberg
Mange (Goat)	1 Elliotdale 2 Middeldrift 4 Mqanduli 1 Victoria East	2 Ingwavuma		
.. (Equine)	1 East London 1 King William's Town 2 Middeldrift 1 Umzimkulu 1 Flagstaff	Estcourt 1 Umvoti		
.. (Porcine)				1 Johannesburg
Psittacosis			1 Ladybrand	
Rabies	1 Maraisburg		1 Bloemfontein 1 Kroonstad 1 Theunissen 1 Wesselsbron	1 Letaba 1 Sibasa 1 Zoutpansberg
Sheep Scab	2 Mafeking			1 Lichtenburg
Tuberculosis (Avian)	1 Umtata			
Tuberculosis (Bovine)	1 Alexandria 1 George 1 Gordonia 1 Komgha 1 Mossel Bay 1 Springbok 1 Taung 1 Williston	1 Durban 2 Pietermaritzburg	1 Bethlehem 1 Boshof 1 Fauresmith 1 Kroonstad 1 Theunissne 1 Virginia	1 Brakpan 1 Nigel 1 Pietersburg 3 Piet Retief 2 Potgietersrus 2 Rustenburg 4 Ventersdorp 1 Vereeniging
Tuberculosis (Porcine)	1 Queenstown			

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Jl. S.A.V.M.A. 30 (2), 1959

THE TREATMENT OF TUBERCULOSIS IN MAN AND ANIMALS WITH ISONIAZID. PRELIMINARY REPORT

H. H. KLEEBERG
Onderstepoort

Isonicotinic acid hydrazide (INH) or Isoniazid — the international name recommended by WHO — was first synthesized by Meyer and Mally in 1912 in Prague. Its tuberculostatic properties were discovered independently in 1952 by two groups of scientists, one in the U.S.A. (Hoffman — La Roche) and the other in Germany (Bayer — Leverkusen). Their work was based on the discovery of thiosemicarbazone by the Nobel prize winner Domagk in 1946. It has proved to be the most potent drug against tuberculosis yet available. Isoniazid is widely used all over the world and is manufactured under different names, alone or in combination with other anti-tuberculous drugs. It is available as oral tablets of 50, 100 and 200 mgm. and is cheaper than the two other principal drugs used against tuberculosis — streptomycin and PAS (para-aminosalicylic acid). Isoniazid is highly soluble in water and is easily absorbed by the alimentary canal and enters all body fluids and tissues readily. It is an ideal chemotherapeutic agent since it is effective in very low concentrations (0.5 microgram/ml. serum). After entering the bacterial cell isoniazid interferes with its metabolism by replacing an important enzyme, thus inhibiting further multiplication of the bacterium. It is ineffective against bacteria which are not multiplying.

Isoniazid is being used very successfully in the treatment of pulmonary tuberculosis, lupus, tubercular meningitis and all other forms of tuberculosis in humans. The results of isoniazid therapy can be particularly well observed in progressive pulmonary tuberculosis. After 3 months of daily administration 65% of the patients gained weight considerably, 70% showed normalisation or improvement of the sedimentation rate, 60% an improvement of the radiological findings, 77% a decrease of the amount of sputum, 60% the disappearance of tuberculosis bacilli from the sputum and 70% a normalisation of the body temperature. However, the sensitivity of the organisms to isoniazid decreased during the same period in 65% of patients. This drug resistance can be found from the 50th to the 70th day of treatment onwards. After five months of administration only 21% of strains recovered from

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patients are fully sensitive to the drug. Today, therefore, monotherapy is no longer favoured but has been replaced by combined regimes such as isoniazid with streptomycin or PAS or all three drugs together. The treatment should be continued for at least 6 months. In cases with persistent chronic lesions which cannot be treated surgically, long term treatment with isoniazid (1 to 2 years) is recommended to prevent haematogenous spread of infection.

Isoniazid has been tested *in vitro* and *in vivo* and proved to be effective against turberculosis in mice, guinea-pigs, rabbits, hamsters, dogs and monkeys. It has also been used in zoo animals, ruminants, small carnivores and kangaroos, with generally good results. In some species reactivation of infection seems to take place soon after cessation of treatment. Italian veterinary investigators were the first to test isoniazid in tuberculosis in cattle. The scope of this article does not permit details of the work, but different authors reported that treatment of heavily diseased animals resulted in improved condition, weight gains, disappearance of most of the clinical symptoms and normalisation of the blood picture. Most of these authors state that the treatment has little or no influence on the intradermal tuberculin reaction. The periods of administration varied from 30 to 190 days and the dosage ranged between 4 and 12 mgm/Kg. body weight. Experiments in Germany and Italy, in which isoniazid was used prophylactically, led to the opinion that the infection can be prevented by the daily dosing of 7 to 10 mgm./Kg.

The Division of Veterinary Services in the Union of South Africa started experimental work on the chemotherapy of bovine tuberculosis in January, 1958, and at present over 1,000 cattle in the field and the Onderstepoort Institute are under study. Of these 450 animals are naturally infected. Firstly, the most practical and economic method of administration was determined. A continuous high serum level of isoniazid is essential for effective therapy and dosing the dry powder produced the highest blood levels. It was determined by chemical assays that a daily dosage of 10 mgm/Kg. body weight created an effective therapeutic serum level for at least 18 hours per day. Several toxic effects in humans, dogs and cats due to isoniazid have been reported but the tolerance in cattle is high. None of the different side effects seen in man could be observed in cattle, even when dosed with 20 mgm/Kg. bodyweight for periods of 6 months. The excretion of isoniazid via the milk causes undesirable contamination. However, assays showed that milk is free of the drug 24 hours after dosing. Twelve hours after dosing, one gallon of milk contains about 1 mgm. isoniazid, and even this amount disappears rapidly on transport, storage, pasteurisation or dilution with other milk.

In accordance with Italian findings we observed a rapid improvement in the general condition of severely diseased animals,

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with weight gain, the cessation of coughing, the closing of open lung processes and the disappearance of tuberculosis bacilli from the milk derived from tuberculosis udders. Up to the present, only one case of reactivation has been observed judged on 6 months' observation on 53 cows (including 15 autopsies) previously treated for 8 months.

Our experiences with the tuberculin test, based on 3 monthly intradermal tests of 330 treated cattle, are contradictory to those of the Italian authors. In 74% of these treated tuberculin reactors, the sensitivity to PPD tuberculin decreased steadily. The characteristics of a typical positive reaction, i.e. pain, heat and diffuse oedema, disappeared gradually and tended to become those of a non-specific hard, circumscribed swelling. After 3 months of daily dosing (10 mgm/Kg.) 12% of the animals gave negative tests and after 6 months 29% were non-reactors. The reaction may again become positive several months after the treatment has been stopped (16 cases to date). The complement fixation titres of 80 treated cattle were followed over periods of 3 to 12 months. In 50% of these cases the titres fell during treatment while in 25% they remained unchanged and in 25% they rose. In 80% of the last category, sensitivity to the tuberculin test decreased simultaneously. This proves that there is no correlation between complement fixation titre and skin sensitivity. A full explanation for the desensitisation of the skin cannot yet be given. The hypothesis that the specific antibodies disappear is contradicted by the following facts:-

(a) the return of sensitivity some time after treatment;
(b) the persistence of positive serum titres;
(c) the finding of lesions containing viable bacilli in some of these animals;
(d) the favourable response to treatment shown by all these animals not correlated to the disappearance of the tuberculin reaction.

A direct interference by isoniazid with tuberculin can also be excluded, since the injection of isoniazid directly at the site of the tuberculin test does not cause any depression of positive reactions. The most likely explanation for the phenomenon of desensitisation is the increased amount of cortisone in the reticuloendothelial system of the skin. It is known that isoniazid stimulates ACTH activity, thereby increasing the weight of the adrenals, probably resulting in an overproduction of cortisone. The depression of the tuberculin reaction by cortisone is well known in man and different animals.

Up to the present, autopsies have been done on 63 adult cattle which had received isoniazid for periods of 2 to 10 months. Generalised cases (8 cows), chronic pulmonary infection (60%) and all other forms of tuberculosis were encountered. Chronic lung lesions showed a particularly good response to treatment. Cavities

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were emptied, abscesses of all sizes were thickly encapsulated and affected lobuli were collapsed and fibrotic Tubercular tissue was replaced by reparative tissue. Chronic lesions of lymph nodes, ranging in size from one to 50 mm. in diameter, showed a thick fibrotic capsule around the calcified necrotic material, which could often be enucleated easily. Such necrotic tuberculous material was chemically analysed for P, Ca and Mg. Compared with the analyses of the same type of necrotic material of non-treated cattle the percentage of P, Ca and Mg was trebled in the lesions of treated animals. The 4 cases of treated chronic lobular infiltrating tubercular mastitis seen at post-mortem, revealed the replacement of the diseased tissue by reparative tissue and collapse of the alveoli.

Bacteriological studies have been completed on 41 cattle, which had received isoniazid for periods of 2-8 months. Response to treatment is shown by the following summarised results:-

(a) In 15 cases TB bacteria could not be detected in the lesions (an average of 10 smears examined per case);
(b) In 22 cases growth on solid culture media could not be obtained (an average of 13 tubes inoculated per case);
(c) In 11 cases fluid media also failed to recover viable TB bacteria (average of 10 tubes prepared per case);
(d) In 24 cases the biological test, using an average of 10 guinea-pigs per animal, was negative;
(e) In 11 cases all three methods failed to recover viable organisms from the lesions.

In vitro tests on the first 15 isolated tuberculosis strains showed that isoniazid resistant strains were already appearing after 80 days of treatment. The last fully sensitive strain was found after 110 days of dosing whereas all strains recovered after 5 months of treatment were fully resistant to the drug. The pathogenicity of these strains is being tested in guinea-pigs, rabbits and calves. The prevention of infection by chemo-prophylaxis with isoniazid has proved to be possible. Non-infected calves treated daily and exposed to natural droplet infection or oral administration of tuberculous milk did not develop lesions whereas the untreated controls became infected. However, the protective value of the drug is limited to the period of its administration. After stoppage of treatment surviving latent bacilli may yet produce lesions. It appears to be necessary to continue dosage for a while after the last exposure to give the body a chance to destroy surviving bacilli by its own defence mechanisms. In the field experiments, 110 non-infected cattle were kept in close contact with the infected herd and prophylactically treated over 8 months. Judged on quarterly tuberculin tests none was infected during the first year of the experiment.

In conclusion, it must be emphasised that the promising results obtained in our treatment of tuberculosis with isoniazid should

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not cloud certain dangers associated with the use of the drug, and lead to its "uncontrolled" use.

The interference of the drug on subsequent tuberculin tests, whether the animal has been cured or not, constitutes a serious threat, not only to the interpretation of the test, but also to the control of the disease.

It should be pointed out that abuse and fraudulent practice could result from the indiscriminate use of the drug.

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References to publications used in this paper can be obtained from the author on request.

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HAEMATOLOGICAL INVESTIGATIONS INTO THE RHIPICEPHALINE TICK TOXICOSIS SYNDROME

S. J. VAN RENSBURG

Onderstepoort

Introduction

It has long been the experience of farmers who introduce cattle into the more severely tick infested regions of the Northern Transvaal that the animals generally suffer a severe setback, particularly if introduced in summer, and it may take up to two years to regain maximum productivity. Although local adverse climatological factors play an important role, the work of Thomas and Neitz(1) indicates that the effect of tick infestation and tick-borne diseases may be underestimated. Irrefutable evidence of a potent toxin injection by *Rhipicephalus appendiculatus* (brown ear tick) has been presented by these authors. Local effects are seen as irritation, skin reaction, subcutaneous inflammatory oedema, and necrosis of regional lymphatic glands. Systemic effects take the form of pyrexia, aggravation of concurrent protozoal diseases, cachexia and death.

Invariably the most prominent change seen at post-mortem was anaemia. There was some doubt whether this anaemia could be attributed to concurrent diseases only, but the unusual persistence of babesiosis and benign gonderiosis, and the ability of G. *mutans* to cause severe anaemia in splenectomized animals were demonstrated. Impairment of the reticulo-endothelial system and bone marrow by the toxin was suggested. Thomas(2) has advanced the hypothetical significance of an aplastic anaemia caused directly by toxic damage to the bone marrow.

These investigations are an attempt to elucidate the course, severity, and nature of the anaemia that invariably occurs, and to study leucocytic changes.

Materials and Methods

Six grade Friesian oxen from the Pretoria district, which had previously only experienced light exposure to ticks and were mostly assumed to be premune to anaplasmosis and babesiosis, were used. They will be referred to in the discussion which follows as "susceptible" oxen. The survivors of heavy tick exposure a year previously, namely a group of grade Red Poll oxen, were

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used simultaneously for comparative purposes. This latter group had by no means recovered from the previous exposure and were in relatively poor condition, some showing severely stunted growth. They will be referred to as "immune" animals.

The peak seasonal activity of *R. appendiculatus* usually occurs during January and February. Both groups were introduced into a relatively heavily tick infested camp on 7.2.58, where they remained for four days, subsequently only being exposed to mild Rhipicephaline activity. The oxen were kept under observation for five months; the following data being determined at intervals of a few days:-

RBC. Erythrocytes (millions per cmm)
PCV. Packed Cell Volume (volume per cent erythrocytes)
Hb. Haemoglobin (Gm per 100 ml)
MCV. Mean Corpuscular Volume (cubic microns)
MCHC. Mean Corpuscular Haemoglobin Concentration (per cent)
WBC. Leukocytes (thousands per cmm)
Differential leucocytic count (per cent)

Estimates of the numbers of ticks on the ears were made by Dr. A. D. Thomas, the following scale being used:-

0–10 (approx. 0–1,000 and more ticks attached per ear)



Figure 1: Brown ticks attached to ears and eyelids of a bull after only three days' exposure on natural pasture.

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The haematological studies were necessarily limited to simple procedures in a field laboratory.

Smears for differential leucocytic counts were made using a wire loop and coverslip from venous blood flowing freely from a 16 G. needle inserted into the jugular vein. Haemoglobin determinations were made using a Sahli haemoglobinometer.

Treatment for babesiosis and anaplasmosis was instituted when necessary.

Large numbers of slaughter cattle showing signs of brown tick infestation were examined at an abattoir, with particular reference to the lymph nodes, adrenal glands and spleen.

Results and Discussion

Clinical Observations: Rhipicephaline activity at the time was not as severe as in previous cases described by Thomas and Neitz, hence the lower mortality. Nevertheless all the distressing clinical signs of Rhipicephaline toxicosis as described were reproduced, the susceptible animals suffering severely, while immune animals were comparatively mildly affected. During the course of the observations the oxen became mildly infested with all other ticks common to this area (*B. decoloratus, R. evertsi, A. hebraeum, H. aegyptium*).

Haematological Studies: Tabulated results from six susceptible and two immune oxen are presented. As the remaining four immune animals were all similar, their results are not quoted.

Red Blood Cell Count: *Immune Group*: In conformity with poor condition and stunting still present as a result of severe exposure a year previously, the mean red blood cell count of the immune group ranged between 5 and 5.5 million during the five months observational period. During the course of the second exposure a mild drop generally not exceeding 1 million occurred in all immune animals. A month after initial exposure, as the numbers of *R. appendiculatus* attached to the ears began to diminish, immediate and rapid recovery took place in 4 of the 6 oxen. The two exceptions both developed mild A. *marginale* relapses during the second month after exposure, the count of 3.8 in one being the lowest recorded in the immune group. With the onset of winter and deterioration in the quality of the grazing, the whole group showed a gradual mild decline in the red blood cell count. Moderate numbers of the erythrocytic forms of *G. mutans* appeared at variable intervals.

Susceptible Group: The onset of anaemia in the susceptible group was more severe. Within a month the mean count had reached 3.2 million, and remained between 3–4 million for the following four months. All six oxen developed *B. bigemina* reactions within the first month, the three most markedly infested with ticks showing the severest reactions. The latter three all developed haemoglobinaemia and counts of less than 2 million,

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one dying and on post-mortem showing in addition *R. ruminantium* infection. Two oxen not as heavily infested with ticks showed only mild *B. bigemina* reactions, the red cell count however declining gradually to the vicinity of two million. The lowest counts coincided in one ox with two *A. marginale* relapses, while the other showed particularly low counts during three *G. mutans* reactions at monthly intervals. All ticks were removed after 12 days from the remaining ox, whereupon prompt partial recovery, similar to that seen in the immune group, occurred. Babesiosis and benign gonderiosis reactions were particularly mild, the red cell count only reaching a low level three months later, following a severe *A. marginale* reaction. All the survivors developed anaplasmosis towards the end of the observational period.

The multiplicity of erythropenia promoting factors in operation decidedly complicate the interpretation of their relative significance. It is clear however that the following are involved:-

(1) *Intravascular haemolysis and/or erythrorrhexis.* In all animals every significant decline in the RBC was correlated with reactions of babesiosis, anaplasmosis and benign gonderiosis, singly or in combination. Generally the severity and persistence of particularly babesiosis and benign gonderiosis were directly proportional to the degree of tick infestation.

(2) *Extravascular blood loss.* The volume of blood removed by the vast numbers of ticks is considerable, and may be judged by the drop of RBC in the immune animals. Prolonged haemorrhage occurred in most oxen from tissues damaged by trauma resulting from irritation caused by tick attachment.

(3) *Hypoplasia of bone marrow.* Initial anorexia and a subsequent low level of nutrition during the winter months are probably important factors concerned in retarding erythrogenesis. One ox stabled and fed on a high protein diet showed much improvement in condition, but red cell counts were only slightly higher than similarly affected animals in the group. This ox, however, showed greater resistance to an anaplasmosis reaction.

Particularly low erythrocytic counts, coincidental with concurrent protozoal diseases, were invariably accompanied by the appearance of all forms of immature erythrocytes in the blood, excluding the possibility of aplasia of the bone marrow.

The sustained normocytic anaemia exhibited by the "immune" group would indicate a hypoplastic condition of the bone marrow. Whether this is due to a residual specific effect of the tick toxin or merely secondary to a general debility is not known.

Packed Cell Volume, Mean Cell Volume, Haemoglobin and Mean Cell Haemoglobin Concentration: From the tables it will be seen that the usual packed cell volume found in the susceptible group during observation was in the region of 18%, while values as low as 11% were recorded. In the immune animals it remained

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rather constant in the vicinity of 24%, the lowest recorded value being 20%.

Normal mean corpuscular volume values found in this area are not as high as those quoted for Great Britain, but appear to be similar to those found by Schalm([3]) in California (mean 45.5 cubic microns). A negative correlation between mean corpuscular volume and red cell count was generally present. While the immune group showed normocytic or only slightly macrocytic cells, the susceptible animals commonly yielded high MCV values. Macrocytosis was markedly aggravated during severe phases of anaemia. These transient macrocytic phases indicate intensification of erythrogenesis and regenerative changes in the bone marrow, and evidently preclude depression of erythrogenesis as the major factor in the anaemia. From microscopic examination it could be seen that numbers of microcytes did, however, appear among the predominating macrocytes during the terminal observations, probably a result of iron deficiency following prolonged parasitic blood loss and poor nutrition.

Haemoglobin results obtained with the simple field haemoglobinometer are considered too inaccurate for detailed interpretation, and therefore are not quoted in the tables. Mean corpuscular haemoglobin concentration determinations however generally indicated a mild hypochromic state in the susceptible group (particularly during low RBC excursions), while immune animals were usually normochromic.

Morphologically the susceptible group may be said to have suffered initially from a transient macrocytic, hypochromic anaemia, while the immune group showed a mild chronic normocytic, normochromic anaemia. The former is no doubt predominantly a result of the severity of concurrent tick-borne diseases, while the debilitating and retarding effects of tick toxin and chronic infections([4], [5]) secondary to severe tick infestations, must be considered in the latter instance.

White Blood Cell Count: A constant mild leucocytosis was present in all the "immune" animals throughout the observations, mean values only varying between 12–14,000 and no noteworthy changes occurring even during severe tick infestation. All severely infested susceptible animals developed leucopenia, which persisted for one to two months, during the climax of *R. appendiculatus* activity, in spite of secondary protozoal and bacterial infections. Less infested individuals showed a more irregular leucocytosis than the immune group. Cattle exposed to even relatively mild tick infestations are commonly found in this area to be showing a leucocytosis.

Differential leucocytic count:

Neutrophiles: After initial increases in susceptible animals, a progressive decline in the relative number of neutrophiles occurred

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and persisted during the period of observation. Sharp regenerative shifts to the left were repeatedly recorded during increased *G. mutans* activity. Removal of ticks from No. 8100 was followed by a temporary neutrophilia, this individual being the only one to show constant values approaching normality during the terminal period of observation. Figures from members of the immune group were less inclined to fluctuate, but generally remained below normal with a constant mean in the region of 20%.

Eosinophiles: The number of circulating eosinophiles in all susceptible animals declined rapidly after exposure to ticks, until they appeared to be virtually absent in most animals. Subsequently they reappeared in small numbers, only the more severely infested individuals developing some degree of eosinophilia later.

In contradistinction, the immune group showed a constant high level, individual values varying between 5%–30% and the mean remaining above 10%. The very prolonged and constant eosinophilia appears to eliminate the possibility of this manifestation being an allergic response to parasitism.

Basophiles: As in many disease conditions, these cells were largely absent from all animals.

It is notable that all leucocytes of the granulocytic series were depressed in susceptible animals, indicating some possible hypoplastic changes in the bone marrow.

Lymphocytes: Initial mild relative decreases resulted from transient neutrophilia and monocytosis in susceptible oxen, the position thereafter being reversed and unusually high lymphocytic levels, the mean varying between 70–80%, being maintained in all animals. Immune animals were inclined to show similar but milder changes, the subsequent mean lymphocytic values varying between 60–70%.

Monocytes: Transient monocytosis during the phase of brown tick attachment and appearance of concurrent diseases, was soon followed by the usual normal levels.

Morphology of leucocytes: The presence of relatively large numbers of immature leucocytes was a salient feature throughout the observations. Neutrophilic myelocytes, metamyelocytes and band forms were observed in all animals in variable numbers. In the susceptible group hyperlobulation of the neutrophile nucleus was conspicuous, particularly during the terminal observational period, indicating a possible need for retention of these cells longer than is usual. Only the eosinophiles of immune oxen were inclined to show hyperlobulation of nuclei. Lymphocytes tended to exhibit increased basophilia, while large lymphocytes predominated over small. Pleomorphism of lymphocytic nuclei was frequent in all animals.

Plasma Proteins: Estimations of plasma proteins in the susceptible animals immediately after the majority of ticks had become detached, revealed normal total protein levels, while globulins were

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significantly increased. A month later globulin levels were still elevated, while the advancement of cachexia subsequent to the detachment of ticks, apparently characteristic of the syndrome, was typified by unusually low total protein levels.

Endocrine Function: Distinct evidence of endocrine malfunction has repeatedly been observed in cattle that have been severely infested with the brown ear tick. During the phase of acute tick infestation, the increased neutrophiles and decreased number of lymphocytes and particularly of eosinophiles, may be interpreted as being due to increased secretion by the adrenal cortex in response to stress([8]). As seen in the attached tables, this position is reversed subsequently, high eosinophile and lymphocytic counts with depressed neutrophiles being a constant finding in the oxen exposed a year previously, possibly indicating an unduly sluggish pituitary adrenal complex([3], [7]).

The adrenal glands of a large number of cattle showing signs of *R. appendiculatus* infestation have been examined at slaughter, with constant findings. Cattle showing signs of acute infestation, i.e. large numbers of ticks without hyperkeratosis and fibrosis of the ears, invariably showed significantly enlarged adrenal glands (figure 2), the cortex being the usual golden brown colour, but, in addition, showing diffuse to irregular areas of congestion, most constantly seen along the cortico-medullary junction. Circumscribed light brown dull areas were occasionally seen in the cortex.

Animals having suffered from severe chronic infestations, i.e. exhibiting hyperkeratosis and fibrosis of the ears, had mildly undersized adrenal glands. On section the consistency of these glands was noticeably increased. The cortex was pale grey to brown in colour, and numerous light coloured strands up to 1 mm. in width were seen extending radially from the medulla to the periphery. Unlike normal cortical trabeculae, which are usually only seen with difficulty, these were particularly prominent in the region of the *Zona reticularis*. Histologically these glands showed prominent cords of collagenous tissue radiating from the medulla, while irregular deposition of fibroblastic tissue, chiefly in the *Z. reticularis*, caused some disorganisation of cortical structure.

Particularly in acutely infested animals, large numbers of cortical cells were seen apparently undergoing necrobiosis. Some nuclei were hyperchromic with crenated nuclear membranes while others were small and pyknotic. Some showed exceptionally pale staining nuclei. Similar cells were seen in the normal adrenal in small numbers, and are regarded by some as degenerating cells. In animals infested with ticks, these degenerative cells tended to occur in groups or strands, and were usually associated with active fibroblastic activity, and congested sinusoids, this process presumably resulting in induration of the cortex. The extent to which these changes may be attributed to severe non-specific

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hyperfunction of the adrenal cortex and/or the direct effects of an assumed tick toxin, is not clear.

Permanently stunted growth, following severe exposure to ticks during the first 18 months of life, has been observed repeatedly. Impairment of normal growth hormone secretion from the hypophysis may be implicated. Further evidence of hypophyseal hypofunction, such as inactivity of the ovaries, is occasionally seen in chronically infested cows.

In the evaluation of any endocrine hypofunction, it must be remembered that certain endocrine glands (pituitary, thyroid, adrenal and gonads) influence the maturation and/or regulation of the number of blood cells, both erythrocytes and leucocytes([3]). Bovines have been contended to exhibit a relative deficiency in the pituitary adrenal complex([3], [7]). Further impairment would therefore appear to lead to reduced efficiency of stress response and hypofunction of the reticulo-endothelial system. It is suggested that this mechanism is concerned in the aggravation of concurrent diseases and the persistent relapses observed following severe *R. appendiculatus* infestation.

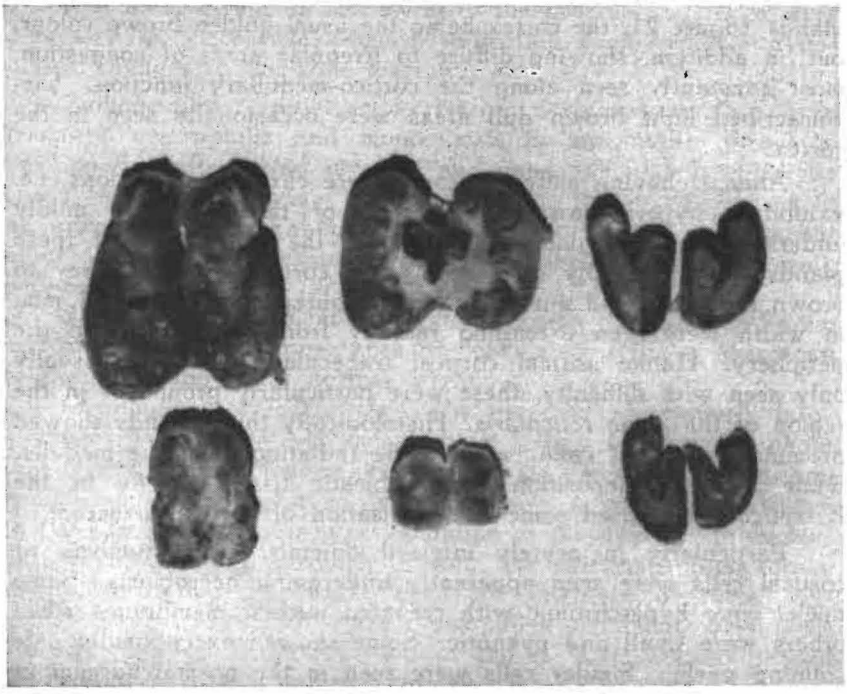


Figure 2: Adrenal glands, inguinal and subparotid lymph nodes of an infested ox (below) in comparison with those of a normal ox of same size (above).

Pathology of lymph nodes: During the examination of the adrenal glands in slaughter cattle mentioned above, a study of

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the lymph nodes was made simultaneously. A close correlation between the size and consistency of the adrenal glands and lymph nodes was apparent.

A mild general enlargement of nodes was present. Regional nodes draining infested parts of the skin were however grossly enlarged while numbers of ticks were present, subsequently becoming smaller and indurated. On section the acutely enlarged glands presented an uneven surface (figure 2), pale grey areas alternating with irregularly defined reddened areas. A patchy greenish discoloration was evident. Chronically infested animals invariably showed firm subparotid nodes, which had an abundance of collagenous stroma throughout the substance. Abscesses were not found infrequently.

Histologically the regional nodes of acutely infested animals invariably showed groups of necrobiotic cells in the cortical tissue, some of which appeared to be undergoing necrosis with karyorrhexis. These areas were generally associated with haemorrhagic infiltration and the presence of large numbers of macrophages, many of which had their cytoplasm distended with yellow to brown pigment. A few to massive numbers of eosinophiles were present, often closely associated with active fibroblastic activity. The cortical tissue adjacent to the lymphatic sinuses and the reticular stroma of sinuses were infiltrated with macrophages, erythrocytes and eosinophiles. Neutrophiles were present in variable numbers, focal purulent processes arising in the region of sinuses occasionally being seen. Bacteria were usually readily demonstrated in smears from nodes showing focal purulent processes without macroscopic abscess formation.

The germinal centres, particularly of chronically infested individuals, generally had a "washed out" appearance and contained few large lymphocytes, the reticular stroma being prominent. The centres were well defined by the presence of a dense layer of small lymphocytes and delicate collagenous stroma around the periphery. Congestion, necrosis and deposition of collagenous tissue within the germinal centres occurred infrequently.

All the changes described were observed in animals where *G. mutans* could not be demonstrated by examination of blood, spleen and lymph node smears.

Diagnosis of Rhipicephaline toxicosis: Little doubt exists that intoxication by the secretions of *R. appendiculatus* is of considerable significance. Death, due to intoxication only, has not been observed and in all probability does not occur, but mortality attributable to aggravation of concurrent diseases, not only protozoal, has been observed not infrequently in the field. Chronic unthriftiness and reduced milk production following severe infestation is possibly of paramount economic significance. A complex problem of neglect and poor management by stock owners is frequently present.

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In practice it appears that calves, and adult cattle, that are moved from one locality to another, are most inclined to suffer, presumably due to the absence of a specific immunity that may be built up by repeated small exposures. High producing dairy stock, well known to respond poorly to stress, appear to be more adversely affected than the hardy beef breeds. However, on one farm running beef cattle, tick control measures were overlooked in one group of animals, resulting in a heavy infestation in this one group. Weeks later this group was noticed to be unthrifty and it was found that their mean red cell count was 48% lower and haemoglobin values 40% lower than the other animals on the same farm.

Clinically, this condition can be suspected on a herd basis only, as individual animals may be suffering from some obscure debilitating disorder. Apart from fibrosis and hyperkeratosis of the ears, chronically infested cattle, on slaughter, most constantly exhibit a moderate to severe atrophy of the spleen, together with the adrenal gland and lymph node changes described. These lesions have been found to be surprisingly constant and are also seen at slaughter in stock in good condition, i.e. in animals which have either recovered or acquired some degree of immunity previous to severe exposure, but nevertheless showing external signs of severe infestation. These changes have not been found in a large number of cattle examined at slaughter which showed no signs of moderate to severe brown tick infestation. There is however no certainty that other species of ticks do not also produce toxins which may contribute to the syndrome, as tick infestation is commonly mixed. Localised necrosis following Hyalomma infestation is well known, while the writer has repeatedly observed pyrexia and apathy in calves suffering from a massive infestation of *Boophilis decoloratus*. Limited observations in slaughtered cattle indicate that *Rhipicephalus evertsi* may be responsible for haemorrhagic changes and enlargement of nodes draining infested regions of the skin.

Experimental Attachment of the Brown Tick on Man

From observations on cattle it was obvious that at the sites of attachment of the ticks a local irritation was caused and symptoms suggestive of pain were evident at times. In an attempt to gain a better understanding of these symptoms, a single brown tick was induced to attach to the forearm of two persons, one male and one female tick being used. Well developed and active specimens were selected from unengorged veld ticks. Engorgement for approximately thiry hours was allowed. The local and unexpected systemic effects experienced were so similar that an individual description is not warranted.

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A sharp focal pain during penetration of the skin, of about fifteen minutes duration, was felt. This was soon followed by intense local pruritus and the appearance of a small papule and surrounding reddening of the skin about two hours after attachment commenced. The area of pruritus gradually increased, spreading in a proximal direction from the site of attachment. Ten hours later deep muscular pain was present in the forearm and the inner aspect of the upper arm, the axillary lymph node also becoming sensitive. While the ticks were still present, the papule increased in size, becoming yellow to white in colour. Intense pruritus and mild muscular weakness continued. After detachment, the pruritus very soon ceased, the papule however remaining for many weeks before disappearing. A very similar pattern of mild but distinct systemic effects was experienced by both subjects, symptoms being more pronounced in the host of the female tick. Pyrexia, reaching a maximum of 102°F in one subject, was present. Tachycardia, lassitude and mental depression were experienced during the last twenty-four hours, as well as mild generalised muscular pain and a slight sensitivity of most lymph nodes.

It is not suggested that this condition may occur naturally in man, as the pronounced local irritation will make the presence of the parasite obvious and lead to its prompt removal.

Conclusions

1. "Susceptible" animals suffering severe infestations of *R. appendiculatus* usually develop an acute transient macrocytic, hypochromic anaemia, which after a period of months reverts to a mild chronic normocytic, normochromic anaemia. The severity of the initial acute anaemia is directly dependent on the severity of concurrent diseases known to cause haemolysis or erythrorrhexis. The concurrent diseases may be new infections or relapses. Fatalities may occur at the most acute stage of anaemia.

2. In no instance could an acute anaemia be ascribed to the direct effects of a presumed tick toxin only. Active erythrogenesis was present during all severe phases of anaemia.

3. A second severe exposure in "immune" animals results in only a mild drop in the red cell count, probably exclusively due to direct parasitic removal of blood. Occasionally mild relapses of benign gonderiosis and anaplasmosis have been observed. A high degree of immunity was apparently present.

4. The degree of brown ear tick infestation appears to determine the severity of all concurrent diseases, and consequently is proportional to the degree of anaemia that is present. For profound acute symptoms and death to occur, it appears necessary for the ears to be so grossly infested that it is virtually impossible for further ticks to attach. It is estimated that a total of 2–3,000 ticks per head would be implicated in the latter instance.

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5. Impairment of the reticulo-endothelial system is evidenced by poor response to the stress of concurrent diseases and relapses, this manifestation possibly resulting from "exhaustion" of the system or sluggish stimulation by the adrenal cortex. Distinct evidence of direct cytotoxic impairment is lacking.

6. It appears that *Gonderia mutans* may be responsible for causing repeated significant declines in the red cell count.

7. The acute syndrome may be followed by a chronic debility extending into months or years, or becoming permanent. This debility appears to be accompanied or caused by long standing damage or impairment of some specialised tissues. Some evidence of the latter changes has been presented, whereby the adrenal glands, pituitary, lymph nodes, spleen, and bone marrow have been implicated. The chronic normocytic anaemia described may be due to a hypoplastic change in the bone marrow.

8. Initial leucopenia followed by leucocytosis and the consistent presence of immature leucocytes in the blood indicates an excessive utilisation of these cells. The many aspects of the syndrome most certainly demand increased antibody formation, phagocytosis, and participation in healing processes.

9. The regularity with which lymph node changes were observed, and the local and systemic effects described in man, leave little doubt that a potent cytotoxic substance is injected by the brown tick. The effect of such a toxin may be expected to depend on the amount present.

Summary

The survivors of a group of animals exposed the year before, to a heavy infestation of *R. appendiculatus*, were subjected to a second severe exposure together with a group that had not previously been exposed. The course of anaemia, concurrent diseases and degree of tick infestation and leucocytic changes were studied.

Some field observations on the significance of tick toxicosis in parts of the Northern Transvaal and Natal Midlands are recorded. A study of the internal organs of a large number of slaughtered cattle showing external signs of infestation was made, with particular reference to the lymph nodes and adrenal glands.

Acknowledgments

This work was initiated under the direction of Dr. A. D. Thomas.

Dr. J. M. M. Brown kindly performed plasma protein determinations and made useful suggestions.

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No. 8077 ("susceptible")

Date	RBC	PCV	MCV	WBC	Lym	Neu	Eos	Mon	Bas	Remarks
21.2.58	4.9	24	49	7.1	55	26	6	13	0	
26.2.	4.3	23	53	4.4	65	23	4	9	0	
4.3.	3.8	19	50	6.4	55	24	5	16	0	G. mutans
10.3.	2.1	13	62	6.0	64	16	2	18	0	B. bigemina
14.3.	1.8	11	61	7.8	68	13	2	16	0	G. mutans
19.3.	2.8	19	68	6.3	67	15	8	9	1	
24.3.	3.0	20	67	6.9	70	15	7	8	0	
31.3.	3.4	21	62	8.6	72	14	10	4	0	
7.4.	3.7	21	57	8.4	67	18	11	4	0	B. bigemina
11.4.	4.1	20	49	7.5	67	15	14	4	0	
23.4.	4.6	23	50	8.1	76	12	7	5	0	
30.4.	4.2	21	50	9.3	67	22	7	4	0	G. mutans
7.5.	4.4	20	45	11.4	85	9	4	2	0	
14.5.	4.5	22	49	10.2	71	21	5	3	0	
21.5.	4.0	21	53	6.6	72	22	6	1	0	G. mutans
28.5.	3.9	19	49	8.3	77	20	2	1	0	B. bigemina
4.6.	2.4	16	67	7.5	77	19	2	2	0	A. marginale
11.6.	1.6	14	87	8.4	80	15	3	2	0	
24.6.	3.2	17	53	7.4	75	14	7	4	0	

Tick estimation	Date	13.2	15.2	18.2	21.2	24.2	28.2	5.3	11.3	21.3
	Scale No.	5	6	8	8	10	9	6	3	1

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(2)

No. 8088 ("susceptible")

Date	RBC	PCV	MCV	WBC	Lym	Neu	Eos	Mon	Bas	Remarks
18.2.58	5.9				50	28	14	8	0	
21.2.		25		10.0	44	31	12	13	0	
26.2.	5.0	21	43	7.3	61	19	1	18	1	
4.3.	3.3	15	45	8.6	51	32	1	16	0	B. bigemina
10.3.	2.2	13	59	5.1	64	27	0	9	0	G. mutans
14.3.	2.1	13	62	5.0	73	15	0	12	0	
19.3.	1.6	11	69	9.6	65	24	0	11	0	B. bigemina
24.3.	2.1	14	69	6.6	65	29	0	6	0	
31.3.	2.8	18	64	6.8	70	23	3	4	0	
7.4.	3.0	17	57	7.0	77	12	7	4	0	
11.4.	3.0	18	60	6.3	73	16	9	2	0	B. bigemina
23.4.	3.9	20	51	8.1	78	11	9	2	0	Stabled and fed
30.4.	4.9	24	49	12.6	72	16	10	2	0	lucerne hay
7.5.	4.7	21	45	10.1	79	13	3	5	0	
14.5.	4.6	22	48	10.6	82	11	4	3	0	
21.5.	4.1	20	49	10.6	79	14	4	0	0	A. marginale
28.5.	3.6	17	47	14.4	75	19	4	2	0	
4.6.	4.4	22	50	10.4	80	17	0	3	0	
11.6.	5.2	28	54	13.0	77	5	17	1	0	
24.6.	5.5	29	53	16.7	70	17	9	3	1	G. mutans

Tick estimation	Date	13.2	15.2	18.2	21.2	24.2	28.2	5.3	11.3	21.3
	Scale No.	7	8	8	10	10	8	3	4	1

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(3)

No. 8082 ("susceptible")

Date	RBC	PCV	MCV	WBC	Lym	Neu	Eos	Mon	Bas	Remarks
17.2.58	4.8				53	22	20	5	0	
21.2.	4.6	20	44	6.3	42	26	13	18	1	
26.2.	3.9	20	51	5.3	67	21	4	8	0	
4.3.	2.7	13	48	10.7	39	31	0	26	0	G. mutans
7.3.	2.1	13	62	12.0	38	45	0	17	0	B. bigemina
8.3.										DIED R. ruminantium

Tick estimation	Date	13.2	15.2	18.2	21.2	24.2	28.2	5.3
	Scale No.	6	7	10	10	8	8	4

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(4)

No. 8104 ("susceptible")

Date	RBC	PCV	MCV	WBC	Lym	Neu	Eos	Mon	Bas	Remarks
17.2.58	5.4				58	29	3	10	0	
21.2.		27		13.0	39	50	3	9	0	
26.2.	5.5	23	42	12.5	58	39	0	12	0	
4.3.	4.2	19	45	9.2	38	34	0	28	0	
10.3.	4.4	19	43	14.2	46	27	1	15	0	B. bigemina
14.3.	4.0	20	50	8.0	53	26	0	21	0	
19.3.	3.6	18	51	13.7	62	21	0	17	0	
24.3.	2.9	16	55	13.0	79	16	0	5	0	
31.3.	2.1	14	66	16.9	69	23	5	3	0	G. mutans
7.4.	2.7	18	67	16.0	73	17	7	3	0	
11.4.	2.7	17	63	12.7	79	12	7	2	0	B. bigemina
23.4.	3.2	18	56	11.2	81	9	5	5	0	
30.4.	2.0	16	80	12.6	62	35	2	1	0	G. mutans
7.5.	2.6	15	58	10.0	67	25	7	1	0	
14.5.	3.3	17	52	8.4	80	16	1	3	0	
21.5.	2.9	15	52	6.4	76	20	0	4	0	
28.5.	2.8	14	50	10.4	69	31	0	0	0	G. mutans
4.6.	3.4	17	50	11.6	65	29	4	2	0	
11.6.	3.3	17	52	9.4	75	20	2	3	0	A. marginale
24.6.	1.9	11	58	12.5	76	19	2	3	0	B. bigemina
16.9.	3.2	15	47	9.7	70	26	0	4	0	Kept on natural pasture only.
23.9.										DIED. Poverty.

Tick estimation	Date	13.2	15.2	18.2	21.2	24.2	28.2	5.3	11.3	21.3
	Scale No.	6	6	6	8	8	8	6	5	1

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(5)

No. 7990 ("susceptible")

Date	RBC	PCV	MCV	WBC	Lym	Neu	Eos	Mon	Bas	Remarks
17.2.58	5.5				70	15	6	8	1	
21.2.		26		7.0	65	19	4	12	0	
26.2.	4.7	26	55	8.1	63	19	2	15	1	
4.3.	4.9	27	55	8.2	58	31	1	10	0	
10.3.	3.5	17	49	7.7	57	25	0	18	0	B. bigemina
14.3.	3.7	19	51	9.7	56	25	0	19	0	
19.3.	3.1	18	58	12.7	63	20	2	15	0	G. mutans
24.3.	3.2	18	56	10.1	64	27	1	8	0	
31.3.	3.0	19	63	11.0	79	14	3	4	0	
7.4.	2.9	17	66	9.4	83	13	1	3	0	
11.4.	2.5	17	68	8.3	79	17	0	4	0	A. marginale
23.4.	2.7	19	70	7.8	79	16	0	5	0	B. bigemina
30.4.	3.4	20	59	6.5	75	22	2	1	0	
7.5.	3.7	20	54	8.0	80	17	1	2	0	
14.5.	4.4	21	48	6.9	77	20	0	3	0	
21.5.	3.7	18	49	6.7	79	14	3	4	0	
28.5.	3.8	21	54	6.4	71	20	5	4	0	
4.6.	3.6	18	50	8.6	75	20	3	2	0	A. marginale
11.6.	3.9	21	54	8.0	76	17	3	4	0	B. bigemina
24.6.	3.9	20	51	9.4	74	17	4	5	0	

Tick estimation	Date	13.2	15.2	18.2	21.2	24.2	28.2	5.3	11.3
	Scale No.	3	5	4	6	6	4	3	1

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(6)

No. 8100 ("susceptible")

Date	RBC	PCV	MCV	WBC	Lym	Neu	Eos	Mon	Bas	Remarks
21.2.58	4.8	24	50	9.6	49	25	7	19	0	ALL TICKS
26.2.	4.4	23	52	10.3	49	29	0	22	0	REMOVED
4.3.	4.7	24	51	13.5	43	49	0	8	0	
10.3.	4.9	22	45	9.7	60	25	0	15	0	B. bigemina
14.3.	5.3	26	49	9.2	53	27	0	10	0	G. mutans
19.3.	4.9	25	50	8.2	68	18	1	13	0	
24.3.	4.9	22	45	9.7	64	23	5	8	0	
31.3.	4.8	23	48	14.3	77	17	2	5	0	B. bigemina
7.4.	5.7	23	40	11.1	80	10	6	4	0	
11.4.	4.7	22	47	15.1	72	17	6	5	0	
23.4.	4.6	22	48	8.8	83	12	1	4	0	
30.4.	4.7	19	40	11.1	76	17	4	3	0	G. mutans
7.5.	3.5	16	46	10.6	77	18	2	3	0	A. marginale
14.5.	2.9	16	55	8.8	73	18	6	3	0	
21.5.	2.8	17	61	6.0	81	12	1	6	0	B. bigemina
28.5.	2.4	15	63	10.8	74	19	2	5	0	G. mutans
4.6.	2.8	16	57	9.8	80	16	3	1	0	
11.6.	2.5	18	72	8.4	82	15	1	2	0	
24.6.	2.9	15	51	8.6	64	27	2	7	0	A. marginale

Tick estimation	Date	13.2	15.2	18.2	24.2	28.2	5.3	11.3
	Scale No.	3	4	5	0	0	0	0

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(7)

No. 6272 ("immune")

Date	RBC	PCV	MCV	WBC	Lym	Neu	Eos	Mon	Bas	Remarks
25.2.58	6.2	26	42	14.9	60	19	11	10	0	
7.3.	5.0	27	54	13.6	59	43	7	10	1	G. mutans
17.3.	5.7	27	47	19.4	39	35	11	15	0	
27.3.	6.8	27	40	14.4	63	23	8	7	0	
9.4.	6.5	28	43	13.0	71	17	6	6	0	
24.4.	5.8	27	47	11.6	78	13	6	3	0	G. mutans
5.5.	6.0	27	45	12.6	74	14	8	4	0	
20.5.	5.8	27	47	9.9	80	14	4	2	0	
3.6.	5.5	24	44	10.1	63	24	10	3	0	
19.6.	6.0	26	43	15.6	55	35	9	1	0	
3.7.	5.4	25	46	15.0	54	37	6	3	0	G. mutans

Tick estimation	Date	14.2	18.2	21.2	24.2	28.2	5.3	11.3
	Scale No.	4	6	6	6	4	4	2

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(8)

No. 7091 ("immune")

Date	RBC	PCV	MCV	WBC	Lym	Neu	Eos	Mon	Bas	Remarks
19.2.58	5.3	30	57							
25.2.	5.0	24	48	10.9	62	12	10	16	0	
7.3.	4.8	25	52	13.1	47	24	18	11	0	
17.3.	4.7	22	47	12.4	42	17	20	20	1	
27.3.	4.4	22	50	14.8	56	18	23	3	0	G. mutans
9.4.	3.8	21	55	13.2	58	10	30	2	0	A. marginale
24.4.	4.1	23	56	12.9	70	12	15	3	0	
5.5.	5.0	25	50	12.2	60	13	25	2	0	G. mutans
20.5.	4.8	23	48	19.6	50	35	12	3	0	
3.6.	4.5	23	51	14.2	56	24	16	3	1	
19.6.	4.4	22	50	14.6	60	14	23	3	0	
3.7.	4.8	22	46	11.5	72	11	15	2	0	G. mutans

Tick estimation	Date	18.2	21.2	24.2	28.2	5.3	11.3	21.3
	Scale No.	1	2	3	4	5	3	1

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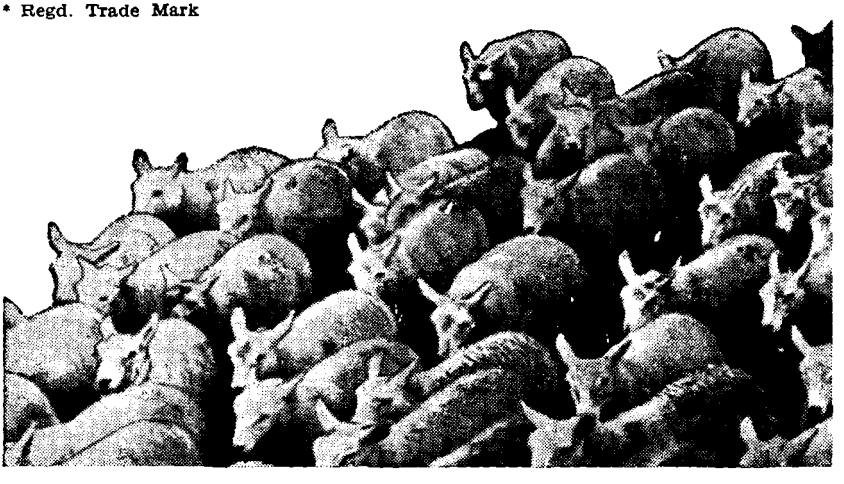
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1. Aust. Vet. Jl. 1954, 30:209.

2. Vet. Rec. 1955, 67:74.

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ADVANCES IN "GEELDIKKOP" (ULOSIS OVIS) RESEARCH

1. THE HISTORY OF "GEELDIKKOP" RESEARCH

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The classical "Geeldikkop" syndrome is a disease of small ruminant animals occurring in most parts of the Karroo, the North-west Cape and Southern Free State. It is characterised by a severe photosensitization, in most cases a very severe type of icterus, best classified as an intrahepatic cholestasis, severe renal lesions, uraemia, blood electrolyte disturbances and certain blood cell dyscrasias.

The disease is responsible for extremely heavy annual loss to the sheep and wool industries of this land, due not only to the high mortality rate, but also to loss of condition, wool production and reproductive abilities after recovery of surviving cases. The causal agent of the disease has for long been considered to be the well known plant *Tribulus terrestris* L. (Zygophillaceae), the "Devil's-thorn" or "Dubbeltjie".

The history of the work on this disease makes fascinating reading, for some of the greatest figures in South African veterinary science have at different times been associated with it. The first official record of the disease is that of Hutcheon(1), the Chief Colonial Veterinary Surgeon for the Cape of Good Hope, in 1886. It is certain that the disease was well known to farmers in the affected areas long before this time under the names of "Dikgeel" or "Geeldikkop", and it was believed to occur amongst sheep throughout the Cape Colony.

Hutcheon thought the disease to be a primary derangement of the liver and possibly of malarial origin. In 1894, one of his colleagues, Dixon(2) described an outbreak of the disease amongst sheep and goats in the Khoup, and suspected that it might be due to *Tribulus terrestris* or the larvae of an insect parasite found in the stalks of the plant.

In the following year Dixon(6) carried out feeding experiments with Tribulus, the results of which proved to be negative;

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he then concluded that the plant was not the cause of the disease. This view was supported by the fact that in the early winter of 1895, at a time when frosts appeared to have destroyed all the Tribulus, Geeldikkop appeared amongst sheep in the Beaufort West district. These sheep had been running on the high plateau and hills of the Nieuweveld and had contracted the disease on being brought down into the flats around Beaufort West. These observations are of note, since here for the first time appears an account of the syndrome later to be designated "Trek-dikkop" or "Lugdikkop".

The symptoms, postmortem findings and occurrence of the disease were then described in great detail by Hutcheon([1], [3]) and Dixon([4], [5]). Dixon also at this time performed numerous inoculation experiments using body fluids and organ pulp emulsions from affected animals, all with negative results.

In two further reports, Hutcheon([7], [8]) reviewed the data collected on Geeldikkop and referred to work done by one of his colleagues, Paine, who also described various aspects of the disease at length, contending that Tribulus was not the agent responsible but held that the disease was of a malarial nature and that a winged insect acted as intermediate host for an infectious agent. Following later inoculation experiments he was unable to support these theories. His contributions appeared in print in 1904 and 1906([9], [10]).

In a report for the year 1905, one of Hutcheon's assistants, Elley, reported his own experiences with the disease in the Beaufort West district([11]), noting the tidal appearance of the disease and the beneficial effects of a change of grazing during outbreaks of the disease. He also inclined to the belief that Tribulus was not responsible for the occurrence of Geeldikkop. In a further report([12]) he expressed the opinion that the oedema was the primary lesion.

Attempts to transmit the disease by inoculation of body fluids from diseased animals were recorded by Spreull([13]) and further unsuccessful dosing experiments using Tribulus by Dixon([14]).

For a while no further reports of work on this disease seem to have appeared. In the years 1915, 1916 and 1917 the disease broke out on an extremely severe scale, practically throughout the entire Karroo, North-western Cape and Southern Free State. The matter was raised in Parliament and requests were made for urgent investigations into the disease.

The work was now taken up by Theiler and his staff. The preliminary enquiries were conducted for him by the late Dr. Walker in the Donkerpoort, Luckhoff and Middelburg areas. In 1917 Theiler himself in company with Dr. I. B. Pole-Evans, then Chief of the Division of Botany, carried out further investigations. The record of this work, which is one of the most comprehensive accounts of the disease extant appeared in 1918([15]). A mass of

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information was collected from farmers in these areas and carefully sifted, and then from this and the earlier investigations he concluded: (a) "The disease may be of malarial origin, notwithstanding the absence of visible parasites in the blood, and its non-inoculability". He did not however consider this possibility very likely; and (b) "That the disease was of vegetable origin". This was considered the most likely cause of the disease. Accordingly a large number of experiments were carried out both at Onderstepoort and in the affected areas, to investigate the two possibilities mentioned. All these experiments are faithfully recorded in his report([15]).

He was unable to produce the disease in healthy sheep by intravenous injection or oral administration of body fluids, organ emulsions or ingesta, nor was he able to produce it by close contact of ailing and healthy animals. Experiments performed at Onderstepoort by feeding to sheep Tribulus emanating from farms where the disease was present, were similarly negative. Various grazing experiments were then conducted on the farm "Bergrivier", in the Luckhoff area. The results of these appear at a first glance conflicting, yet they are highly significant.

In one of these experiments, thirty-five sheep were allowed to graze freely on a pasture consisting of Tribulus, *Pentzia virgata* and various grasses. None of these sheep developed symptoms of the disease. In a further experiment, twenty sheep were confined to a kraal which had been denuded of all vegetation, and were fed Tribulus *ad libitum*, which had been freshly collected from the veld. Ten days after commencement of this experiment the first cases of the disease appeared, and shortly sixty per cent of the animals used had contracted the disease. The Tribulus which was used in this work was at first in a wilted state, but later, at the time the disease was produced, was growing luxuriantly and in full flower.

From these results he concluded that Tribulus was the agent responsible for the disease, but apparently only when it was eaten exclusively, or at least in large quantities. In three following series of feeding experiments, based on his successful one, he was then unable to reproduce his results. He attributed these negative findings to some undefined change which must have occurred in the conditions favourable for the evolution of "Geeldikkop", affecting either the plant itself or in the weather.

It is of considerable interest to note at this stage, during the course of these experiments he observed the growth of a fungus, described as "a new species of Calletotridium", on the Tribulus plants. It appeared to be influencing the growth of these plants to a considerable degree, and he considered the possibility of this fungus thus modifying the toxicity of the plant. Recent developments to be discussed later in this paper show that this view of Theiler's deserves reconsideration He was. in this work,

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also able to disprove the popular theory that the larval parasite present in the stems of some of the plants was responsible for the disease.

From these experiments he concluded: "that Tribulus is the exclusive cause of Geeldikkop and this only in a particular stage of growth, the flowering one".

In the same report the pathology, symptomatology, treatment and control of the disease were discussed at length. An interesting fact which emerges is that the pathological changes described by him are at variance with those mentioned by later workers and those found in the recent investigations. For instance, he found as constant lesions in the liver, cirrhotic changes, proliferation of the bile ducts and in some cases necrosis of the parenchymal cells. Recent studies of the liver pathology of the disease indicate that this organ is innocent of such changes and, with the exception of severe bile pigmentation and mild fatty infiltration in some cells only, the liver parenchyma and supporting tissues appear unaffected.

It was only some while after this that it was realised that there occurred in the Karroo another clinical entity characterised by severe icterus, but in which photosensitization did not appear, namely Enzootic icterus (the toxaemic jaundice of other lands). In this disease there is a sudden severe haemolytic crisis, a retention icterus and a hepatitis of varying severity culminating in cirrhotic changes and kidney lesions. This condition as it occurs in South Africa is only mentioned briefly by De Kock in two papers in 1928([16], [17]).

That Theiler might have encountered at this time cases in which Geeldikkop and Enzootic icterus co-existed seems very likely, especially in view of recent work. It is very likely that even in present times the Enzootic icterus syndrome has been responsible for some confusion in the pathology of other disease conditions encountered in sheep in the Karroo areas. For instance, Belonje([18]) described the presence of a severe jaundice in field cases of Wesselsbron virus disease. The existence of this icterus is not satisfactorily explained by the nature of the viral hepatitis.

A full account of the pathology and chemical pathology of Enzootic icterus and its relation to the Geeldikkop syndrome will be published later in this series. Recently liver lesions highly reminiscent of those described by Theiler have been produced by the author following prolonged administration of *Pteronia pallens* L. (Scholtzbossie) to a rabbit. It is noteworthy that this plant is almost invariably found on farms where Enzootic icterus occurs as a definite clinical entity.

Following Theiler's epic contribution to this work, ten years elapsed before the next published work appeared, when in 1928 three papers appeared. De Kock([19]) in a description of "swelled head" in rams (now known to be a bacterial infection) discussed

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the relationship of this condition with Geeldikkop and believed it to be due to a group of toxic principles, which develop in plants and have a peculiar predilection for the head of sheep; an idea already mooted by Theiler in 1918. Steyn([20]) described a condition termed "dikoor", occurring in young lambs (4 months to 1 year old), running on old lands in Natal, the Free State and Transvaal. This condition simulated Geeldikkop very closely, but did not appear to be due to Tribulus, but rather to *Panicum maximum*, the only plant common to the old lands on which the disease was seen. It is of some considerable interest that he found this Panicum to be severely infected with a fungus (later identified as *Ustilago* sp.) but was unable to reproduce the condition on feeding such plants to sheep at Onderstepoort. Attention is drawn to the fact that the disease apparently occurred only in lambs. An apparently identical condition occurs amongst lambs of the same age group in Norway and is known as "Alveld". It has recently been described by Ender([21]) and is believed to be caused by the plant *Narthecium ossifragum* L. (*Lilliaceae*). Recent work on Geeldikkop, to be published later in this series, affords what might be a possible explanation for the occurrence of these conditions in lambs.

Quin([22]) discussed the two conditions described by De Kock and Steyn and indicated the close similarity of "dikoor" with "Geeldikkop" and the relationships between the two conditions regarding incidence and seasonal variations. He intimated that if "Geeldikkop" could be produced by other plants widely different from Tribulus, then it and other forms of "dikkop" should be regarded as a symptom complex and not diseases *per se*. This idea was borne out by his subsequent work, and today we subscribe to the idea that Geeldikkop as it occurs in the Karroo might be only one manifestation of a basic and as yet undefined condition, which may appear in other outwardly unrelated forms.

Quin then proceeded to cite experiments carried out in the Victoria West district, which had established the following facts:-

(a) Sheep grazing on wilted Tribulus developed the disease;

(b) sheep grazing on the green luxuriant dubbeltjie did not develop the condition. These findings are contrary to those made by Theiler but are in accordance with some of his later observations and our own recent experiences.

(c) The head and ears of the sheep show a marked vulnerability towards the formation of oedema, as seen in natural cases of the disease. He cited work by Tainter and Hanzlik([23]) where such oedema had been produced following subcutaneous crural injections of p-phenylene-diamine.

He indicated clearly that the toxic agent present in Tribulus was of a very elusive nature and that many factors were probably concerned in determining the toxicity of the plant. The principles

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which produced the icterus and photosensitization might not be the same in both cases.

The period from 1928 until Quin's untimely death in 1950 was one of great productivity as far as results in this work were concerned. In 1929 Quin[24] published a further report in which various grazing experiments performed on a farm in the Burghersdorp district and drenching experiments done on the same farm and at Onderstepoort, were described. He was again able to prove that Tribulus produced the disease and this time claimed that all growth stages of the plant were responsible. He noted the abrupt disappearance of the disease after heavy rains had fallen and found that subsequent drenching with the juice of the plant in the green late fruiting stage or hay made from plants in a similar stage of growth, produced only sudden death associated with Methaemoglobinaemia in most cases. A further paper followed up this report in 1930 in which further drenching experiments with aqueous extracts of tribulus hay were described[25] Sudden death again associated with Methaemoglobinaemia was seen in many instances.

His next paper[26] published in 1931 was to initiate a long series of research on photosensitization in sheep. He demonstrated the photosensitizing effect of crude haematoporphyrin on sheep and small laboratory animals, and noted that the skin lesions and oedema produced were identical to those seen in Geeldikkop. No icterus was however evoked. It was noticed that the condition only developed when sheep were exposed to direct sunlight and that black pigmentation of the skin protected these areas completely from the harmful effects of sunlight. Application of brown or black dyes to the skin seemed to afford the best measure of artificial protection.

Henning[27] in his book published in 1932 reviewed the work which had been done to date on "Geeldikkop" and "Dikoor".

In a successive series of papers by Quin and co-workers many interesting and important facts came to light. Various fluorescent dyes, after intravenous injection into sheep were found to produce intense photosensitization, but no icterus[28]. The lethal factor present in Tribulus which produced Methaemoglobinaemia was identified as inorganic nitrite, chiefly the potassium salt formed from pre-existing nitrate in the plant under the influence of enzymic oxidation-reduction systems [29, 34]. *Hypericum ethiopicum* was shown to produce photosensitization when drenched to sheep, and a deep red fluorescent pigment extracted from it was shown to be responsible[30]. Similarly it was shown that *Lopholaena coriifolia* produced severe liver damage, mild icterus but no photosensitization[31]. The typical symptoms of Geeldikkop were then produced in further drenching experiments, using *Lippia Rehmanni* (Pears) and *Lippia pretoriensis* (Pears)[32]. This latter work proved to be a major step forward for now at last plants were

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found with which the disease could be produced almost at will for laboratory studies.

Quin rapidly followed up this success with his classical work on the effects of surgical obstruction of the normal bile flow in sheep([33]). Not only was the expected regurgitation icterus produced but the experimental animals were found to be intensively photosensitive as well. This was a hitherto unknown occurrence and it appeared likely that the excretion of some fluorescent compound, possibly a normal constituent of bile, was interfered with following bile duct ligation. This compound then apparently accumulated in the blood circulation and so gave rise to the pohtosensitization. The stage was now set for one of the most important findings yet.

Quin and Rimington([35]) then reported the finding in the blood of such experimental cases, a fluorescent pigment which differed in its absorption spectra from any known haem derivatives. It was then found in the bile of normal sheep, and its daily excretion under various conditions was determined. The same pigment was then identified in the serum and bile of natural cases of Geeldikkop.

They were able to isolate it in pure crystalline form and identify it as Phylloerythrin, a porphyrin derived from chlorophyll. Injection of 0.1 gm. of the substance into a sheep produced an intense but transient photosensitization, not accompanied by icterus. The same response was evoked by phylloerythrin prepared in the laboratory from chlorophyll.

Phylloerythrin and its relation to chlorophyll had of course been known for years, having been first demonstrated in herbivorous faeces by Marchlewski in 1904([36]), but these workers were able to show that its chief site of formation is in the rumen as the result of microbial activity, and that it is mainly absorbed in the duodenum and small intestine.

In the same paper they showed that drenching with *Lippia Rehmanni* produced not only the characteristic icterus, but that retention of phylloerythrin also occurred and was responsible for the photosensitization produced. On a chlorophyll free diet no photosensitization was produced and only the icterus was seen. They were able to conclude that the plant contained a single factor which caused simultaneous insufficiency of the liver in eliminating bile and phylloerythrin.

It is interesting to note that in this paper the histopathological lesions of the liver of natural cases of the disease are described as: "only very slight changes — e.g. fine fat droplets and necrosis in some parenchymal cells". Full experimental details of this classical work and the isolation, characterisation and biological formation of phylloerythrin appeared in 1934 and 1935([37], [38], [39]).

Steyn then reviewed the problem of photosensitization diseases

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in his book, "The Toxicology of Plants in South Africa"([40]), and a brief account is also given of the syndrome of "Lugdikkop".

Quin and Rimington([41]) reviewed the problem of Geeldikkop in the light of their knowledge to date and put forward some useful thoughts regarding the management and control of outbreaks of the disease.

Quin then commenced his studies on the bile flow of merino sheep under various conditions and by means of biliary fistulae was able to measure the daily output of bile in these animals. The effects of *Lippia Rehmanni* and other well known liver poisons on bile flow were studied, and he was able to show conclusively that dosing of the plant causes a complete disappearance of bile pigments from the bile, as well as a marked decrease in the flow of bile, which may even entirely cease. It was only after a period of four to five days that normal bile flow and pigment excretion was resumed. These effects were not observed after the administration of the other liver toxins used. The administration of various cholagogues during the period of Lippia-induced icterus did not stimulate bile flow or pigment excretion again. He concluded from this work that the toxin present in Lippia had a paralysing effect on the excretion of bile by the liver, the sluggishness of bile flow then resulting in the pigments escaping into the small blood and lymph vessels and so entering the general circulation. This work appeared in print in 1936, some while after it had been carried out([42]). The liver lesions seen in Geeldikkop are once again described as "very little morphological change microscopically, except for bile staining".

During the course of 1935, Rimington and Quin([43]) announced the isolation of an icterogenic principle from *Lippia Rehmanni*. Preliminary chemical work on this substance which they named icterogenin was described, and it appeared to be of a resinic acid nature. It was found to be physiologically active and upon dosing to sheep produced the symptoms observed in Geeldikkop and Lippia poisoning. The sodium salt of icterogenin was found to paralyse isolated strips of intestine placed in the Dale bath in concentrations as low as 1 in 200,000.

In a further paper([44]) dealing in detail with the isolation procedures and preliminary chemical work on icterogenin, three crystalline forms of the substance are described designated icterogenins A, B and C. All three forms were physiologically active and were believed to be isomeric forms of the same compound. Their structure was then partially elucidated. It was noticed that not only did icterogenin induce the characteristic icterus and photosensitization but oral dosing produced a marked decrease in rumen motility. The compound appeared to be exceedingly toxic following intravenous injection, doses of 1 gm. producing death within a few hours. In the same paper the distribution of icterogenin between the leaves, stems and roots of Lippia plants,

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throughout the year, was reported. The greatest concentration of the compound was found to occur in the root bark. A transference of icterogenin from leaves to root bark was found to occur normally and the growth of young leaves was found to be associated with an increased production of icterogenin. Further studies on the seasonal distribution and translocation of icterogenin in the plant were made by Roets[45]. The substance is apparently present in the plant as the free acid.

A further report of an outbreak of Geeldikkop amongst sheep due presumably to Panicum species, by Rimington and Quin appeared in 1937[46], but they were unable to isolate any active substances from this plant.

Some of the morphological and physiological aspects of Tribulus were reported by Henrici in 1938[47], and she also made studies on variations in the pigment and mineral content of the plant.

During 1940 Steyn and Van der Walt investigated an outbreak of a disease among dairy cattle near Durban associated with icterus and photosensitization. They were able to incriminate as the cause of the condition the ornamental plant *Lantana camara* L. (*Verbenaceae*), and in drenching experiments performed on sheep with the plant on their return to Onderstepoort, they were able to evoke a condition essentially similar to natural Geeldikkop and Lippia poisoning. This work was published some time later[48].

From this plant Louw[49] was able to isolate a crystalline compound which he named Lantanin. This substance was also found to be capable of producing severe icterus and photosensitization.

Van der Walt and Steyn had some time previously reported finding another plant suspected of causing Geeldikkop, viz. *Asaemia axillaris* (Thunb.) Harv., the "vuursiektebos" (*Compositae*)[50]. However no work seems to have been done on the chemistry of this plant.

In a series of experiments, Riemerschmid and Quin[51] studied the effect of sunlight on the skin of sheep artificially photosensitized with eosin and phylloerythrin, the latter photosensitization being produced with Lantana camara and Lantanin. They were able to show that the wavelengths of light which produced the photosensitization reactions in the skin of these sheep correspond to the absorption maxima of eosin and phylloerythrin in each case. In both instances these absorption maxima fall chiefly within the visible portion of the spectrum.

In two further communications Louw reported further work which had been done on Lantanin[52, 53]. As the name Lantanin had already been given to an alkaloid extracted from another Lantana species elsewhere, he suggested that the name of the compound found by him should be changed to Lantadene A. During the course of this work he noted the occurrence of another

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crystalline substance along with the former compound, which he then accordingly named Lantadene B. It was found to be physiologically inactive. In these papers the partial elucidation of the structure of these compounds is discussed.

Some indications for the control of Geeldikkop which should be impressed on farmers in the effected areas appeared in a short paper by Quin in 1948([54]) and a further excellent review of the problem of photosensitization in this land appeared during the following year in Steyn's second *monograph*([55]). The development of photosensitization and icterus following ingestion of the severe hepatotoxin produced by the toxic alga (*Microcystic toxica*) appears in this work. Further details of this condition may be found elsewhere([56], [57], [58]).

For a while it seemed that chemical work on Tribulus had taken second place to the chemistry of all the aforementioned plants. However, interest in it was renewed when Sapiro([59]) in a series of experiments, studied the formation of nitrite from nitrates by rumen micro-organisms, and the action of nitrous acid on chlorophyll. He found that when sheep are on a poor diet, the nitrite formed from nitrate by the rumen microflora is slowly utilized by these, and may accumulate in dangerous amounts. The occurrence of four crystalline compounds after nitrous acid is allowed to act on chlorophyll, none of which produced photosensitization or icterus after dosing to sheep, is reported.

Henrici followed up this renewed interest in the chemistry of Tribulus with her work on the starch and sugar content of the plant under various conditions([60]). She also reported the occurrence of a substance with properties similar to the saponins in some species of Tribulus, and claimed that zinc deficiency in certain soils was responsible for the appearance of the "saponin-like" compound, but put forward no evidence to support this view.

During the course of 1952 an outstanding review of the photosensitization diseases in domestic animals was published by Clare, wherein all the available information on the mechanisms involved in these conditions was presented and the various forms of photosensitization classified according to their genesis([61]). A further excellent review of the subject bringing this knowledge up to date was published again by the same author in 1955([62]). A noteworthy fact discussed in this latter review was that the author was able to produce in rats, dosed with alcohol or ether extracts of *Panicum miliaceum*, a severe primary photosensitization not accompanied by liver lesions or icterus. He found this photosensitization to be due to four chlorophyll derivatives produced as artefacts during the preliminary drying of the grass. It would appear from this work that the livers of small rodents are not well enough adapted to excrete large amounts of the lower degradation products of chlorophyll.

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During the course of their brilliant investigations into the constitution of the triterpenoid compounds, Barton and co-workers in 1954 were able to elucidate the structure and stereochemistry of the icterogenins and Lantadene B. Barton and De Mayo([63]) found icterogenin to be a pentacyclic triterpene acid, and that the icterogenins B and C reported earlier by Rimington were solvated forms of this compound. During this work they discovered the co-existence with icterogenin in *Lippia Rehmanni*, of a new triterpene acid, which they designated rehmannic acid. In a further paper([64]) they reported this compound to be a desoxy icterogenin. During the same year Barton *et al*([63]) were able to show that Lantadene B was isomeric with rehmannic acid and deduced its structure. A full account of the chemistry of these compounds is also to be found in the recent works by Simonsen and Ross([66]).

For a while after the untimely death of Quin, research work on Geeldikkop faltered somewhat, but in January, 1955, investigations were once more resumed on a large scale, this time as part of a joint research project between Onderstepoort and the Council for Scientific and Industrial Research. This collaborative effort was soon to prove most fruitful. The occurrence of saponins in Tribulus, as reported by Henrici([60]) was re-examined by Enslin and Wells([67]), who were able to isolate crude saponins in yields of 0.5–2.0% of the dry weight of the plant. Following chromatographic separation of the products of acid hydrolysis of the crude saponins, they were able to isolate two crystalline steroidal sapogenins, provisionally designated sapogenins A an B. The former was shown to be identical with Diosgenin, and the latter appeared to be a new sapogenin. After partial hydrolysis of the crude saponins with the very active β-glucosidase, elaterase, a pure white desglucosaponin was obtained which was found to paralyse a duodenum strip in a dilution of 1 : 45,000.

Adelaar([68]), during a severe outbreak of the disease, repeated the earlier inoculation experiments using blood from affected animals, with negative results.

In view of the dramatic developments which had occurred overseas during the last decade in the fields of bile pigment and porphyrin metabolism, and in the genesis of icterus, it was decided to commence a comprehensive study on the biochemistry of bile pigment excretion in the sheep as well as the chemical pathology of various forms of jaundice in this animal. Such a programme was accordingly initiated and entailed the initial study and development of tests for evaluation of hepatic function in the sheep, the conjugation and excretion of bile pigments and dyestuffs like bromosulphonphthalein by this animal, the chemical pathology of experimental bile duct ligation, icterogenin induced jaundice and natural cases of Geeldikkop. These studies were further supported by haematological and histopathological work on all the conditions

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concerned and later as the need became evident, by studies on renal function, copper metabolism and electrolyte balance.

This work will be reported on in forthcoming papers in this series and elsewhere. However it soon became clear that the severe jaundice and dye retention seen in icterogenin poisoning and in natural cases of "Geeldikkop" were due to a biochemical lesion affecting the hepatic conjugation systems for bile pigments and dyestuffs, and also the excretion of these compounds. It appeared that the toxic substances in the plants concerned or substances derived from them biologically seemed to compete in or inhibit the enzyme systems concerned with such conjugations. There was furthermore a definite intereference with the excretion of such pigments that were conjugated as well. Whether the same holds for porphyrins like phylloerythrin or not, is not at all clear.

It was demonstrated that at least three well defined conjugates of bromosulphonphthalein appear in the bile of sheep after injection of this compound, and that this conjugation is markedly interfered with by icterogenin. In natural and artificially induced Geeldikkop the presence of at least two bilirubin conjugates thought to be mono and diglucuronides, as well as free bilirubin, has been found in the blood circulation. It is certain that Geeldikkop in its natural forms is a far more complex disease condition than was believed by earlier workers. A close relationship between this disease and Enzootic icterus has also been demonstrated.

Because of the apparent lability of the toxic substances present in Tribulus, and the large amounts of material required for the extraction of sufficient of these compounds for dosing experiments on sheep, it soon became very clear that much of this work would have to be carried out in the field during the times that the disease appeared. Such conditions were also a prerequisite for studies on the biochemistry and chemical pathology of natural cases of the disease. These field studies were then made possible by the very generous gift to this Institute of the Mobile Laboratory, by the Trustees of the Stock Diseases Research Fund. This mobile unit and the potentialities which it offers for work of this nature will be discussed in the following paper.

Two extensive field investigations, using this equipment, have already been conducted, the first during January and February of 1958 and the most recent work during the period November, 1958, to January, 1959. Following the first of these investigations De Kock and Enslin([69]) were able to report the isolation and characterisation of four sapogenins obtained from the crude saponins present in Tribulus. These were all steroidal sapogenins containing the spirostan nucleus and were identified as diosgenin, ruscogenin, gitogenin and 25D-spirosta-3:5-diene. The degradation of ruscogenin to 1β, 3β dihydroxypregna-5 : 16 dien-20-one and 1β, 3β dihydroxypregn-5-en-20-one is also described. As will be seen later in the following papers these latter findings might

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be highly significant. A further communication on the saponins and sapogenins of Tribulus is shortly to appear elsewhere([70]). Some of these compounds have so far been tested for icterogenic activity, but although many interesting features of the disease were produced, we have not been able to produce the characteristic intrahepatic cholestasis or photosensitization. That one or more of these compounds enters into the aetiology of the disease seems very likely, but it would appear that other factors must operate simultaneously.

There exists in New Zealand a disease amongst sheep termed "Facial eczema", which is very closely akin to Geeldikkop. Recently workers on this disease have reported finding a fungus on grass pastures, where the disease occurs, *Sporodesmium bakeri*, which under certain circumstances elaborates a hepatotoxin capable of producing the disease([71], [72], [73]). In view of these very important findings it was decided to re-investigate the role of a fungus in the genesis of Geeldikkop as suggested earlier by Theiler([15]) and Steyn([20]). A number of fungi growing on Tribulus during outbreaks of the disease have been isolated in pure culture and tests with these are due to commence shortly. It is thought possible that one or more of thee fungi might produce the toxic agent or might under certain circumstances act on substrates present in the plant (e.g. the saponins and sapogenins) increasing their toxicity. This work will be reported on later.

During our work on Lippia induced jaundice the icterogenin which was used was actually a mixture of icterogenin and rehmannic acid, as isolated from the root bark of the plant. A chromatographic method was developed([74]) which allowed for separation of the mixture into two major fractions, namely an icterogenin fraction and a rehmannic acid fraction. Sufficient of these materials then became available for testing and we were able to demonstrate that the toxicity of the mixture as hitherto used on sheep, was due to the rehmannic acid fraction and not icterogenin itself, as had always been believed. This work will be reported on later in this series.

Summary

A review of the literature and the research work carried out on Geeldikkop from the time that the disease was first described until the present date, is presented. Various new important findings and views are described and the icterogenic effects of Lippia rehmanni in sheep are reported as being due to the rehmannic acid fraction of the crude mixture of triterpene acids, hitherto described in the literature as "icterogenin".

Acknowledgments

The permission of the Director of Veterinary Services for the publishing of this review is gratefully acknowledged.

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INTRAHEPATIC CHOLESTASIS IN A RHODESIAN RIDGEBACK DOG: A CLINICOPATHOLOGICAL STUDY

W. D. MALHERBE
Onderstepoort

SUMMARY

Primary intrahepatic cholestasis is a form of biliary obstruction which has apparently not been described in veterinary literature. This paper records a clinicopathological study for some 2½ months of this syndrome which resulted from accidental subacute poisoning or sensitization of a dog with acetarsol. It was characterized by severe icterus, relatively acholic, clayey faeces, high values for plasma alkaline phosphatase, total cholesterol, total and conjugated bilirubin. Tests of hepatocellular involvement were generally negative, and haematological examination revealed the absence of anaemia and the presence of eosinophilia. The question of differential diagnosis is discussed.

INTRODUCTION

The usual form of obstructive jaundice in which there is total, partial or intermittent occlusion of the bile passages by tumours, calculi or strictures, is well known to clinicians in both medical and veterinary fields. It is characterized by a more or less distinct clinical picture and by biochemical findings differing in many respects from those in other types of jaundice involving the hepatic cells themselves. Prominent among these findings is the absence or irregular appearance of small amounts of stercobilinogen and urobilinogen in faeces and urine, indicating nonpatency of the bile passages. Clinically there are usually found: severe icterus in the visible mucous membranes, reasonably good habitus, some loss of condition, and the passage of pale, clayey, sour-smelling stools and very dark brown urine. The temperature is usually within the normal range.

In South Africa on account of the widespread incidence of canine babesiosis the vast majority of jaundiced dogs is found to be suffering from this disease in a severe form or as a result of neglect of treatment in good time. This fact is of greater differential diagnostic importance than in most other countries.

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Such animals are however extremely depressed and usually still feverish on presentation. Their stools are yellowish, as a rule, and of normal to soft consistency, and urine may be strongly coloured by haemoglobin as a result of massive haemolysis or is more generally dark brown and bile laden. Large amounts of "urobilinogen" are present.

During the past two decades attention has been drawn in human medicine to a less frequently occurring type of obstructive jaundice in which all the extrahepatic part of the biliary tract is patent and the gall bladder is usually virtually empty. This phenomenon has been reviewed by Johnson and Doenges[1] and has been named primary intrahepatic cholestasis[1, 2] or intrahepatic obstructive jaundice [1, 3] and has been found to occur in some cases of viral hepatitis of man[6], arsphenamine poisoning with jaundice [1, 4], and in chlorpromazine jaundice[5, 11, 13]. It has also been recorded in association with sublingual administration of methyl testosterone[7, 9], with the treatment of myelomatosis in man with 4-4'-diamidinodiphenylamine[8], and with various other substances.

This intrahepatic cholestasis was usually found to be due [1, 3, 11, 13] to obstruction of the minute bile ducts in the region of the portal triads, usually with a more or less heavy infiltration of round cells, polymorphonuclear cells, histiocytes and sometimes eosinophiles in this area, surrounding small bile ducts or ductules and presumably causing pressure on them and thus occluding them. Aschoff (*cit.* [3, 13]) has picturesquely referred to the junction between the bile capillaries and bile ducts at the lobular periphery as being the "Achilles' heel of the biliary system". Characteristically the hepatic cells themselves are not greatly affected for the most part and lobular architecture remains undisturbed. Inspissated bile and bile plugs in the small bile capillaries are regarded as the dominant lesion.

From recent work there seems also to be a possibility here of disturbances of the handling of bile by enzyme systems at the hepatocellular level and that "obstruction" could arise from inhibition or insufficiency of such systems. In the veterinary field, observations in South Africa[20] have revealed this type of obstruction with a histological picture innocent of portal infiltration, in so-called "geel dikkop" of sheep thought to be associated with ingestion of the plant *Tribulus terrestris* under certain conditions.

The importance of intrahepatic obstruction arises naturally from the fact that its treatment is medical whereas in the usual form of "post-hepatic" obstruction, i.e. in the vicinity of the gall-bladder or common duct, it is essentially surgical.

Intrahepatic cholestasis has received but scant attention in veterinary medicine and it is the purpose of this paper to record a case in a dog which had been considerably overdosed with acetarsol. The association with arsphenamine poisoning in man seems obvious.

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Material and Methods

The dog in question — a Rhodesian Ridgeback bitch aged 2½ years — was presented for clinical pathological study on February 9, 1959, as she was suffering from a severe degree of jaundice and had not been responding at all to normal treatment for liver injury.

She had been noticed to be icteric on January 23 after a ten-day course of 24 grains of acetarsol ('Stovarsol' M&B) daily. As her weight was 30 Kg. this represented a little over twice the recommended dosage. As the clinical pathology strongly indicated obstruction, exploratory laparotomy was performed on February 14. The gall-bladder was found to be empty and the common duct patent, and the liver appeared to be completely normal except for dark colour.

Some days after the operation the animal contrived to tear out the stitches of the laparotomy wound and opened it up, heralding a period of less good habitus, loss of condition and a generally "toxic" appearance. This will be referred to as the "toxic" period.

A long acting corticotropin preparation ('Acton prolongatum') was injected, 10 units daily from February 17–20, and courses of prednisolone from February 28 to March 4, March 12 and 13. Apart from some bouts of diarrhoea she remained cheerful and recovered gradually. Icterus receded slowly till at discharge on April 27 it was becoming very faint.

The animal was subjected to a battery of tests at intervals over a period of about 2½ months to follow the progress of recovery. These tests included:

a. Haematological examination, the results of which are tabulated in Table I.

b. Differential plasma protein determination by fractionation by 23% sodium sulphate salting out([26]) and using Weichselbaum's biuret reagent([27]).

c. Prothrombin time by Quick's one-stage method.

d. Total cholesterol, and ester percentage (Zlatkis, Zak & Boyle([22]) and MacIntyre & Ralston 1954 (*cit.* [23]).

e. Blood uric acid (Brown [24]).

f. Serum glutamic oxalacetic and pyruvic transaminases (King [25]).

g. Zinc sulphate turbidity (Kunkel, 1947, *cit.* [23]).
Thymol turbidity([23]).
Colloidal gold flocculation([23]).

h. Quantitative estimation of conjugated and free bilirubin by Malloy & Evelyn's method([21]).

i. Routine urine examination, including tests for conjugated bilirubin and for urobilinogen.

j. "Urobilinogen" in faeces([23]) with Terwen's solution as standard.

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TABLE I. HAEMATOLOGICAL FINDINGS.

	Feb. 9	Feb. 14	Feb. 18	Feb. 24	Mar. 3	Mar. 10	Mar. 18	Mar. 25	Apr. 2	Apr. 9	Apr. 22
Erythrocyte sedimentation rate mm./hr. (Wintrobe)		0	2	25	6	1	4	5	4	6	1
Erythrocyte count, 10^6/cu.mm.	7.86	7.86	6.81	5.44	5.51	5.20	4.31	5.19	5.23	5.0	6.83
Haemoglobin, gm./100 ml.	16.35	16.85	16.35	11.4	11.6	13.45	10.06	13.45	14.4	13.05	15.35
Haematocrit. %		50	39	33	32	32	34	38	41	36	44
Mean corpuscular vol. cu. μ (Wintrobe)		64	57	61	58	62	79	73	78	72	64
Leucocyte count 10^3/cu.mm.	10.9	15.5	18.2	20.1	23.1	8.3	7.8	6.7	6.9	7.2	8.4
Neutrophile polymorphs %		52	67	70	84			66	62		66
Lymphocytes %		10	14	12	8			19	16		21
Monocytes %		4	5	8	8			5	5		4
Eosinophile polymorphs %		34	14	10	0			10	17		8

TABLE II. PROTOCOLS OF TESTS MAINLY AFFECTED BY HEPATOCELLULAR DAMAGE.

	Feb. 9	Feb. 14	Feb. 18	Feb. 24	Mar. 3	Mar. 10	Mar. 18	Mar. 25	Apr. 2	Apr. 9	Apr. 22
Total protein gm. %		6.50	6.32	5.3	5.46	5.10		5.3	5.3	6.0	5.01
Albumin gm. %		4.04	3.5	2.82	3.86	3.04		3.08	3.0	3.16	2.21
Globulins gm. %		2.46	2.82	2.48	1.60	2.06		2.22	2.3	2.84	2.8
Prothrombin time: secs.		19	15	17.5	17	16.5	16.5	16	16.5	17	
Cholesterol ester: % of total						58	56	76	66	69	63
Blood uric acid mg. %		2.10	2.32	2.15	1.99	2.04	1.00	1.48	1.22	0.92	0.89
SGO-T (King)	240	200	240			187	226	223	200	146	92
SGP-T (King)	420	384	429			429	454	479	408	471	125
Zinc sulphate turbidity		0.25	0	0.1	0.2	0	0.1	0.2	0.2	0	0.1
Thymol turbidity		8.1	7.8	5.4	11.6	4.8	0.5	0.35	0.35	0.35	0.25
Thymol flocculation		—	+	+	+	∓	+	+	+	—	—
Colloidal gold flocculation		—	—	—	—	—	—	—	—	—	—
Plasma iron mcg. %			194	200		146	92	96	100	50	88

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k. Bromsulphalein (BSP) retention (5 mg. per Kg. dose).
l. Plasma alkaline phosphatase (King and Armstrong *cit.* [23]).
m. Plasma iron (Marrack, *cit.* [23]).
n. Plasma amylase (Gomori, [28]).

The results are summarized in Tables 2 and 3, representing tests useful for indication hepatocellular damage and obstruction to the biliary outflow respectively.

Results

Haematology (See Table 1)

Erythrocyte sedimentation rate (Wintrobe tubes) was normal throughout, except at the test on February 24 during the "toxic" period when wound healing was delayed.

Erythrocyte count was also affected somewhat during the same period, but anaemia was never a feature of the disease picture.

Haemoglobin and the *haematocrit percentage* followed substantially a similar pattern.

Mean corpuscular volume (Wintrobe) showed a slight microcytosis during the "toxic" period.

Leucocyte count showed a significant rise during the same period, with a concomitant rise of neutrophile percentage and a relative drop in eosinophiles which had been high. Corticoid therapy had possibly been effective in this respect.

Tests Largely Affected by Hepatocellular Damage (Table 2)

Differential protein determination. There was a mild drop in total protein during the period of delayed wound closure, but at no stage was the normal albumin/globulin ratio adversely affected.

Prothrombin time remained normal during the whole period of the study.

Cholesterol ester percentage was not determined during the first half of the period. Hence no conclusions could be drawn from the available figures.

Blood uric acid was slightly elevated, notably during the "toxic" period.

Transaminase activity, particularly that of the pyruvic transaminase, remained elevated till late in the recovery phase.

Turbidities and flocculation tests were negative with the exception of thymol turbidity which showed moderately increased figures.

Tests Largely Affected by Obstruction

Bilirubin estimations. The prompt diazo-reacting fraction (bilirubin glucuronides) was always predominant. Total bilirubin was initially high (up to nearly 20 mg. per 100 ml.) but dropped rapidly during the recovery phase.

Bile pigments in urine followed the same pattern as the concentration in the plasma.

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TABLE III. PROTOCOLS OF TESTS MAINLY AFFECTED BY OBSTRUCTION OF BILIARY DRAINAGE.

		Feb. 9	Feb. 14	Feb. 18	Feb. 24	Mar. 3	Mar. 10	Mar. 18	Mar. 25	Apr. 2	Apr. 9	Apr. 22
Plasma Bilirubin mg. %	Total	17.10	11.15	19.60	15.90	15.20	11.80	4.35	2.45	1.35	0.45	0.20
	Conjugated	12.90	11.15	11.20	9.60	9.1	5.90	2.20	0.90	0.65	0.20	0
	Free	4.20	0	8.40	6.30	6.1	5.90	2.15	1.55	0.7	0.25	0.20
Conjugated bilirubin in urine		*+	*+	*+	*+	**+	++	+	+	+	—	
Urobilinogen in urine		+		+	±	±		+	+	+	+	
Stercobilinogen in faeces			+	±	±	±	41.6 mg %			25.2 mg. %		
BSP Retention %		72	78	83.2	56.8	62.4	57.6	46.4	34.8	29.6	12.8	7
Alkaline phosphatase (King-Armstrong units)		178	188	499	329		276	264	303	189	148	68
Total cholesterol mg. %		438		539	438		538	440	427	336	253	207

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"Urobilinogen" in faeces and urine was scanty during the period when the faeces was clayey, greyish and rancid smelling, but appeared to improve steadily with improvement of faecal consistency.

Bromsulphalein retention reached a maximum of 83.2% on February 18 (during the "toxic" period), just after corticotropin therapy had been initiated, and thereafter decreased steadily.

Alkaline phosphatase was consistently high, reaching its highest peak of 499 King-Armstrong units on February 18 and then receding slowly.

Total cholesterol (free plus ester cholesterol) was high on February 18 and March 10 and then came down gradually.

Serum iron was not done regularly but was markedly elevated till some time after total bilirubin had started receding. It tended generally to parallel the decline of BSP retention.

Routine *urine examinations* showed never more than traces of albumin; pH usually around 6; reducing substances negative; ketone bodies negative; and in the centrifuged deposit a few heavily bilestained granular casts were occasionally seen. Fluctuations in specific gravity reflected no more than the state of hydration at the time.

Blood urea nitrogen was determined regularly and always gave figures well within the normal range.

Plasma amylase figures were erratic and generally on the low side.

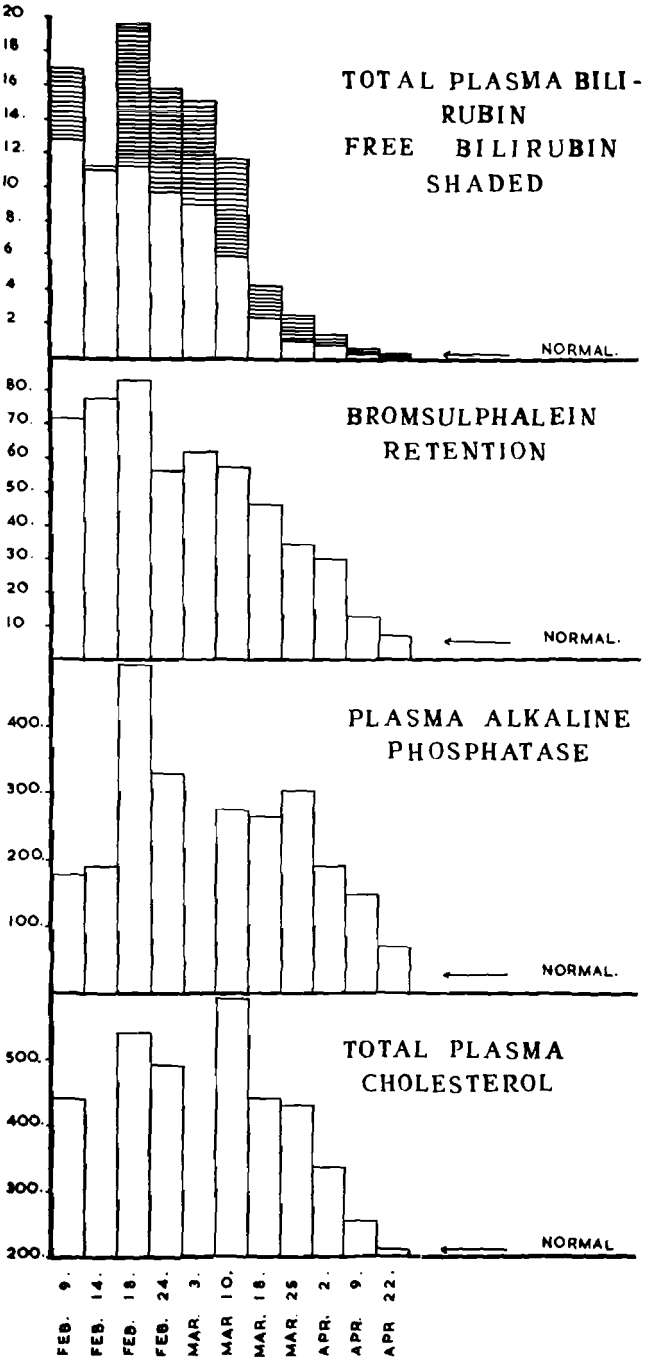
Blood sugar was generally in the vicinity of 60 mg. per 100 ml. and showed no significant fluctuations.

Discussion and Conclusions

It was immediately evident, when the case was submitted for clinico-pathological study, that the most striking points of difference between the more usual cases of jaundice seen in South Africa (*Babesia canis* infection) and the present one were absence of anaemia, splenomegaly, fever, marked malaise and kidney involvement. Clearly in this case the liver was primarily involved and haemolysis could be excluded.

The diagnosis of obstructive jaundice emerged at once with high figures for plasma alkaline phosphatase([4], [10], [11], [13]) total cholesterol and high total and conjugated bilirubin. This was supported by the relative acholia and abnormal physical appearance of the faeces and the low content of urobilinogen in the urine in spite of an abnormally heavy load of conjugated bilirubin. Moreover the animal tended to be more cheerful than one would expect when considering the degree of icterus. It has been frequently remarked e.g. by Johnson and Doenges([1]) that in human medicine patients with cholestasis "feel and appear considerably less ill than those with a comparable degree of jaundice caused by hepatocellular disease".

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The degree of icterus is frequently affected considerably by the functional integrity of the kidneys. Clearly this played no role in the present case as there was at no stage any evidence of kidney involvement.

Albumin synthesis in the liver was unaffected as was prothrombin production. Turbidity and flocculation tests were essentially negative with the exception of thymol turbidity which showed slightly elevated values — a picture found fairly often([11]) in cholestasis due to chlorpromazine jaundice. Popper and coworkers([12]) have however pointed out that these tests are not of much use under these conditions, since, when there is considerable regurgitation of bile, there is inhibition of their functioning, particularly of zinc sulphate turbidity, thus masking the possible presence of parenchymal disease. Whether this holds in animals has not yet been clarified. Cholesterol ester percentages were estimated only when recovery had set in, and therefore permit of no conclusions. During this period certainly they were quite normal. Uric acid levels in the blood were slightly elevated above those considered normal.

The general picture then was a lack of any marked hepatocellular involvement.

A few points seem to merit additional discussion.

The *eosinophilia* in the peripheral blood such as noted in this case has been found to occur "not infrequently" in chlorpromazine jaundice. Gebhart et al([11]) and Shay & Siplet([13]) have speculated on the aetiology. They have suggested allergy, but against this mentioned cases where renewed chlorpromazine treatment had no untoward effect, and pointed out the inconsistent results of steroid therapy. In this instance there seemed to be a general improvement after corticoids were introduced, but this finding could conceivably have been coincidental. It has been the experience in human medicine([1]) that corticoids have been dramatically useful in the "cholangiolitic" types (human viral hepatitis) but not in the "cholangiohepatitic" ones due to chemical causes (arsphenamine, etc.). One could suggest the possibility of adrenocortical hypofunction as playing some role in the present instance.

The *plasma alkaline phosphatase* determination has been regarded by Gebhart *et al*([11]) and Shay and Siplet([13]) as the most sensitive means of detecting early "sensitivity reactions" to chlorpromazine. The former authors found that is was the last of the liver function tests to return to normal limits on recovery. Hanger and Gutman([4]) used only this test and cephalin flocculation to distinguish between hepatogenous and cholestatic forms of post-arsphenamine jaundice. The test was certainly useful in the present instance and had, even at the time of discharge, not returned to normal.

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Total cholesterol content of plasma similarly followed accepted patterns in that it was significantly elevated, with a more or less normal ester moiety. The highest value (589 mg. %) was found after the bilirubin figure had started receding. This was in accord with the experience of Gebhart et al[11] and Hanger and Gutman[4].

Blood uric acid appeared to be a less useful test. Values were elevated during the "toxic" period. In a comparison of uric acid determinations and BSP retention Morgan[17] has concluded that the latter is a considerably more sensitive test.

Transaminases. In a recent study[14] Rinaldo and Remp have stressed the usefulness of the glutamic oxaloacetic transaminase (SGO-T) determination in cases of hepatitis where biochemical findings otherwise were equivocal. They comment, however, that it is not helpful in differentiating between cirrhosis, metastatic malignancy or liver disease due to biliary obstruction, where the values generally are low. In viral hepatitis, in which liver cell necrosis takes place in many cases, very high values are usually found at some stage of serial tests. It has also been suggested by Shay and Siplet[13] that necrosis may not be necessary but that increased permeability of liver parenchymal cells may permit of the increased passage of transaminases into the blood stream.

The glutamic pyruvic transaminase (SGP-T) has a higher activity in liver than in other organs[15, 16] and elevation in serum is a better index of liver cell necrosis than that of SGO-T. In experimental surgical obstruction in dogs Cornelius[16] demonstrated moderate rises in both SGO-T and SGP-T as a result of secondary centrolobular fatty changes, but very much lower than in the case of livers poisoned with carbon tetrachloride.

In the present study the values of both were elevated to a moderate extent until the recovery phase was well advanced. SGP-T proved more sensitive in that figures were higher.

Serum iron has also recently[14] been shown to be of considerable diagnostic value as an index of hepatocellular involvement. From the determinations done here it would appear that figures were slightly elevated during the "toxic" period but very soon returned to normal. Stone *et al*[19] showed that significant rises of serum iron was associated mostly with liver cell necrosis and that serum levels remained more or less normal in such conditions as cirrhosis and obstruction.

The question arises in retrospect whether cholecystography and biopsy at the time of operation could have provided really useful additional information. According to Johnson and Doenges[1] biopsy is not conclusive in these cases, which may defy accurate diagnosis unless typical portal inflammation plus sparing (or relative sparing) of hepatic cellular morphology can be demonstrated. The views of Popper and Schaffner[2] are in agreement with this. Similarly cholecystography as a general rule does not

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provide anything like certain diagnosis in these cases. Shorter and co-workers[18] quote considerable support for this view. They point out that "radiological examinations are notoriously unsatisfactory in the presence of jaundice". Exploratory laparotomy proved to be of considerable value in removing any doubt as to the site of the obstruction in the case under study, but this procedure is of course open to objection if it can be avoided at all.

Acknowledgement

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FIELD TRIALS ON SOME ANTHELMINTHICS FOR CATTLE

R. K. REINECKE and J. A. SCHUTTE

INTRODUCTION

Various workers have shown that certain organic phosphorus compounds are highly effective larvicides and vermicides. Levine *et al.* (1956) showed that 0, 0-dimethyl 2,2,2, trichloro-1-hydroxy-methyl phosphonate killed the larvae or prevented their development, when mixed with horse faeces containing strongyle eggs at at molar concentration of 0.0001 to 0.00025.

This compound more commonly known by the trade name of "Neguvon" when used in animals, or "Dipterex" when used for insect control, has been used as an anthelminthic in Australia. Riek and Keith (1958) showed that for cattle at dosage rates of 44 mgm. per Kg. (2 grams per 100 lb. body weight), "Neguvon" was highly effective against *Haemonchus placei,* and *Oesophagostomum radiatum.* At dosage rates of 110 mgm/Kg. (5 grams per 100 lb. body weight) this compound was highly effective against *Cooperia pectinata, Cooperia punctata, Bunostomum phlebotomum* and *Trichostrongylus axei.* These results were confirmed in a later publication by Riek (1958).

Gordon (1958) showed that "Neguvon" was highly efficient against *Haemonchus contortus,* in sheep at dosage rates of 55 mg./Kg. and less; but only effective against *Trichosrongylus spp.* at dosage rates of 88 mg./Kg. when preceded by prestimulation of the oesophageal groove reflex with copper sulphate. This drug gave variable results at different dosage levels against *Oesophagostomum columbianum.*

Another organic phosphorous compound 3-chloro-4-methyl-7-oxycoumarine diethyl thiophosphoric acid (Bayer "L21/199", "Muscatox", "Asuntol") was tested by Gordon (1958) as an anthelminic for sheep. He found that the effective dosage rate of this compound against *H. contortus, O. columbianum* and *Trichostrongylus spp.* was too toxic for general use.

Herlich and Porter (1958) carried out critical tests with "Asuntol" (Bayer L21/199). In calves the drug was dosed at the rates of 25 mg., 12.5 mg. and 6.25 mg. per Kg. live weight. At the three dosage levels the drug was highly effective against *Cooperia* spp, (*C. punctata* and *C. pectinata*), *H. placei* and

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O. radiatum. It was most effective against *Ostertagia ostertagi* at 25 mg./Kg. and against *T. axei* at both 25 mg. and 12.5 mg./Kg. The results were erratic against *Nematodirus helvetianus* and *B. phlebotomum* at different dosage levels. These authors only described mild transitory toxic symptoms 24 to 72 hours after treatment at these dosage levels, e.g. diarrhoea, lacrimation, salivation, inappetence and listlessness.

In critical trials in sheep these authors found "Asuntol" highly effective against *H. cortortus, Ostertagia circumcincta* and *T. axei* at 12.5 mgm./Kg. At 25 mg./Kg. it was also effective against *Trichostrongylus colubriformis, Cooperia curticei* and *Strongyloides papillosus*. In lambs toxic symptoms of an even milder degree than in calves appeared within 12 hours of dosing and disappeared 12 hours later.

Riek (1958) reported that "Asuntol" was effective against *H. placei* und *Cooperia* spp. at (5.5 mg./Kg.) and effectively controlled *O. radiatum*. It failed to control *T. axei* and *B. phlebotomum* at this dosage level which was the maximum safe dose for cattle according to this author.

Materials and Methods

"Neguvon" was administered as a ten per cent solution per os and "Asuntol" as a suspension varying in concentrations from one to five per cent. The former is soluble at concentrations of 14% or lower while the latter is only soluble in a few parts per million. In some cases the "Neguvon" used was a 95% powder, in others a 50% wettable powder or a 62% emulsion. "Asuntol" in the form of a 30% wettable powder was diluted to a 5% suspension. In some experiments the compounds were mixed and active ingredients were present in the proportion ten parts "Neguvon" to one part "Asuntol" as a wettable powder. This mixture was added to water so that the "Neguvon" was present as a 10% solution and the "Asuntol" as 1% suspension.

Cattle used in these trials varied in age from 3 months old calves to adult cows and steers 8 years of age and older, their weights varying from 70 to over 500 Kg. They were infested with *Haemonchus placei* (Place 1893), *Cooperia* spp. (*C. pectinata* and *C. punctata*) (v. Linstow 1901), *Oesophagostomum radiatum* (Railliet 1896) and to a lesser extent with *Trichostrongylus axei* (Cobbold 1879) and *Bunostomum phlebotomum* (Railliet 1900).

No premedication with sodium bicarbonate or starvation prior to treatment was carried out.

Egg counts were made on the day of treatment and in one case six days later and again 28 days later, but in all other cases at periods varying from 9 to 28 days after dosing. The effect of the treatment on the various nematodes was estimated by the difference in faecal egg counts before and after treatment, and the conversion of the egg counts to the various species by

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the differentiation of infective larvae recovered from faecal cultures. Control groups were also examined.

The groups cannot be compared with the results obtained by Herlich and Porter (1958) who carried out critical trials. The results obtained by Riek and Keith (1958) and Riek (1958) are also of more value in assessing the effects on the various nematodes since egg counts and larval differentiation were carried out at weekly intervals, two weeks before and one week after treatment, and in most cases two or more post-treatment counts were carried out as well.

Most of the farms on which these drugs were tested were 150 to 200 miles from the laboratory. The collection of specimens and transportation to the laboratory took considerable time, and facilities and staff were just not available for more frequent collection of specimens. In addition, apart from toxicity trials, live weights had to be estimated and these drugs were administered in much lower dosage rates than shown in the results below.

Toxicity trials were carried out at Onderstepoort and Armoedsvlakte. Cattle were weighed before dosing and "Neguvon" was administered at a dosage rate of 150 to 200 mg./Kg. "Asuntol" was dosed to cattle at the rate of 30 mg./Kg. Since only a few animals were included for cholinesterase determinations they are not included in the results and only clinical symptoms, mortality, etc., are mentioned.

Results

1. The efficiency of "Neguvon" and "Asuntol" as anthelminthics.

(a) *"Neguvon".*

Thirteen animals were used in this experiment, five of which were kept as untreated controls. A "Neguvon" emulsion was dosed at the rate of 110 mg./Kg. with a maximum dose of 20 gm. The mean egg counts were reduced by 94.4% in the treated animals while the controls only fell by 3.8% nine days after treatment.

The effect on the various species is shown in Table No. 1. At the dosage rate of 110 mg./Kg. (5 gm. per 100 lb. body weight) this drug was highly effective against *H. placei* and *O. radiatum* and every effective against *Cooperia* spp. Unfortunately only 2 animals were mildly infested with *B. phlebotomum* and *T. axei* and the results against these two species are not included in the table.

Table No. I.

The efficiency of "Neguvon" administered per os at a dose rate of 110 mg./Kg.* with a maximum dose of 20 gm. against some common internal parasites of bovines.

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Species	No. of animals treated	Percentage reduction in faecal egg counts. No. of animals affected.		
		80–100%	60–80%	Less than 60%
†*Cooperia* spp.	7	6	—	1
H. placei	6	6	—	—
O. radiatum	5	5		

* Live weights assessed.

† *Cooperia* spp. means *C. punctata* and *C. pectinata*.

(b) *"Asuntol"*.

Cattle were dosed at the rate of 15 mg./Kg. with a maximum dose of 5 gm. at Armoedsvlakte Research Station, Vryburg. Faeces were collected from 10 out of 23 animals dosed, as well as from 10 controls on the day of dosing; 6 days later and again 28 days after dosing. There was a 100% reduction in the faecal egg counts of the dosed group six days after dosing, and only one animal showed a much reduced egg count 28 days after dosing and the remaining 9 animals in the dosed group remained negative. The mean egg counts of the control group only fell by 11.6% over the 28 day period.

TABLE No. II.

The efficiency of "Asuntol" administered per os at a dose rate of 15 mgm./Kg.* with a maximum dose of 5 gm. against some common internal parasites of bovines.

Species	No. of animals dosed.	Percentage reduction in faecal egg counts. No. of animals affected.		
		80–100%	60–80%	Less than 60%
**Cooperia* spp.	10	10	—	—
H. placei	9	9	—	—
O. radiatum	7	7	—	—

* Animals weighed before dosing.

† *Cooperia spp.* means *C. pectinata* and *C. punctata*.

In table No. II is will be noted that this drug was highly effective against *Cooperia* spp, *H. placei* and *O. radiatum*. Only two animals had a mild infestation of *T. axei* so that the results against this species were not included in the table.

(c) "Neguvon" 10 parts and "Asuntol" 1 part.

Combination of these two compounds in the proportion of active ingredients of 10 parts "Neguvon" to one part "Asuntol" were dosed to 59 animals of various age groups. Weights of cattle were assessed and subsequently found to be considerably higher than assessed when checked with a weigh-band. These experiments were carried out on four different herds, three in the

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Rustenburg District and one in the Reitz District. Faecal egg counts in animals that were dosed fell on an average, by from 74.3% to 98%. In the control group on one farm egg counts fell by 71.8% but in all other groups rose on an average, by from 6% to 21% during the periods of the experiments.

TABLE No. III.

The efficiency of "Neguvon" 10 parts and "Asuntol" 1 part against **H.** *placei.*

Dosage in mg./Kg.* of "Neguvon" and "Asuntol"	No. of animals dosed.	Percentage reduction in faecal egg counts. No. of animals affected.		
		80–100%	60–80%	Less than 60%
110N* : 11A*	14	14	—	—
88N : 8.8A	10	10	—	—
55N : 5.5A	18	18	—	—
44N : 4.4A	10	10	—	—

* N indicates "Neguvon" and A indicates "Asuntol".
† Weights assessed.

TABLE No. IV.

The efficiency of "Neguvon" 10 parts and "Asuntol" 1 part against *Cooperia* spp. (*C. pectinata* and *C. punctata*).

Dosage in mg./Kg.* of "Neguvon" and "Asuntol"	No. of animals dosed.	Percentage reduction in faecal egg counts. No. of animals affected.		
		80–100%	60–80%	Less than 60%
110N* : 11A*	17	8	1	8
88N : 8.8A	10	7	1	2
55N : 5.5A	22	16	1	5
44N : 4.4A	10	10	—	—

* N indicates "Neguvon" and A indicates "Asuntol".
† Weights assessed.

TABLE No. V.

The efficiency of "Neguvon" 10 parts and "Asuntol" 1 part against *O. radiatum.*

Dosage in mg./Kg.* of "Neguvon" and "Asuntol"	No. of animals dosed.	Percentage reduction in faecal egg counts. No. of animals affected.		
		80–100%	60–80%	Less than 60%
110N* : 11A*	16	14	—	2
88N : 8.8A	9	9	—	—
55N : 5.5A	21	21	—	—
44N : 4.4A	11	10	1	—

* N indicates "Neguvon" and A indicates "Asuntol".
† Weights assessed.

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As can be seen in Tables III and V the combination of "Neguvon" 10 parts and "Asuntol" 1 part was highly effective at all dosage levels against *H. placei* and *O. radiatum*. Against *Cooperia* spp. the results were erratic as shown in Table No. IV, although in 41 animals out of a total of 59, the combination of these compounds was highly effective.

TABLE No. VI.

The efficiency of "Neguvon" and "Asuntol" at a dosage rate of 110 and 11 mg./Kg.* respectively, against *T. axei* and *B. phlebotomum*.

Species	No. of animals dosed.	Percentage reduction in faecal egg counts. No. of animals affected.		
		80–100%	60–80%	Less than 60%
T. axei	10	10	—	—
B. phlebotomum	3	—	—	3

* Weights assessed.

The species *T. axei* and *B. phlebotomum* were present in most animals tested in small numbers only. However, in Table No. VI the results of 10 animals with a moderate *T. axei* infestation are shown and at 110 mg./Kg. of "Neguvon" and 11 mg./Kg. of "Asuntol" the results showed high efficiency for these drugs. This, however, was not the case in the three animals that were affected with *B. phlebotomum*, where the results were most disappointing.

2. *The toxicity of "Neguvon" and "Asuntol".*

(a) *"Neguvon".*

Two animals were dosed at 180 mg./Kg. and a further two at 150 mg./Kg. These were large cross-bred cows whose weight exceeded 400 Kg. These cattle were dosed strictly according to their weight. These animals were kept under observation at Onderstepoort.

A few days later 11 animals varying in age from yearlings to adult cattle and in weight from 100 Kg. to over 400 Kg, were dosed at Armoedsvlakte. They were dosed at the rate of 150 mg./Kg. with a maximum dose of 50 grams.

Within three-quarters of an hour all the animals showed toxic symptoms. At first they were restless and passed small quantities of soft faeces. Symptoms of colic appeared soon thereafter, the animals turning their heads to their flanks, lifting their limbs and kicking at their abdomens and flanks. Salivation and an increased respiratory rate accompanied these symptoms. Some of the animals started walking backwards. Most of the animals lay down and were in obvious pain, twisting their bodies, rolling and pushing their necks under the fence. Most animals rose to their feet when approached, but moved with a stilted and swaying

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action. Muscular tremors were seen in all animals and most of them made grinding noises with their teeth. Within a few hours the symptoms abated and the animals appeared to recover although they did not graze for 12 to 48 hours after being dosed. No mortality occurred.

(b) *"Asuntol"*.

Five animals were dosed at Onderstepoort at a rate of 30 mg./Kg. These animals varied from young weaners to adult cattle. No maximum dose limitation was imposed, one animal receiving 13.1 gm. of "Asuntol".

At Armoedsvlakte a few days later 41 animals were dosed at the rate of 30 mg./Kg. with a maximum dose of 10 mg. Their ages varied from 3 months to just over 2 years and their weights from 70 Kg. to 382 Kg.

One yearling Fries heifer which weighed 127 Kg. and received 3.71 gm. "Asuntol" developed marked toxic symptoms similar to those described above under "Neguvon" poisoning 3 hours after dosing. Salivation, dyspnoea, muscular tremors and colic were marked and the heifer was unable to rise. A subcutaneous injection of 0.5 grain of atropine was administered which relieved the symptoms, but the heifer was unable to rise and died 12 hours later. Apart from cyanosis, subepicardial and subpleural ecchymoses and petechiae as well as hyperaemia and oedema of the lungs, the *post mortem* examination was negative.

Most of the animals dosed reacted only 10 to 14 days and some even 48 hours after dosing. The symptoms were similar to those described for "Neguvon" poisoning but more severe and of longer duration. Although the severe symptoms disappeared after a few hours, the larger proportion of the animals only grazed and appeared clinically normal two to four days after dosing, and a few animals took five to seven days to recover. No further mortalities occurred and no further abnormalities were noted a week after dosing.

A further group of 23 animals were dosed at the rate of 15 mg./Kg. with a maximum dose of 5 gm. at Armoedsvlakte. Apart from a little diarrhoea and slight inappetence 24 to 48 hours after dosing no further symptoms were noted.

At Onderstepoort 100 animals were dosed with "Asuntol" at the rate of 20 mg./Kg. The maximum dose was 6 gm. Only 6 calves received less than the maximum dose; 94 of the animals exceeded 300 Kg. in weight and therefore were given the maximum dose. Similar remarks apply to these animals as to those dosed at the rate of 15 mg./Kg. Transitory mild toxic symptoms were noted after one or two days with no mortality.

Discussion

The experimental results of the field trials described above confirm to a large extent the work of Riek and Keith (1958),

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Herlich and Porter (1958) and Riek (1958). Both "Neguvon" and "Asuntol" were highly effective and potent anthelminthics against *H. placei* and *O. radiatum* at very low dosage rates when combined, and effective against *Cooperia* spp, and *T. axei* at higher dosage rates. "Neguvon" at a dosage rate of 110 mg./Kg. did not appear to be as effective as "Asuntol" at a dosage rate of 15 mg./Kg. against *Cooperia* spp.

According to Riek and Keith (1958) and Riek (1958), "Neguvon" was highly effective against *B. phlebotomum* at dosage rates of 110 mg./Kg. as well as against *Cooperia* spp. Although not confirmed in our observations, it must be remembered that live weights were assessed when "Neguvon" was used as an anthelmintic, and *Bunostomum* infestations were so light that no conclusions could be drawn from the results.

Where "Asuntol" was used the animals had been weighed on a weighbridge and the weights were known, which resulted in more accurate dosing. This may account for the close agreement with Herlich and Porter's (1958) observations on *Cooperia* spp., *H. placei* and *O. radiatum*.

There appears to be no object in combining these drugs for higher efficiency. They appear to be equally effective against *H. placei* and *O. radiatum* at low dosage rates. The efficiency of "Neguvon" against *B. phlebotomum* and *Cooperia* spp. according to Riek and Keith (1958) and Riek (1958), is noted only at dosage rates of 110 mg./Kg. The results where "Neguvon" and "Asuntol" were combined must therefore have been due largely to the latter as far as the effect against *Cooperia* spp. was concerned at low dosage levels. This was confirmed where "Asuntol" was dosed at the rate of 15 mg./Kg.

The toxicity of these drugs, however, differed from those recorded in the literature. The relatively low toxicity of "Neguvon" confirmed Riek and Keith's (1958) observations. Their recommended maximum dosage rate of 5 gm. per 100 lb. body weight (110 mg./Kg.) was quite safe under field conditions, as toxic symptoms, though marked, caused no mortality at higher dosage rates, i.e. 6.6 gm. per 100 lb. body weight (150 mg./Kg.).

"Asuntol" was dosed to over 100 animals at the rate of 20 mg./Kg. with a maximum dose of 6 gm., and to 37 animals at the rate of 15 mg./Kg. with a maximum dose of 5 gm., with mild toxic symptoms and no mortality. Only at 30 mg./Kg. were symptoms severe and one heifer died out of a total of 46 animals dosed. Riek's (1958) observation that 0.25 gm. per 100 lb. body weight or 5.5 mg./Kg. was the maximum safe dose, did not agree with the results reported above. Herlich and Porter (1958), however, reported only mild toxic symptoms 12-24 hours after dosing at the rate of 25 and 12.5 mg./Kg. Our observations were more in agreement with those of the latter authors.

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In view of the small numbers of animals dosed, it is felt that care must be exercised in the use of these drugs. The results have proved the value of these drugs as anthelminthics. At a dose rate of 5 gm. per 100 lb. body weight (110 mg./Kg.) "Neguvon" is effective against *Cooperia* spp. and *B. phlebotomum*. At 2 gm. per 100 lb. body weight (44 mg./Kg.) this drug is effective against *H. placei* and *O. radiatum*. At these dosage rates the drug is apprently safe for use.

"Asuntol" is a safe and effective anthelminthic at a dose rate of 0.67 gm. per 100 lb. body weight (15 mg./Kg.) with a maximum dose of 5 gm. against *Cooperia* spp., *H. placei* and *O. radiatum*.

Under field conditions, where animals cannot be weighed, it is advisable for veterinarians to use a weighband* and measure the heart girth measurement, which is a more accurate method of assessing weight than by merely looking at an animal. Bonsma and Neser (1951) have drawn up tables which may be referred to and in the authors' and others' experience these weights are extremely accurate when checked against the actual weight on a weighbridge. (Farming in South Africa, November, 1951, Reprint No. 93.) According to Van Marle (1959) the mean difference between the actual weight of cattle and the assessed weight using heart girth measurement is as little as 3.2% with a maximum error of 20%.

Summary

1. "Neguvon" is highly effective against *Cooperia* spp. and probably also against *B. phlebotomum* at dose rates of 5 gm. per 100 lb. body weight (110 mg./Kg.). It is a highly efficient anthelminthic against *H. placei* and *O. radiatum* at a dose rate of 2 gm./100 lb. body weight.

2. "Asuntol" is a safe and effective remedy against *Cooperia* spp., *H. placei* and *O. radiatum* at a dose rate of 0.67 gm. per 100 lb. body weight.

3. Toxic symptoms were noted at dosage rates of 6.75 gm. per 100 lb. body weight (150 gm./Kg.) with "Neguvon" and 1.34 gm. per 100 lb. weight (30 mg./Kg.) with "Asuntol."

Acknowledgements

Our thanks are due to the Director of Veterinary Services and the Chief, Division of Animal Husbandry and Dairying for facilities to carry out these investigations. We particularly wish to thank Mr. van Marlé, Officer in Charge, Armoedsvlakte, for placing nearly 100 animals at our disposal for toxicity and anthelminthic trials and to the various farmers who helped us with the

* Weighbands are measured off in cm. with the corresponding weights marked on the band.

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field trials. Finally our thanks are due to Drs. Andersen and Stampa of Agro-Chem., S. Africa Ltd., for the unlimited supplies of "Neguvon" and "Asuntol" and their helpful suggestions.

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THE PROFESSIONAL PROVIDENT SOCIETY OF SOUTH AFRICA

REPORT OF THE BOARD FOR THE YEAR ENDED 31ST DECEMBER, 1958

The above report and balance sheet is to hand.

It is surprising to note that only 14 veterinarians are members out of a total of 1,436. We feel that many of our members do not fully appreciate the benefits they may derive from this society and urge them to consider the matter of joining very seriously.

The assets of the Society are shown at £373,229 and the administrative costs are only 5.77% of the income, which was £112,364 for the year under review.

ADDITIONAL BENEFITS

The New Group Life Assurance and Hospitalisation schemes introduced towards the end of the year met with a most encouraging and satisfactory response. As the time of writing 1,019 members had subscribed for the Group Life Assurance scheme with an aggregate sum assured of £4,249,600 and 1,044 members with 3,063 dependants had joined the hospitalisation scheme. Up to date one claim of £5,000 has been paid out under the assurance scheme, whilst benefits paid out under the hospitalisation scheme already amount to many hundreds of pounds. These optional benefits are most advantageous additions to the already splendid benefits afforded by membership. Those who have not taken up these benefits should give serious consideration to doing so and, if necessary, refer to the Society for any clarification or explanations required. Existing members at the 1st November, 1958, the date of commencement of the schemes, have now been given up to the end of May to join the schemes without the need for submitting proof of insurability.

Judging by the popularity of the hospitalisation scheme, a full medical insurance scheme would fill an urgent need and should meet with instant success. The Board is at present investigating this matter and should be in a position to offer members a most attractive scheme within the course of the next few months.

PENSION FUND

After making representations to the Minister of Finance for tax concessions to self-employed persons on contributions for pension purposes, your Board initiated the appointment of a committee representative of some eight professional organisations to continue negotiations with he Commissioner for Inland Revenue. We are confident that the desired amending legislation will not be long delayed and a suitable pension scheme will be launched as soon as possible after the Income Tax Act has been amended.

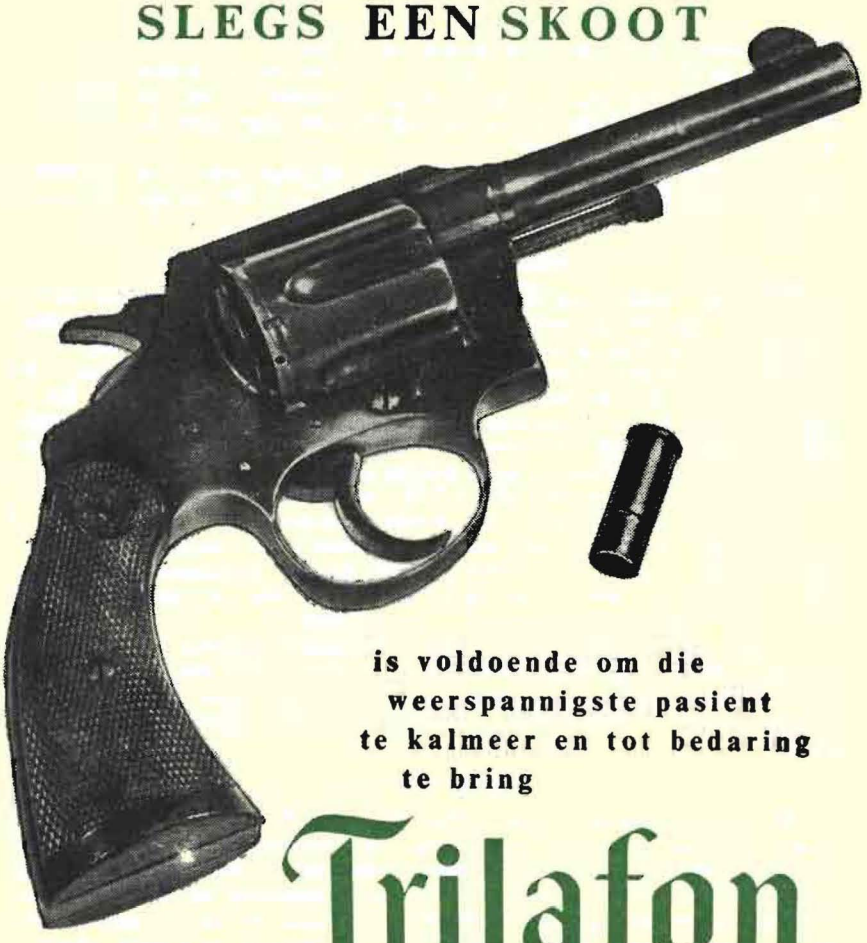
GENERAL

With its splendid record of achievement ,the Society may well look to the future with every confidence. It affords its members and their dependants a measure of protection not previously enjoyed by professional persons. Of paramount importance is the fact that this organisation is run by the professions for their own benefit, with the interests of members its first consideration. Its growing strength ensures that it will be able to add yet further to the comprehensive protection already offered and that it will continue to contribute greatly to the security and stability of the professions in this time of stress and uncertainty.

The address of the Society is: 903 Medical Arts Building(P.O. Box 6268), Jeppe Street, Johannesburg.

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SLEGS EEN SKOOT



is voldoende om die
weerspannigste pasient
te kalmeer en tot bedaring
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Jl. S.A.V.M.A. 30 (2), 1959

A COMPARISON OF DILUENTS AND STORAGE TEMPERATURES FOR SWINE SEMEN

D. H. G. IRWIN

Department of Surgery, Faculty of Veterinary Science, Onderstepoort

Introduction

As a step towards the establishment of a swine artificial insemination centre at Guelph, Ontario, investigations were carried out in 1957 to determine which of several diluents and which storage temperatures were most satisfactory in maintaining moility in stored boar semen.

Diluents and Storage Temperatures

The diluents employed included some previously described (Roy, 1955 and Salisbury *et al*, '41) and others based on sterile, homogenized, whole milk. The latter group was found to hold promise in an earlier series of trials (Barker, 1956).

The composition of the diluents used was:-

1.	Egg-yolk-citrate	Yolk	30 ml.
		Sodium citrate	3 gm.
		Distilled water	to 100 ml.
2.	Egg-yolk-milk	Yolk	30 ml.
		Milk	to 100 ml.
3.	Milk	Milk	100 ml.
4.	Glycine yolk-water	Glycine	2 gm.
		Yolk	30 ml.
		Water	to 100 ml.
5.	Glycine-yolk-milk	Glycine	2 gm.
		Yolk	30 ml.
		Milk	to 100 ml.

Yolk was prepared from fresh eggs by excluding white and yolksac, and beating in a Waring Blendor for two minutes at low speed. Milk was prepared by pasteurising homogenized whole milk as sold to the public by a commercial dairy in Guelph.

Semen in these diluents, and undiluted control semen, was store at 5°, 12° and 20°C.

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Donors and Collection

The two donors used in this preliminary trial were a 35-month-old Yorkshire board, and a 24-month-old Landrace boar, both of whose fertility was proved, the former by natural service and the latter by artificial insemination (Barker).

Artificial vaginas with 500 ml. polythene collecting vials similar to those described by Polge were used for the collection of semen. Into the collecting cone of the artificial vagina used on the Yorkshire board a plastic filter was fitted, as used by Norwegian and Swedish workers. Artificial vaginas were equipped with foam rubber in the distal end, so as to simulate the cervix. In addition the glans, when it protruded beyond the artificial vagina during maximal erection, was gently grasped manually through the latex lining. No pulsating action on the liners was employed and no lubricant was used. Collections were made twice weekly from June to early August. A slight drop in semen filtrate volume occurred towards the end of this time.

Procedure

Semen from the Landrace was filtered through filter paper. Dilution was standard throughout, being one part of semen to four parts diluent, as this was found by Barker to be satisfactory. All samples were stored in sterile screw-cap vials, at the three holding temperatures. Young *et al* (1957) used the same dilution rate and compared, *inter alia*, corked versus sealed storage vials.

To diluted samples and controls alike penicillin and streptomycin were added since these antibiotics were recommended for this purpose by Coronel and Masankay-Arenas (1954).

The stored vials were inverted gently several times before two or three drops were withdrawn with sterile pasteur pipettes. Any two samples to be examined that day were chosen in random pairs. The fluid from each sample was placed on one glass slide, in separate drops and each covered with a cover slide. The examination was done on the electrically warmed stage described by MacPherson (1957) under the low power of a microscope. The drops were examined alternatively several times during a two minute period on the warm stage, and the highest motility noted was recorded for each.

Evaluation

An arbitrary scale of motility was adopted ranging from a maximum designated 7, to no motility designated 0. The average readings of at least six different samples from each of the two boars (i.e. at least 12 samples) was taken as the figure from which the histogram was compiled.

Conclusion

Over a three day storage period, milk at 5° and yolk milk at 5° and 20° seem to be the best of the environments tested for

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maintaining motility in swine spermatozoa. Motile spermatozoa were observed 19 days from collection date in swine semen diluted in yolk-citrate (in one out of 12 samples) and 14 days from collection date (in 3 samples out of twelve) in milk diluent. These observations refer to samples kept at 12°C, see Table 1.

The histogram shows something peculiar. This is a "recovery phase" or increase from a certain level of motility to a higher rating on a subsequent date. This phenomenon is manifest by semen diluted in milk at 5° on the 7th day, and on the 10th day at 12°C and by semen diluted in yolk glycine-water on the 4th day at 5°C and on the 5th day at 12°C.

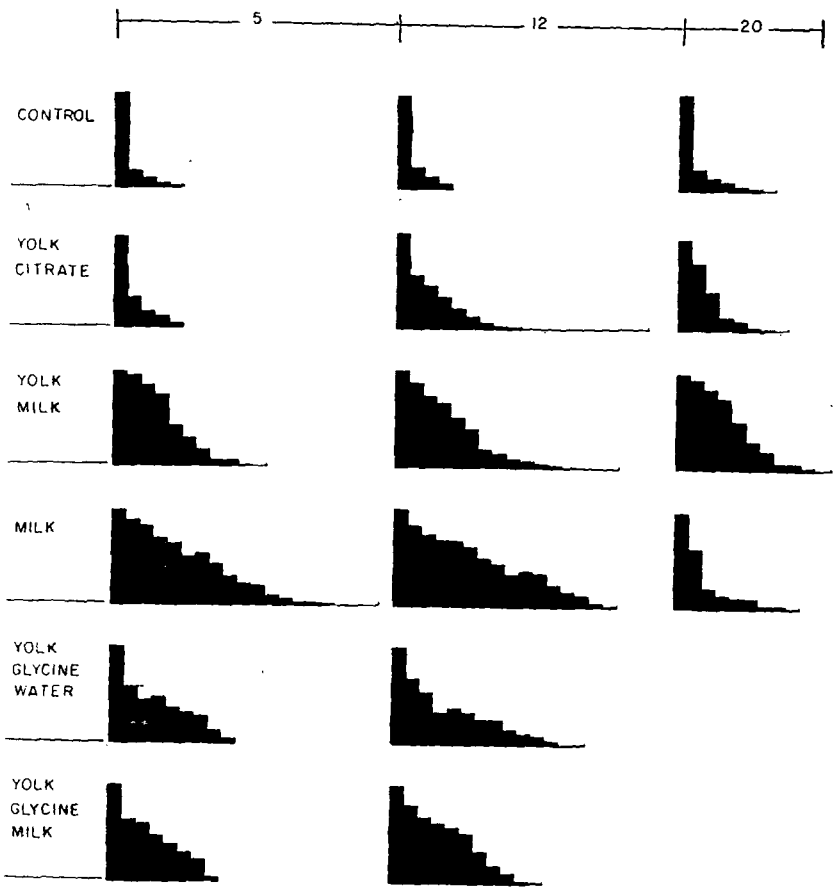


TABLE NO 1

Histogram depicting daily motility in swine semen stored in different temperatures and diluents. The general pattern is a daily drop, exceptions being seen in milk at 5° and 12°C on the 7th and 10th days in glycine yolk-water, and at 5° and 12°C on the 4th and 5th days respectively.

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Discussion

(a) *Motility as criterion of storage environment.* Of available laboratory techniques, motility was shown to bear the closest correlation to fertility in bull semen (Cummings, 1954). There does not appear to be much correlation between motility and fertilizing ability in ram, boar and fowl semen (Danzier, 1956, and Polge, 1956). Nevertheless, motility was chosen as the criterion for comparison for want of an agreed superior one for swine semen evaluation.

(b) *Bacterial Contamination.* The factor of bacterial contamination is felt to be of great importance in the longevity of swine spermatozoa in vitro, particularly at the holding temperature of 20°C. This is in agreement with a similar supposition expressed by Dilliello, Poelma and Faber (1957), relating to bovine semen. The principle incidental contaminants encountered in these trials were *Escherichia coli* and *Pseudomonas,* neither of which were adequately controlled with penicillin and streptomycin. The addition of neomycin, chloramphenicol or aureomycin was suggested by plating semen from our boars on sheep-blood-agar with sensitivity discs. However, the cleaning of the boar's belly and prepuce, surgical removal of the preputial diverticulum and making every other effort in attempting to collect an aseptic sample of semen should be investigated before antibiotics are fully evaluated in this regard.

The Yorkshire board was less adept in gaining intromission than the Landrace, and this is the likely reason why semen collected from the latter boar remained motile longer than that from the former, since contamination is more likely to result from a penis which has touched the dummy and outside of the artificial vagina before intromission. This is especially true when the semen was stored at 20°C.

Summary

The report of a preliminary trial on the maintenance of motility of swine spermatozoa in various diluents, holding temperature of 5°, 12° and 20°C, is presented. Bacterial contamination complicates evaluation. Yolk-milk and milk were the most promosing of the diluents tested. There was little to choose between holding temperatures of 5° and 12°C, with 20°C being roughly equal to these in the case of yolk-milk diluent.

Acknowledgements

This pilot test was done in Guelph by kind permission of Dr. T. Lloyd-Jones, Principal of the Ontario Veterinary College, under the encouraging supervision of Professor C. A. V. Barker, at whose suggestion the work was undertaken. Sincere thanks

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are extended to these gentlemen as well as to Professor J. Ballantayne and Dr. MacPherson, O.V.C., for many favours in this connection.

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SLEEPY FOAL DISEASE IN NATAL

A. LITTLEJOHN
Mooi River

The clinical syndrome known as "Sleepy Foal Disease" has been recognised in various horse breeding areas in Europe and in America since the turn of the century, if not before. Belonje(1) states that *B. viscosum equi* is responsible for one-third of all foal deaths in the Newmarket area in England but that the position in South Africa is unknown. Dimock, Edwards and Bruner(2) found that 31.5% of all foal deaths in a series of 810 deaths investigated at the Kentucky Agricultural Experiment Station were attributable to *B. viscosum equi.*

Etiology: B. viscosum equi, a Gram negative ovoid non-sporing organism 1.5 x 0.8 μ.

Infection: The natural method of infection is unknown. The disease has been reproduced by the intravenous injection of *B. viscosum equi* into foals 3–7 days old(2). The organisms have also been demonstrated in the intestinal tract and in the tonsillar area of normal adult horses. The infection has also been demonstrated in the foetus(2).

The organism must therefore be regarded as a natural inhabitant of the digestive tract of normal adult horses and possibly also of many foals. Clinical evidence suggests that infection may be ante-natal or post-natal, by oral route or through the umbilicus. Attempts to reproduce the infection as a pre-natal disease in foals by the introduction of organisms into the uteri of three mares proved unsuccessful(2). Maguire(3) is of opinion that the acute infection in new-born foals ———— is undoubtedly a pre-natal invasion and that 90% of foals born to carrier mares are diseased at birth and will reveal typical signs of infection at a very early age.

Symptoms: In a series of 23 recorded cases of S.F.D. seen by the writer, symptoms were, without exception, seen within 48 hours of birth. Clinically, peracute, acute, and subacute cases were recognised. In four cases which can only be described as peracute, the onset of symptoms was violent, sudden, and within 24 hours of birth. The first symptoms shown were those of colic, the foal going down, rolling, kicking and showing classic signs of internal pain. Purging began soon after, followed by great depression, protration and death. In two of these peracute cases the faeces were haemorrhagic shortly before death. Two of the

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four peracute cases (4 and 5) died four hours after the onset of symptoms, the other two (6 and 7) dying six and twelve hours respectively after the onset of symptoms. This gives an indication of the extreme virulence of the infection. The fact that peracute cases have not been seen by the writer since 1955 can not be explained. Acute cases showed none of the violent symptoms described above. The symptoms were characteristic extreme lassitude, fever, evil-smelling diarrhoea occasionally blood-stained, injected mucous membranes and a disinclination to suckle. An almost pathognomonic sign is a half-hearted attempt to suckle; after a few moments the sucking movements cease (although the teat is still in the foal's mouth) and the eyelids droop and close. Then the head falls away and the foal stands with head down and eyelids closed. Hence the name. An almost constant symptom which does not appear to be mentioned in the literature is the passing of small amounts of urine at frequent intervals, accompanied in many cases by a certain amount of irritation. This is possibly due to a rapid decrease in the pH of the urine. (*B. viscosum equi* is an acid-producing organism in vitro.)

Subacute cases showed the above symptoms to a milder degree, usually on the second day after birth.

Pathology: Autopsies were performed on four cases. All four showed signs of an acute systemic infection, with petechial haemorrhages on peritoneum, pleura, epi- and endocardium, and degeneration of the liver and kidneys. A haemorrhagic enteritis was present in all cases, so marked in No. 5 that the jejunum contained pure blood for two feet of its length, and the ileum and colon contents were markedly bloodstained. A curious feature of this case was that the urinary bladder was extremely haemorrhagic and partially digested, and was completely ruptured as a result of the histolysis. Very little urine was present in the peritoneal cavity. This autopsy was carried out within one hour after death, so that post mortem changes could be ruled out. This case died within 24 hours of birth. The histolysis of the bladder bore a marked resemblance to that seen by the writer in a case of anthrax in a bovine which was inadvertently opened.

In No's. 5, 6 and 10, typical lesions could be observed macroscopically in the kidneys as pinhead-sized abscesses just visible on the cut surface.

In Nos. 6 and 10, diagnosis was confirmed by the bacteriological and pathological examinations of specimens submitted to Onderstepoort Laboratory. In No. 4, a bacteriological diagnosis could not be made from specimens submitted to Allerton Laboratory, but the pathology was regarded as indicative of *B. viscosum equi* infection. Specimens from No. 5 were not submitted.

Treatment: Leader([4]) emphasised that very early diagnosis and treatment is the best line of attack, and indicated streptomycin as the antibiotic of choice. Belonje([1]) also advocates streptomycin

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as early as possible in the course of the disease. Maguire[3] states that streptomycin is valauble especially if combined with blood transfusion from the dam, but that terramycin has been spectacularly successful in treating subacute and chronic *B. viscosum equi* infections in adult horses.

As can be seen from Table 1, during the 1956, 1957 and 1958 breeding seasons, chloromycetin (P.D. & Co.) was used almost exclusively intramuscularly and per os, with consistently good results. Response to treatment with chloromycetin was found, with one exception, to be quicker than with other antibiotics, a factor of prime importance.

TABLE 1.

Case No.	Year	Course	Treatment	
1.	1953	Subacute	Terramycin 330 mgm. intramuscularly for 3 consecutive days.	Recovered
2.	1953	Subacute	Terramycin 330 mgm. intramuscularly for 3 consecutivev days.	Recovered
3.	1954	Acute	Terramycin intramuscularly 500 mgm. 21.8.54 Terramycin intramuscularly 500 mgm. 22.8.54. Streptomycin intramuscularly 4 gm. Terramycin per os 500 mgm. b.i.d. 23.8.54.	Recovered Very Weak Foal
4.	1955	Peracute	Dead on arrival.	Autopsy
5.	1955	Peracute	Dead on arrival.	Autopsy
6.	1955	Peracute	Terramycin intravenous Dextr- Saline intravenous	Autopsy
7.	1955	Peracute	Terramycin intravenous Blood transfusion	Died No Autopsy
8.	1955	Subacute	Terramycin intramuscularly 330 mgm. for 3 consecutive days.	Recovered
9.	1955	Subacute	Streptomycin 4 gm. intramuscularly daily, plus 500 mgm. aureomycin every six hours.	Died No Autopsy
10.	1956	Acute	Chloromycetin per os 500 mgm. every six hours.	Died Autopsy
11.	1956	Acute	Chloromycetin 1 gm. intramuscularly daily for 3 consecutive days. 500 mgm. every twelve hours.	Recovered
12.	1956	Subacute	Chloromycetin per os	Recovered
13.	1956	Subacute	Chloromycetin i.m. and per os.	Recovered
14.	1957	Subacute	Chloromycetin i.m. Streptomycin per os.	Recovered
15.	1957	Subacute	Chloromycetin i.m. and per os.	Recovered
16.	1957	Subacute	Chloromycetin i.m. and per os.	Recovered
17.	1957	Subacute	Chloromycetin i.m. and per os.	Recovered
18.	1957	Acute	Chloromycetin i m. and per os.	Recovered
19.	1957	Acute	Chloromycetin i.m. and per os.	Recovered
20.	1957	Subacute	Chloromycetin i.m. and per os.	Recovered
21.	1958	Subacute	Chloromycetin i.m. and per os.	Recovered
22.	1958	Acute	Chloromycetin i.m. and per os.	Recovered
23.	1958	Subacute	Chloromycetin i.m. and per os.	Recovered

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From the writer's limited experience, chloromycetin emerges as the antibiotic of choice in acute and subacute cases. In the peracute case the intravenous route must be preferred with terramycin, the antibiotic of choice, supplemented by chloromycetin intramuscularly and per os. Energetic supportive measure must be taken in the peracute case until the antibiotic is able to overcome the infection. These measures include blood transfusion, dextro-saline, antihistaminics. In general, the earlier a case is treated, the more favourable the prognosis. The dosage rate of chloromycetin was 20 mgm./Kg. intramuscularly once daily plus 500 mgm. orally twice daily. No deleterious effects were ever observed as a result of treatment, and treatment was never required for more than four consecutive days.

Case No. 10, which died in spite of treatment, had been treated for a day by the stud manager with sulphonamides, and was already in a poor state when seen. Records of some cases in 1954 and 1953 are not available.

Prevention: As Belonje[1] points out, since the exact mode of infection is unknown, it is difficult to suggest a method of prevention and control. The importance of good breeding hygiene in reducing the incidence of the disease is stressed. Maguire[3] has, since 1914, used a vaccine, prepared from *B. viscosum equi*, injected weekly for six weeks into pregnant mares beginning at the eighth month of gestation. Satisfactory prophylaxis has been obtained in 95% of cases.

In 1958 two studs in which the infection had been demonstrated were advised by the writer to give every foal born chloromycetin per os during the first two days of its life. The dosage recommended was 500 mgm. twice daily. In neither of these two studs were further cases of S.F.D. observed during the 1958 breeding season. It would therefore appear that prevention by this method is effective. No practitioner is entitled to suggest that the administration of antibiotics to every thoroughbred foal born should be a permanent and desirable feature of stud management. Nevertheless, in the absence of immunological prophylaxis, it should be advised in the studs where the disease is prevalent.

In conclusion several points may be noted in the control of this disease. Firstly, *B. viscosum equi* has been demonstrated to be very widely distributed and commonly present in the digestive tract of normal adult horses. Secondly, the antigenic characters of variant strains differ markedly from strain to strain[2], thus ruling out any vaccines other than autogenous. Thirdly, specific agglutinins have not been detected in the sera from dams of infected foals[2].

Since all mares can be regarded, for all practical purposes, as harbouring the organism, it follows, in the author's view, that if all foals are not infected with the organism prenatally, they are sure to ingest the organism soon after birth. The question

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of pre- or post-natal infection then becomes of secondary importance, and the question of what "triggers-off" the acute fulminating infection in a new-born foal assumes primary interest.

The writer's experience supports the observation of Leader(4) that poor, weak foals are more often affected than strong foals. Nutritional factors can therefore be incriminated in the etiology of the disease, and it may well be that the dam transmits to the foal a congenital inability of the reticulo-endothelial system, to manufacture antibodies speedily enough and in sufficient quantities to repel invasion by the facultatively pathogenic *B. viscosum equi.*

The question whether we as clinicians are doing the thoroughbred breeding industry a disservice by keeping alive foals which in turn breed more susceptible foals, is one which is not likely to loom large in the eyes of the breeder, and in any case can only be decided by posterity.

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Jl. S.A.V.M.A. 30 (2), 1959

ON THE TECHNIQUE OF REPAIR OF THE POSTERIOR CLEFT PALATE

C. F. B. HOFMEYR
Department of Surgery, Faculty of Veterinary Science
Onderstepoort

RESUME

(1) A brief description is given of the usual method of repair of cleft palate.
(2) The reason why this technique is not applicable in clefts of the posterior hard palate is stated.
(3) A new technique involving an autograft from the uvula is described for repair of the latter type of cleft palate.

Repair of the cleft palate is not frequently referred to in veterinary literature. One reference to operation for this condition in animals could be found in the literature of the last decade.

The accepted technique is an incision just inside the cheek-teeth between these and *A. palatina major*. After freshening the edges of the cleft, the muco periosteum is separated from the palatine process of the maxilla. These flaps are united by sutures in the midline and the now gaping wounds at the site of the first incisions allowed to heal by granulation [Amman (1957)].

As *A. palatina major* emerges opposite the first molar tooth at the anterior palatine foramen this technique is impractical and dangerous for clefts in this region and posterior to it.

It is believed that the technique about to be described is new and enables the surgeon to affect safe closure.

A cleft in the posterior palatine region is usually congenital, but it can be acquired. When a small dog is attacked by a big one, he often cries out with wide open mouth. It then happens that the big dog can grasp the maxilla of his victim in his mouth so that the mandibular canine teeth penetrate the palate and cause an oro-palatine fistula. Most of these cases seen showed the fistula far back. Other cases with a complete median division of the palate can be partly repaired by the method described above. After healing the technique to be described can be followed to effect closure of the posterior part of the opening.

With regard to the genetic basis of this complaint it is necessary for the surgeon to warn his client against breeding with

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any cases showing the congenital defect. In the case of household pets the operation is advisable because of the unpleasant return of food through the nose, causing sneezing, and often, chronic rhintis.

CASE REPORTS:

CASE (1).

Subject: Dachshund dog of 18 months old in good general clinical condition. In the posterior third an oro-nasal fistula was present slightly off the median line (probably acquired) and measuring 2.5 x 1.5 cm.

Operation: Under Sagatal (M & B) anaesthesia the edges of the cleft were freshened. With the knife a mucous membrane flap was outlined as follows: The base was dorso-anterior to the tonsil on the one side, the posterior incision starting somewhat

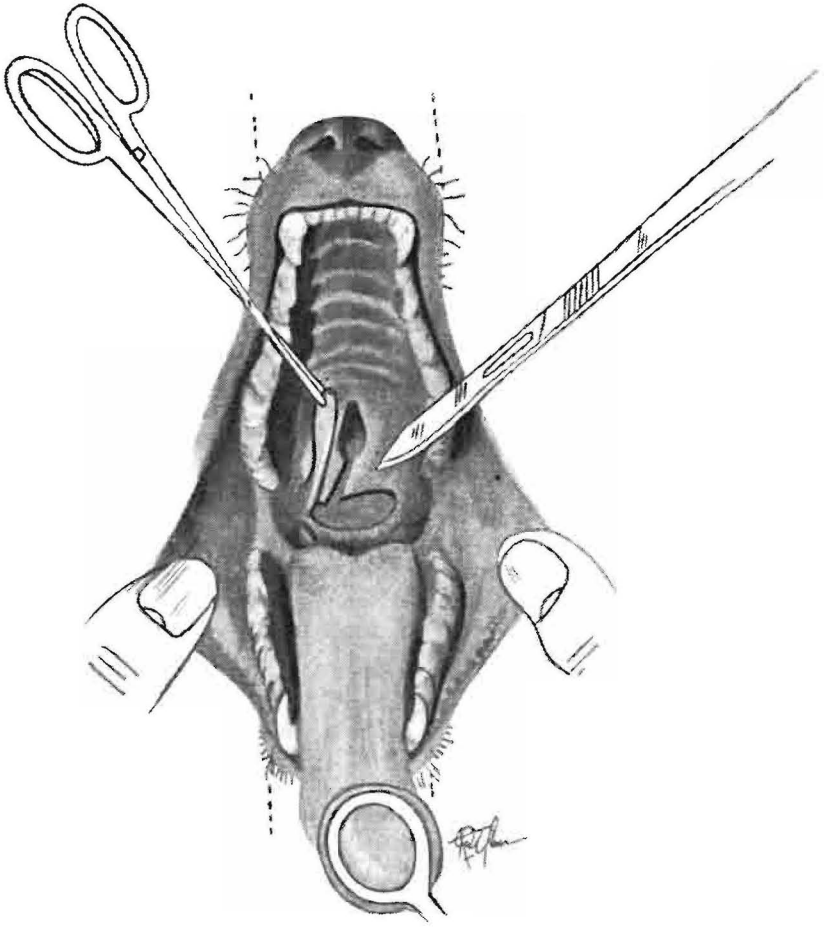


Fig. 1.
TECHNIQUE OF REPAIR OF POSTERIOR CLEFT PALATE

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more medial than the anterior incision in order to preserve the blood supply. The rest of the graft was taken from the soft palate right across it to anterior to the opposite tonsil (Fig. 1). The mucous membrane was dissected off the soft palate, taking as much sub-mucous tissue with it as possible, but avoiding to go so deep as to puncture the mucous membrane of the naso-pharynx. An incision through the muco-periosteum was now made from the posterior extremity of the cleft to the base of the graft. The muco-periosteum was dissected off laterally to make a bed for the pedicle of the graft, which was then rotated and sutured to edge of the muco-periosteum of the prepared bed and of the cleft. Interrupted silk sutures were used.

After the operation the tip of the graft was noticed to be

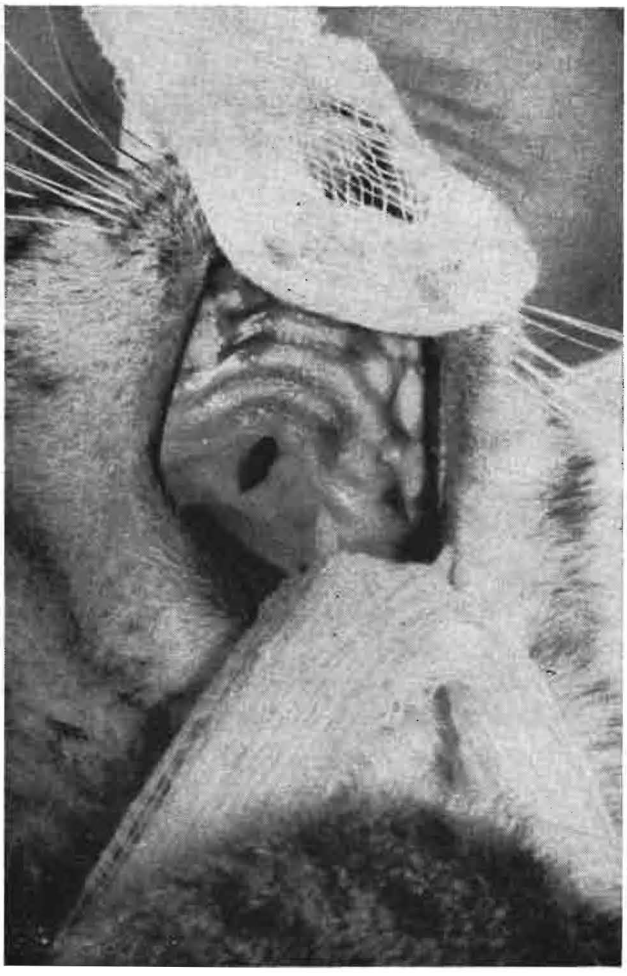


Fig. 2.
THE LESION BEFORE OPERATION

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slightly cyanotic. The autotransplant healed very well except for the very apex, where a small cleft persisted. It was attempted to close this cleft by using a swing graft from the muco-periosteum on two subsequent occasions, but this did not materially reduce the remaining cleft. The reason is that the graft had to be very limited in size in order to avoid the palatine artery at the anterior palatine foramen. The lack of elasticity of the muco-periosteum also militated against a good fitting suture line without tension. The final result was a cleft of 0.5 x 0.3 cm. The return of solids stopped and of fluid was limited.

It is considered that complete closure of the cleft would have been permanent but for a little too much tension on the pedicle leading to poor circulation and non-union of the apex of the graft.

CASE 2.

This was a tom-cat of one year old — clinically normal except for a median cleft in the posterior part of the palate. In relation to the size of the palate the cleft was slightly smaller than the one in the dog.

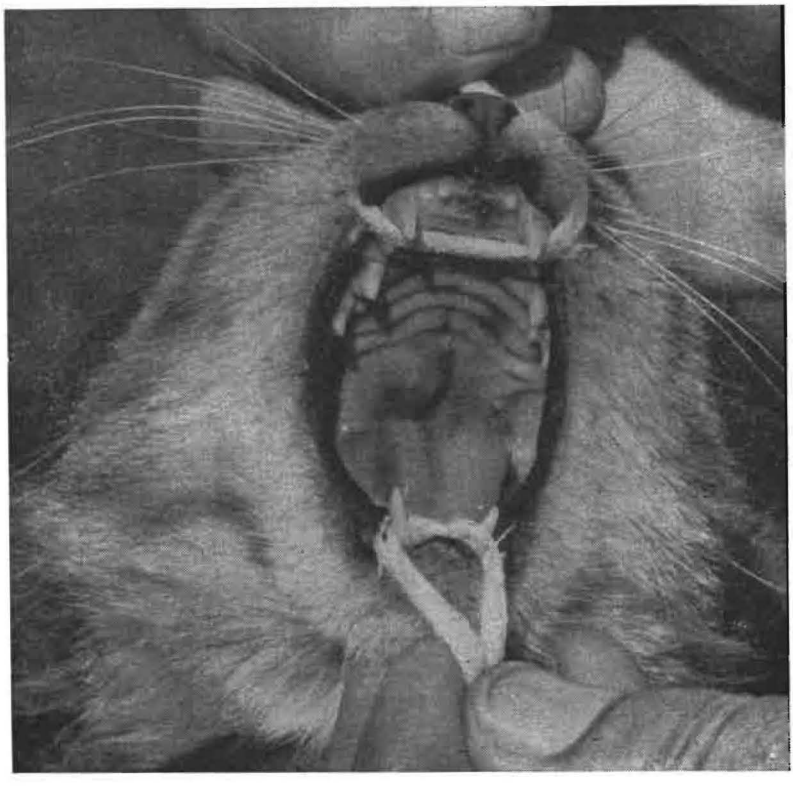


Fig. 3.
AFTER OPERATION

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This operation was also performed under Sagatal. Great care was taken to ensure, if anything, too big a soft palate flap. Otherwise the operation was the same as the one described. Complete healing took place without complication (see Figs. 2 and 3). As in the first case no attempt was made to suture the wound in the soft palate.

Discussion

The technique as described can obviously only be applied in the area that can be reached by a flap derived from the soft palate i.e. the maximum length of this graft is the width of the soft palate. This is not much forward from the first molar. As this is the area where the muco-periosteum cannot be effectively mobilised, the autograft should fill a need.

Acknowledgement

The author wishes to express his appreciation to Mr. R. B. Trengrove, a final year student, for making the very fine drawing, and to Mr. A. M. de Bruyn for the photos.

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SUPPORTIVE TREATMENT IN CALF ENTERITIS

K. VAN DER WALT

Onderstepoort

SUMMARY

The effects of enteritis in the body are enumerated and general principles of treatment are discussed. Administration of electrolyte fluids, Vitamin A and other vitamins, minerals and proteins is recommended. The correction of intestinal flora by the feeding of sour milk is stressed.

With the introduction of sulphonamides, antibiotics and furazolidone, the treatment of gastro-intestinal infections in calves has become easier and mortality has decreased spectacularly.

With more specific treatment there is a tendency to rely solely on the chosen drug to the exclusion of supportive treatment. Re-emphasis of this important part of the treatment does not seem out of place as the speed of rehabilitation of a sick calf is directly proportional to subsequent weight gain and performance. In fact, failure to restore the animal to normality in a short time often results in the feeding of an uneconomic runt.

Intelligent application of supportive after-care should be based on the recognition of the following fundamental changes which take place in any severe attack of enteritis regardless of the primary cause.

1. Rapid loss of energy through the refusal of food and the ability to absorb food.
2. Shock due to histamine and toxin release from the inflamed intestine which, after absorption, has a profound influence on all body tissues.
3. Dehydration which affects organ function, elimination via the urinary system, and, through haemoconcentration, slows the circulation and embarrasses the heart.
4. Loss of electrolytes with impairment of vital physiological functions through a dangerous shift of body electrolytes.
5. Depletion of vitamin, mineral and protein stores which persist after clinical recovery.
6. Specific antibacterial drugs either eliminate or bring about profound changes in the normal gastro-intestinal flora.

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7. A combination of some or all the above-mentioned changes cause stress and can eventually precipitate fatal shock in spite of specific antibacterial treatment.

Principles of Treatment

1. Correct diagnosis of the cause of enteritis is essential during the first examination. The course is always rapid and any delay in the selection of the specific antibacterial drug may prove disastrous.

2. The state of stress or shock determines the choice of supportive treatment. For the not-so-ill calf an injection of an antihistamine drug combined with the administration of a litre of dextrose-saline containing 5–10 ml. absolute alcohol, is often sufficient.

More frequently, however, the animal appears extremely dull, uninterested, and obviously ill. In these cases the metabolic stimulation and antistress action of the adrenocorticoids is most rewarding. Prednisolone (delta-hydrocortisone) at the rate of 2 mg./Kilo bodyweight is preferred to prednisone (delta-cortisone). In the shocked and moribund calf the administration of hydrocortisone hemisuccinate (30–50 mg.) intravenously is often life-saving.

3. The supply of energy, correction of dehydration and electrolyte balance, and the treatment of toxaemia are best accomplished by giving a blood transfusion with the concurrent administration of 5%–10% dextrose in physiological saline.

Using 3 grams sodium citrate dissolved in 100 ml. of physiological saline as an anticoagulant, a litre of blood is drawn (preferably from the dam) and administered intravenously through a filter-drip apparatus. Twelve to fifteen minutes are taken over the administration of a litre of blood In the dehydrated animal dextrose saline solution (one litre intraperitonally) should be given in conjunction with the blood. This ensures that the fluid balance is corrected with the aqueous solution and enables the body to utilise the nutritional and protective properties of the plasma proteins and antibodies without using it primarily for hydration.

Although some more elaborate solutions are in use, physiological saline with 5% or 10% dextrose added, adequately restores electrolytes lost through the bowel and supplies readily available energy. It is easiest to give the slightly warmed solution intraperitonally.

A warning should be given about the thoughtless administration of concentrated dextrose solutions in dehydrated animals. Any highly concentrated solution draws more fluid from the tissues before it can be utilised and this action can have fatal consequences.

Dosing barley water, strained oatmeal or limewater by mouth, especially as a vehicle for drugs, assists in combating dehydration. At no time is it advisable to withhold clean drinking water from a calf suffering from enteritis.

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4. The use of astringents and emollients combined with specific chemotherapy shortens the course of diarrhoea and minimises fluid loss. The incorporation of an antacid such as sodium bicarbonate is of value in the beginning of treatment. Drugs of value are carron oil, tannic acid, tinct. opii, chlorodyne (Tinct. Chlorof. et Morph. B.P.), bismuth subnitrate, kaolin, pectin, etc. Largactil (1 mg./Kilo) is extremely valuable in preventing severe straining during active diarrhoea. This drug can be used even in severely ill animals.

5. Depletion of the never abundant Vitamin A store in the body takes place rapidly with enteritis. Failure to recognise this fact is undoubtedly one of the main causes of prolonged unthriftiness and explains why calves often succumb to secondary infections in spite of antibiotic treatment.

Vitamin A (50,000–100,000 units), Vitamin B 12 (100 mcg.) and Vitamin B complex should be injected as a routine and repeated after three days if necessary.

Vitamin A may also be dosed *per os* in older calves. The freshness and potency of cod liver oil or vitaminised oil should be established before dosing, if use is made of this as a source of Vitamin A. The gelatin-coated granulated powdered form of Vitamin A is more dependable.

Phosphorus and calcium depletion can be corrected by the feeding of bonemeal but the easiest and most successful way appears to be the regular addition of a balanced mineral-vitamin powder to each feed. If done from the commencement of bucket feeding this additive alone decreases the incidence of scours remarkably.

Protein loss is counteracted by the addition of two whole raw eggs to each feed in bucketfed calves. In the older calf fed on a good calf meal, protein in the diet is sufficient.

6. It is often not appreciated that specific antibacterial therapy changes the normal flora of the intestinal tract and that a prolonged disturbance influences digestion and food consumption profoundly. This again accounts for unthriftiness and liability to secondary infection. Soured milk (2–4 oz.) should be added as soon as feeding is resumed and continued until the animal appears completely healthy. The soundness of this fact is illustrated in calf-feeding schemes where the entire diet consists of sour milk from the third day. The incidence of enteritis in these schemes are minimal and it appears that calves are not in the least affected by irregular feeding, or varying quantities of milk per feed.

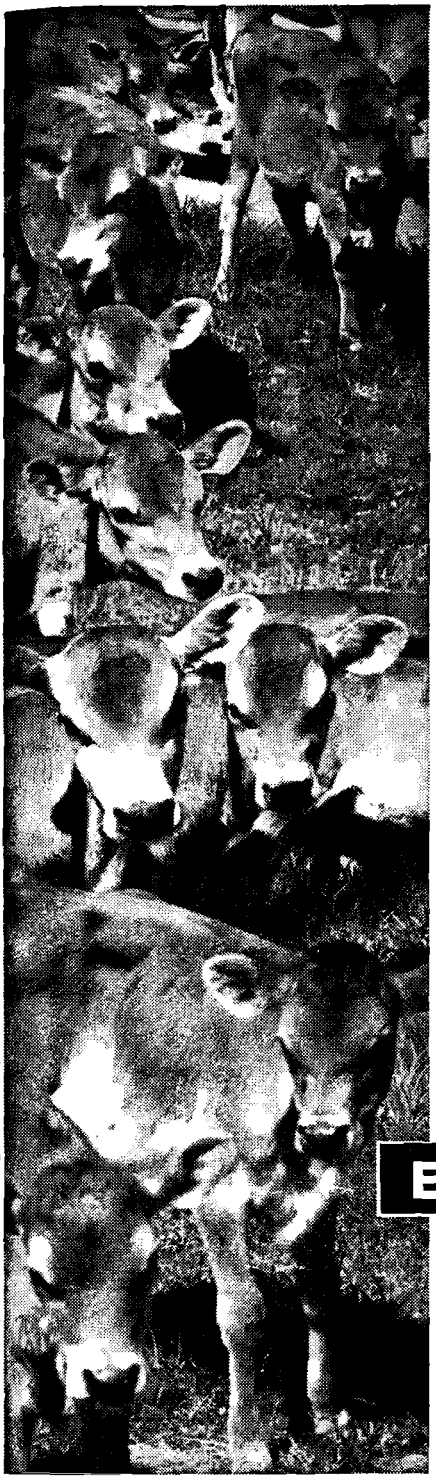
In the older calf, with established ruminal digestion, the dosing of 12 oz. of fresh (not more than one to two hours after collection) ruminal fluid shortens the time of return to normal physiological digestion.

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The administration of parenteral electrolyte fluids, vitamins and minerals, is essential in every case of enteritis. Furthermore the veterinarian will best serve his client by leaving very elaborate instructions regarding after care and rehabilitation of calves suffering from enteritis. This will prevent calves just recovered from enteritis from becoming an economic burden and serving as a dangerous source of infection for other calves.

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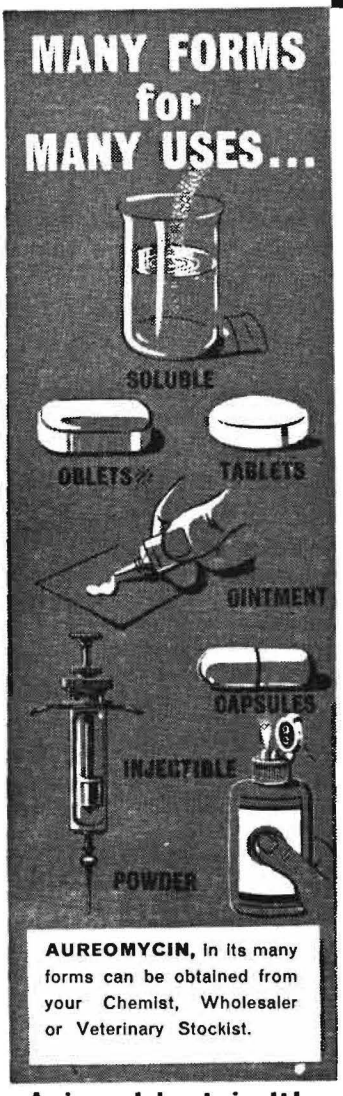
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HISTORICAL NOTE

H. P. A. DE BOOM
Onderstepoort

From the "Natal Almanac and Register" for 1889, kindly lent to me by Dr. L. G. Steel, Ingogo, emerges the interesting fact that the salary of the Veterinary Surgeon for the Colony of Natal, a post then held by Samuel Wiltshire, M.R.C.V.S., was £300 per annum, compared to salaries of £100 to £200 for District Surgeons, £150 for the Superintendent and Dispenser of the Durban Hospital, and £400 for the Medical Superintendent of the Pietermaritzburg Lunatic Asylum. Wiltshire was the first Government Veterinary Officer to be appointed in the Colony; a position amongst the pioneers of veterinary science in South Africa justly may be claimed for him.

His report for the year 1887 contains anecdotes which reveal how certain frustrating situations, like the poor, will always be with us. Referring to a proposed investigation into mortality amongst a troop of horses he writes: "At the time appointed for my visit, an animal, said to be very bad with the disease in question, was to be ready for my examination, both *ante-* and *post mortem*. I did not reach the farm till evening, and, as the animal was in the veld, it was decided to leave it till the following morning; but when morning came it was nowhere to be found, and the greater portion of the day was spent searching for it without success. As no other case was to be had, I was compelled to return without accomplishing anything."

To understand some of Wiltshire's remarks on horse-sickness, one must refer to Nunn's publication on the subject, which appeared in the Veterinary Journal of 1888, Volume 26, page 38. J. A. Nunn, F.R.C.V.S., F.R.G.S., arrived in Natal in 1887 to take up duties in the Army Veterinary Department of Natal. He was apparently well equipped for research work on infectious diseases He recounts how the prepared cultures brought from England and kept in his cabin during the voyage nearly had come to grief in the Bay of Biscay, the seawater fortunately only penetrating halfway through the plugs of some of the tubes. How well can our colleagues, working in rural areas, even today commiserate with Nunn's struggles to keep incubators going without gas or electricity, and to heat anything without getting beakers blackened and messed up with soot.

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Anyway, Nunn was able to distinguish between the bacilli, seen in the blood about two hours after death from horse-sickness, and anthrax bacilli. He failed to isolate bacteria from living cases and was of the opinion, on epizootological evidence, that horse-sickness was not contagious, but of "Malarious origin", i.e. caused by disease-producing air. This was ten years before viruses were first recognized as cause of disease.

Wiltshire concedes that horse-sickness is "not true anthrax", but finds it difficult to take leave of the old idea that it is closely identified with that disease. The reason he states: "The bacillus, supposed germ of the disease, is not readliy found until stained by an aniline dye" (thus disregarding Nunn's distinction between putrefactive and anthrax bacilli) and also "the sudden great mortality." He continues: "An important point on which I differ entirely from Mr. Nunn, is the way in which the disease obtains access to the body. He considers it to be of malarious origin; I contend that there is nothing to justify such an opinion, and consider it to be derived from the soil, and to be conveyed into the system by means of food (grass chiefly), and possibly, in extremely rare instances, in the water, and not by the air at all." "Thus even if we admit that this affection is not typical anthrax, I fail to see why it should not be classed as anthracoid, as it has hitherto been, seeing that in many respects it differs about as much from true anthrax as one pea does from another pea."

Some of his pronouncements, however, still hold true: "I cannot hold out any hope of finding a specific cure of it." Others have a familiar ring: "I have never been furnished with the means of conducting experiments and investigating disease, and what I have been able to do in that way has been under great difficulties, and principally at my own expense." He then records the successful subinoculation of a goat from a case of horse-sickness: "... it was very ill for two days; and its temperature rose to 105° Fahrenheit."

Other diseases dealt with in this report may be mentioned by heading, adding some of Wiltshire's remarks.

SCAB: "Much benefit" is anticipated from the newly amended Scab Law, amendments suggested by communication with "experienced farmers" and passed with "slight alterations". During the first half of the year 36,468 cases of scab amongst 388,087 head of sheep were reported by Sheep Inspectors, and during the second half 23,701 out of 443,018 head. The cases included clean sheep "being under licence" which "had to be returned accordingly."

LUNGSICKNESS: "... there has been a great deal . . . even amongst those inoculated and supposed to be safe . . . much greater care should be exercised to prevent contact with cattle suffering from the disease and some stricter regulations are needed

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to prevent lungsick animals from spreading the disease in the reckless way that now prevails."

REDWATER: "... deaths of several valuable shorthorn cattle from redwater and other causes . . . so far no means of lessening the mortality have been discovered . . . pedigree shorthorns that have been bred up in a delicate way are less likely to survive than those bred up in the English straw yard under more natural conditions . . . also the more mature animals . . . would be better able to bear up against the effects of the change of climate, disease, etc." The latter conclusion is at variance with present-day ideas, and probably the result of theorizing rather than critical observation. Only when one takes note of statements, such as those made above with reference to redwater and horse-sickness, does one realize the serious handicap imposed on our predecessors by their ignorance of insect transmission of disease.

GLANDERS: Wiltshire inspected a glandered horse, "one of several cases that have occurred." He is very optimistic; "now that people are on their guard, and the law more efficient, I do not fear its extension very much"; — a hope come true after many years.

QUARTER EVIL: This disease, referred to as "sponziekte", "prevails extensively every year."

OTHER DISEASES: "... there were the usual losses from 'blue tongue', 'heartwater', and other causes, of which *very little is ever reported to anyone*, and, when it is, it is *usually long after* the losses have occurred, or *too late to do anything.*" (Italics mine.)

PREVENTATIVE MEDICINE: Wiltshire was well imbued with the idea of preventative medicine, as his remarks under lungsickness and scab bear out. He warns against the danger of importing high-bred cattle infected with tuberculosis, "not yet met with in cattle", but "the public cannot be too much on the alert against its introduction into herds of the Colony; and, should it manifest itself, should take prompt and decisive action to stamp it out." Sound advice unheeded! He stresses the importance of disinfection and carcase disposal, and quotes at length an instance of a horse dying "after a few hours' illness, accompanied by considerable swelling of the throat and other parts of the body." Fourteen out of eighteen ducks, who gobbled up blood spilt from the carcase, died, a dog sickened, and a large number of sheep died seven days after being placed in the shed where the horse had died five months previously. By implication the disease was "meltsickness". In Nunn's publication the same outbreak is mentioned. A definite diagnosis of anthrax was made and Nunn recounted how six "kaffirs" had died out of forty to fifty who had sickened after handling or consuming carcase material. Finally Wiltshire points out the necessity for "some proper supervision

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over meat offered for sale, a duty which can only efficiently be performed by a person educated for the work, and expert with the microscope."

Wiltshire was gratified to "observe rapid increase in fencing of the land" and "the increasing attention to the feeding of stock in the winter, and the recognition of its value."

With nostalgic reluctance one takes leave of the chatty style of yesteryear. Science has come a long way; human foibles remain.

ERRATUM

In the article "The Treatment of *Filaroides osleri* infestation with Dictycide" by J. E. Dorrington which appeared in Vol. 30, No. 1 (March, 1959), page 27, of this journal, the dose is erroneously given as 1 gram per 10 lbs. body weight. This should read 1 grain per 10 lbs. body weight.

DEDUCTIONS FOR INCOME TAX PURPOSES

The Commissioner for Inland Revenue has advised (R.18/223 dated 19.2.59) that subscriptions to the Association and to Medical (Veterinary) Journals will be allowed as a deduction for income tax purposes.

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CHIRURGIESE NUUS — SURGICAL NEWS

GAST VORLESUNG, GENT

KOK, O. V. S., en VAN DER SPUY, J. C. (1958): Hartarres tydens Chirurgiese Operations — South African Practitioner 3(4).

Een uit elke twee pasiënte wat onder narkose sterf, beswyk aan hartarres op die operasietafel. Oorsake kortliks:

(a) Onvoldoende suurstof aan miokardium a.g.v. versperring lugweë, onderdrukte asemhalingssentrum, ens.

(b) Bykomende faktore soos hiperkapnie — baie belangrik — tree op sowel as prikkeling van die vagus en simpatiese senuwees en oorgevoeligheid teenoor die narkosemiddel.

Behandeling onder ideale omstandighede:

(a) Staak narkose.
(b) Dien 100% suurstof toe.
(c) Intubeer tragea.
(d) Onmiddellik torakotomie tussen 4de en 5de ribbes en hartmassering, 80 keer per minuut.
In geval van hartstilstand:
(e) Atropien gr. 1/50 en 10 ml. fisiologiese soutoplossing in linkerventrikel indien vermoed word dat hartarres deur vagusrefleks veroorsaak is.
(f) Kalsium chloried 10 ml. 5% oplossing in linker ventrikel.
(g) Adrenalien 0.3 ml. van 1:1000 in 10 ml. fisologiese soutoplossing — *maar alleen as hart pienk is* — anders kan fibrillasie veroorsaak word.

In geval van ventrikulêre fibrillasie: Tree op soos (a)–(d) en dan verder as hartkleur maksimaal verbeter het: (e) Skokke met defibrillator met tussenpose van 0.1 tot 1.0 sekonders.
(f) Prokaïen hydrochloriede — 10 ml. 1% oplossing — linker ventrikel.
(g) Kalsium chloried 10 ml. 2% oplossing — linker ventrikel.

Ten laaste die versigtige toediening binneaars van vog, bloed en vasopressormiddels. 'n Lys van items word gegee vir 'n hartmasseringsdrom. Die enigste instrument werklik nodig vir onmiddellike hartmassering is 'n mes. As geen apparaat vir suurstoftoediening onder druk beskikbaar is nie, word hartmasseer deur laparotomie gedoen.

C.F.B.H.

VAUGHAN, L. C. (1958): Studies on Intervertebral Disc Protrusion in the Dog. Brit. Vety. Jnl. 114(12). This is the fourth in the series of papers.

In outlining medical treatment emphasis is placed on nursing and the employment, particularly, of Vit. Bl.

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The surgical treatment comprises:

(a) Laminectomy, and

(b) Disc fenestration.

The former is performed by midline dorsal incision, exposing the dorsal spines, three of which are removed, the centre one being over the herniated disc. Access is gained to the spinal cord by drilling with a hand drill through a posterior lamina but not over the protrusion. The opening is enlarged with nibbling forceps, great care being taken to avoid venous sinuses. The herniated disc is then removed. Results have been so poor that this operation is not justified, unless it can be modified. It is, however, important to know of these failures.

Disc fenestration is much less hazardous, can be carried out on the whole of the vertebral column and had a recovery rate of eight out of 13 cases. Two deaths took place within 24 hours. The evaluation is, however, made difficult by the fact that spontaneous recovery occurs. The cervical discs are approached ventrally, those posterior to the 12th thoracic vertebra dorso-laterally and the other thoracic vertebrae by the transthoracic route. A small circular piece is removed from the disc and all of it is curetted out. Any herniated portion into the spinal column is left untouched.

C.F.B.H.

DEBACKERE, M., VANDERPLASCHE, M., PAREDIS, F. (1959): Vergelijkende Studie over de Resultaten Bekomen na Sectio Caesaria en na Foetotomie bij het Rund — Vlaams Diergeneeskundig Tijdschr. 28(1).

Van 337 keisersneë is 320 antwoorde op aanvraag ontvang. Van 320 gevalle is 16 vernietig — 7 met bestaande ruptuur van die uterus voor operasie en een elk a.g.v. hydro-allantoïs, lebmaagulcus, en breuk van die nek femur. Alleen ses gevalle skyn in direkte verband met operasie te staan — dus 2%. Daar is gevind (Vanderplasche e.a.) dat 50% van lewende foetusse doodgaan — 23% tydens vervoer na kliniek, 13% tydens operasie en 14% na geboorte. Uitgebreide data word verder verstrek t.o.v. operatiewe behandeling, en voorkoms van steriliteit en so meer.

Gedurende 1955-1957 is 80 volledige foetotomies gedoen onder epidurale verdowing en met die gebruik van die Thygesen foetotoom. Op navrae is 74 antwoorde gekry. Sewe gevalle is dood — 3 van uterusskeur en 2 van bloeding. Twee ander is binne ag dae dood. Verdere analises word gegee t.o.v. naoperatiewe behandeling, steriliteit, ens.

Daar is tot die slotsom geraak dat die twee metodes min of meer gelykwaardig te beskou is in die algemeen. Vir die eienaar is keisersneë egter duurder en in foetotomie is die foetusse steeds dood.

C.F.B.H.

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BARRET, E. P., and NICOL, J. (1958): Displacement of the Bovine Abomasum. A Clinical Note on Surgical Intervention — Vety. Rec. 70(51).

This condition is receiving more and more attention in the veterinary press. These authors report on 4 cases. No particular attention is paid to symptomatology as the purpose of the article is to describe the treatment.

An attempt was made in the first place to replace the abomasum by casting and rocking. This having failed the one author passed his hand through a left sublumbar laparotomy incision underneath the rumen and the other author, using the same incision passed his hand over the top of the rumen and then downwards. The displaced abomasum then being handed on from the one operator to the other. The second case responded to rocking. The third case was operated upon unsuccessfully as interposition of the omentum caused great difficulty when handling of the abomasum was attempted. The second operator then, at a subsequent operation, moved his hand over the rumen until the right abdominal wall was felt, then down along the wall until the liver was palpated, from there to the omasum and then to a position to receive the abomasum from the hand of the first operator. In the last case replacement was regarded as unsatisfactory but the case improved and recovered in 10 days.

C.F.B.H.

AMMANN, Prof. K. (1957): Hauttransplantation bei Pferd und Rind — Mededelingen der Veeartsenij School van de Rijksuniversiteit van Gent 2(2).

Hierdie referaat is een van 'n aantal oor hierdie onderwerp deur prof. Ammann of afkomstig uit sy veterinêr-chirurgiese kliniek aan die Universiteit Zürich. Na 'n beskouing oor die genesingsproses van wonde en die verskillende metodes van verloorplanting word die tegniek van Braun in besonderhede beskrywe. Klein stukkies dermo-epiteel word ongeveer 0.5 cm. diep in die granulasieweefsel van 'n wond ingeplant sodat dit beskerm is teen uitdroging, meganiese invloede en so meer. Die epiteel groei nou na die vel en vorm 'n eilandjie wat spoedig aansluit by naburige eilandjies om sodoende spoedig die rou wond te bedek. Groot dosisse pantoteensuur word ingespuit om die kwaliteit van die littekens te verbeter.

C.F.B.H.

STÖBER, M., und RIEGER, TH. (1959): "Tranquillizer" (Megapten, Combelen, und Decantan) bei der Behandlung der Schlundverstopfung des Rindes — Deutsche Tierärztliche Wochenschrift 66 (2).

The article deals with 22 cases of obstruction of the oesophagus treated with tranquillizers, after other therapeutic measures had failed. In 4 cases the foreign body could be removed manually and in the other 18 by probing. Some cases became recumbent for a time after medication. The dose chlorpromazine employed was 150–350 mgm. intravenously for a 500 Kg. cow.

C.F.B.H.

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LAWSON-DONALD, D. (1959): Transfixation Pinning of Mandibular fractures in the Dog and Cat — Wiener Tierärztliche Monatschr. 46(1).

'n Oorsig word gegee van die baie metodes wat gebruik word. Die metode wat L aanbeveel is: 'n Vleklose staal pen 3/32" deursnee 18/8 word reghoekig op die middellyn deur die onbeseerde kant van die kakebeen geboor, oor die tussenkaakruimte deur die sagte weefsel gestuur tot deur die ander deel van die kakebeen net agter die breuk. Tandwortels moet vermy word. In 6 jaar het L. 38 gevalle (27 honde en 11 katte) op hierdie manier behandel met sukses. Daar is geen na-operatiewe ongerief nie.

C.F.B.H.

BOOK REVIEWS

DISEASES OF DOMESTIC ANIMALS IN NEW ZEALAND by Technical Committee N.Z. Veterinary Association Inc. (1st edition) Wellington: Editorial Services Limited, 1958 x + 240 p.p. Price 35s.

This reference book contains approximately 175 summaries of diseases and diseased conditions important to veterinarians in New Zealand. The presentation of the material is extremely brief and in regular form. It contains a wealth of information devoid of detail which makes it an ideal handbook in practice.

Owing to the brevity of the text many statements are seemingly incomplete. For instance "Coenurosis" is mentioned as a differential diagnosis on pages 45 and 219 but does not figure in the list of diseases.

The Committee set out to give only a concise summary of principal facts and in this it has admirably succeeded.

This book should be a valuable aid to the practitioner, also in South Africa.

G.C.v.D.

DIE KALF: SY VERSORGING, VOEDING EN SIEKTES, deur Prof. dr. M. W. Henning, Landbounavorsingsinstituut, Universiteit van Pretoria. Uitgewers: Die Voortrekker Pers, Bpk., Johannesburg. Prys £1.12.6.

Hierdie omvattende boek wat die moeilikhede van kalwers grootmaak beskrywe kan aan die Boer, Landbou-deskundige en Dierarts aanbeveel word. Die boek hanteer in deel 1 die seleksie, versorging, behuising en voeding van die kalf, vernaamlik die grootmaak van kalwers op melkvervangerrantsoene en voedselbenodigdhede; in deel 2 besmetlike siektes, in deel 3 uitwendige en inwendige parasiete, en in deel 4 ander siektetoestande soos vergiftigings, opblaas en oorerflike gebreke.

Die boek is mooi gedruk met duidelike prente en tabelle.

An English edition of this book is being prepared and should be published soon. (Author's note.)

G.D.S.

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DIE WICHTIGSTEN OPERATIONEN DES TIERARZTES IN DER PRAXIS — Dr. med. vet. Theodor Hahn + neubearbeitet von Dr. med. vet. Rolf Teuscher. No figs. Pp. 159, 1958. Terra-Verlag, Konstanz. Price (?) DM. 12,80.

The contents of the book are arranged firstly to present a general section including equine lameness, anaesthetics, control, wounds, bandages and shoeing. The second part includes the special surgery of the equine, bovine porcine and carnivore species — each in a separate sub-division.

This work, as the title shows, deals only with those operations regarded as important. Veterinarians practising under conditions different to those in Germany may therefore fail to find reference to operations which they regard as important.

Sub-headings are used sparingly and the printing is fine, so that perusal is not as convenient as it could have been. Even so, very interesting information is to be obtained from these pages, e.g. Forssell's operation — at best not pleasant to perform — is simplified in such a manner that it occupies 30-40 minutes or less.

As a method of rumenotomy, Götze's technique is described as is the method of Weingart. The former technique is never employed in South Africa — all the operations for removal of a foreign body from the reticulum being basically the same as Weingart's.

In operation for torsion of the uterus in the cow some distinction should have been made as to the degree of torsion.

In cases of torsion of much more than 180 degree, the reviewer has found the technique described impractical, whereas if the rotation is about that, rectification without laparotomy is usually possible.

The technique of oophorectomy in the bitch is far behind that generally practised in South Africa. In contrast, the methods described for drainage of the external auditory meatus are in advance of those mostly seen here.

Reading through this book has been interesting and rewarding — an opinion that will be shareed by others who have acquired it.

C.F.B.H.

TIERARZTLICHE OPERATIONSLEHRE — Prof. Dr. Dr. h. C. Ewald Berge and Prof. Dr. Melthior Westhues. 264 figures. Pp. xi + 374, 1956. Verlag: Paul Parey, Berlin und Hamburg. Price: DM. 29.60.

This book is divided into the following divisions: 1. *General Surgery* embracing 31 pages devoted to a description of general procedures, sutures, etc. Ten pages are devoted to methods of osteosynthesis. It would have been an improvement if some information was given about electrolysis associated with metal implants of certain types. 2. *Anaesthesia* occupies the next 50 pages. The illustrations of nerveblock anaesthesia are particularly valuable. 3. *The following chapters* deal systematically with operations on the head, eyes, neck, thorax, abdomen and tail, uro-genital system and limbs of all domestic species. There is a concluding division on restraint.

In general the information is presented clearly and briefly. All surgeons will not be in agreement with all the operative techniques described, but every one will appreciate the fine illustrations and the clear headings and sub-headings in the text.

A notable omission is description of the transthoracic repair of diaphragmatic hernia. The abdominal approach is described. The method of removing air from the thorax postoperatively by employing catheter, plastic tubing and bottle with water, deserves mention.

In final analysis, this book can be recommended to every veterinarian

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interested in surgery. There is a great deal that pleases and instructs, and the manner in which information is presented makes reference rapid and easy.

C.F.B.H.

CLINICAL VETERINARY SURGERY — Vol. I — GENERAL PRINCIPLES AND DIAGNOSIS — by R. H. Smythe, M.R.C.V.S., Examiner in Surgery to the Royal College of Veterinary Surgeons. Sixteen plates and 56 figures. Pp. x + 362. 1st Ed. 1959. Crosby Lockwood & Son, Ltd., London. Price 42s.

As indicated, this is the first volume and therefore only part of the eventually to be completed work As it means in effect, seeing only part of the whole, reviewing is complicated accordingly.

Smythe devotes considerable space to what can be termed general surgery. This is most commendable as there is a general tendency — even among those who ought to know better — to forget the basic principles of surgery in their haste to attempt advanced surgery. One matter of basic importance, the reviewer feels, merits more attention than it receives in this book, namely shock. Much that is important here has been omitted. In the section on surgical conditions of the alimentary tract of the dog and cat the oesophagus has been overlooked and certain conditions of the stomach like pyloric spasm. In contrast, an itneresting description is given of a condition in cattle than is as yet not widely known — displacement of the abomasum.

This may be taken as a fairly representative feature — omission of certain conditions and interesting discussions of others. The illustrations, although they serve their purpose, lack artistic finesse.

Final judgement of this book must await the appearance of the companion volume(s). Yet at this stage there is much in this book to commend it. The basis of approach is the broad one of general practice. This will be appreciated by those whose practices include all domestic species in significant numbers. For students it will serve as an introduction to clinical surgery — again with the emphasis on the general approach.

C.F.B.H.

GREPE UIT DIE AFRIKAANSE BAKTERIOLOGIE — deur G. C. van Drimmelen, D.V.Sc., Pretoria, 1958; 80 bladsye. 18/-.

In hierdie beknopte handleiding oor *Geneeskundige Bakteriologie* word die studie van potogeniese kieme van verskillende oogpunte benader.

Daar word, onder andere, beskryf hoe monsters vir 'n bakteriologiese studie versamel en ondersoek word. Al die vernaamste serologiese, biochemiese en biologiese metodes van ondersoek word kort maar saaklik verduidelik. Die belangrikste metodes wat aangewend kan word vir die afsondering van bakterieë in suiwer kultuur en vir die kweek van bakterieë in kweekbodems, word gegee. Daar is ook 'n kort beskrywing van die praktiese toepassing van die bakteriologie. Dit word opgevolg deur 'n uiters handige lys van mikrobiologiese terme.

Dit wemel van nuttige inligting en kan met vrymoedigheid aanbeveel word vir enigiemand wat in mikrobiologie, en besonder patologiese bakteriologie, belangstel.

As dit net 'n "inleiding" is van 'n groter werk wat beoog word, kan met groot belangstelling na die verskyning van die groter werk uitgesien word.

M.W.H.

NOTE

The following facts were omitted from the last issue of the Journal:

The price of "Newsom's Sheep Diseases" is 72/- and that of "Evolution of the Veterinary Art" is 55/.

The London publishers of both books are Baillière, Tindall and Cox Ltd.

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SOUTH AFRICAN VETERINARY MEDICAL ASSOCIATION

MINUTES OF THE MEETING OF COUNCIL HELD ON MONDAY, 4th MAY, 1959, AT THE MEAT BOARD BUILDING, PRETORIA

PRESENT:

Dr. H. P. Steyn (President), Drs. R. du Toit, P. S. Snyman, L. W. v.d. Heever, C. F. B. Hofmeyr, M. de Lange, A. F. Tarr, H. H. Curson, J. W. Pols, A. M. Diesel, M. C. Lambrechts, M. W. Henning, S. W. J. van Rensburg, E. M. Robinson, G. D. Sutton (Hon. Treasurer), S. van Heerden (Hon. Secretary) and R. K. Loveday (Co-opted).

Apologiese for absence received from Drs. R. A. Alexander, P. J. du Toit and R. Clark.

2. MATTERS ARISING FROM THE MINUTES:

Antibiotics in milk. After discussion, Council agreed to write to the Secretary of Health, enclosing a reprint of Dr. Meara's paper and requesting an interview after the Secretary's return from the Parliamentary Session.

Also agreed to obtain sufficient reprints of this article as cheaply as possible for despatch to members of the Dairy Industry Control Board and the Cheese Manufacturers' Association.

Reserve Prize Fund. The Chairman of the Finance Committee proposed the utilisation of £1,000 of the above fund to create a bursary fund for post-graduate research, and the balance of the fund to be used as a loan fund for veterinary students or graduates requiring assistance with tuition fees, research, publication or scientific films. The Finance Committee also considered that the closing of the Book Fund should be considered, in order to release the capital amount (approximately £2,200) tied up in this fund for other purposes and to eliminate the considerable amount of administrative control required at present for the very limited profit which was being made.

After discussion, Council agreed that both these matters should be considered at its next meeting, and that the Finance Committee would provide a short report for circulation with the Agenda of the next meeting.

Dr. Henning requested that the question of payment of the Editor of the Journal and financial aid from the Department of Education, Arts and Science also be considered at the next meeting.

Maud Bales Scholarship. The Hon. Treasurer reported that Mr. C. S. Nicholson had been selected from the 7 applications received to the re-advertisement of the scholarship, and that the Trustees had agreed with this choice. Confirmed.

A letter regarding the failure of locums to honour their contract was also discussed. It was agreed that this matter be brought to the profession's notice in the Journal and also be added to the letter sent to new graduates. It was also decided that the legal adviser be requested to write a short article on contracts for the Journal.

Dr. Tarr informed Council that the Town Planning regulations of Pietermaritzburg required a veterinarian opening a new surgery to advertise this fact, giving his full name, by means of two advertisements in two daily newspapers. Council decided that the Town Clerk of Pietermaritzburg be requested to explain the necessity for the advertisement and whether names are necessary, also that his attention be drawn to the provisions of the Veterinary Act in regard to ethical conduct and advertising.

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Congress, 1960. This matter had been referred to all branches and the Cape Western Branch had invited Council to hold the 1961 Congress in Cape Town. A letter from the Director of Veterinary Services was also read in which the implications of a change of venue were fully elaborated. After consideration, Council agreed to leave this matter in abeyance until 1961, but to accept provisionally, the Cape Western invitation, while pointing out that the difficulties involved had not yet been completely solved.

A Veterinary Co-op. Agreed to write, forwarding a copy of the draft contract and indicating that Council will only approve of an appointment under proper conditions. Full-time emplyoment in conformity with the Ethical Code was also acceptable. Council also agreed that the President would meet the directors if required, and that the expenses of his visit would be borne by the Association.

Draft contract. Agreed to hold over this matter for further consideration at the next meeting.

Training of Meat Inspectors. The Public Health Group's report was handed in and approved.

3. COMMITTEE OF ENQUIRY INTO FOOT AND MOUTH DISEASE.

After Dr. Diesel had explained the questionnaire to Council, it was agreed to appoint the President as a one-man Committee (with powers to co-opt) to present evidence on behalf of the Association, if deemed desirable.

4. AMENDMENTS TO THE VETERINARY ACT.

It was agreed to roneo the proposed draft and circulate this for consideration at the next meeting.

5. FINANCIAL:

(i) A letter from Messrs. Glover & Dyer (Pty.) Ltd., requesting an increased fee was read and the Finance Committee's recommendation to increase their fee from £25 to £35 per month as from the commencement of the financial year, viz. 1st April, 1959, was approved by Council. The Hon. Treasurer was requested to ascertain what reduction in this payment would result from the abolition of the Book Fund.

(ii) The recommended donation of new books to the value of £15.12.0 to the Students' Library as listed by the Hon. Treasurer, was approved.

(iii) A Finance Committee recommendation that the expenses of delegates to special meetings be paid for out of Association funds was accepted in principle and agreed to.

6. MEMBERSHIP:

(i) Applications for membership, proposed by Dr. C. F. B. Hofmeyr and seconded by Dr. R. du Toit had been received from Drs. W. L. Jenkins and R. Clatworthy. These were approved by Council for submission to the next Annual General Meeting.

(ii) The deaths of Dr. G. Watt of Florida and Dr. Cordy of Natal were reported to Council and the usual token of respect was paid.

7. CORRESPONDENCE:

A letter from the Commissioner of Inland Revenue allowing tax rebate on subscriptions to the Association and to Journals was to be published in the Journal.

Letters of sympathy sent to Drs. Bishop and Hellberg and Mrs. Runciman in their bereavement.

A cutting from the S.A. Medical Journal regarding the exemption of Medical practitioners from the need for a licence for conveyance of medical supplies was read.

Letter from Agro-Chem. regarding prescription of remedy by veterinarians read. Council decided this would be maintained if remedy was not registered.

Training of Animal Nurses. Approved in principle and the S.P.C.A. Johannesburg is to be requested to carry on with the curriculum, etc.

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8. GENERAL:

The Hon. Secretary's recommendation that Dr. R. K. Reyneke be requested to represent the S.A.V.M.A. and convey greetings to the forthcoming Australian V.A. Congress was approved.

The President stated that Council meetings were being held too infrequently for the proper conduct of the Association's affairs, and on his suggestion it was agreed to meet every two months and that the Hon. Secretary should deal with minor matters as he saw fit.

The intention of the Potchefstroom Municipality to appoint a part-time veterinarian was noted.

The meeting closed at 6.50 p.m.

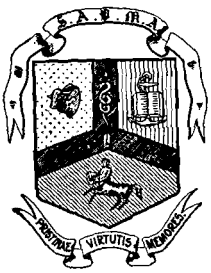
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RETURN OF OUTBREAKS OF SCHEDULED DISEASES IN THE UNION OF SOUTH AFRICA DURING THE PERIOD 1/3/59 TO 31/5/59.

DISEASE	CAPE	NATAL	O.F.S.	TRANSVAAL
Anthrax	1 Barkly West 3 East London 1 Indwe 2 King William's Town 1 Komkha 1 Middeldrift 1 Namaqualand		2 Bloemfontein 1 Brandfort 1 Bultfontein 1 Ficksburg 1 Reitz 2 Wesselsbron	1 Ermelo 3 Potchefstroom
Bacillary White Diarrhoea	1 Wynberg		1 Bethlehem 1 Reitz	1 Ermelo
I[illegible] mitis and [illegible]tis				2 Pietersburg 3 Rustenburg
Foot and Mouth Disease				7 Pilgrimsrest 9 Barberton
Fowl Typhoid	2 Albany 1 Gordonia 3 Kokstad			1 Pretoria
J[illegible]ne's Disease	1 East London			
Lumpy Skin Disease	1 Cala 1 Elliot 1 Elliotdale 1 George 1 Glen Grey 14 Indwe 3 Mqanduli	1 Durban 1 Entonjaneni 2 Eshowe 4 Klipriver 1 Lions River 1 Mapumulo 1 Nkandhla	3 Bethlehem 1 Boshof 5 Bultfontein 1 Clocolan 1 Ficksburg 10 Harrismith 2 Frankfort	1 Bronkhorstspruit 3 Ermelo 8 Heidelberg 6 Johannesburg 1 Klerksdorp 1 Piet Retief 2 Potchefstroom
[illegible] Skin e	5 Ngqeleni 1 Port St. Johns 3 Tsolo 4 Umtata	2 Ngotshe 1 Nongoma 1 Umlazi	1 Hoopstad 5 Jacobsdal 4 Kroonstad 1 Ladybrand 10 Reitz 1 Senekal 1 Vrede	2 Potgietersrust 3 Pretoria 3 Randfontein 2 Rustenburg 4 Springs 1 Ventersdorp
Mange (Bovine)	1 Malmesbury			
Mange (Goat)	1 Elliotdale 2 Kentani 4 Middeldrift 1 Peddie	1 Inanda		
Mange (Equine)	1 Bathurst 2 Umtata		1 Harrismith	
Psittacosis				1 Pretoria
Rabies	2 Vryburg		2 Bloemfontein 1 Senekal	11 Letaba 1 Lichtenburg 3 Zoutpansberg
Sheep Scab	5 Mafeking			4 Lichtenburg
Trypanosomiasis (Dourine)	1 Kuruman 1 Vryburg			
Trypanosomiasis (Nagana — T. Simiae)		3 Hlabisa		
Tuberculosis (Bovine)	1 Cape Town 1 De Aar 1 East London 4 Gordonia 1 Mossel Bay 4 Prieska 1 Wellington	2 Durban 1 Impendhle 1 Umlazi	1 Kroonstad 1 Reitz 1 Ventersburg 1 Viljoenskroon 1 Vrede	1 Bronkharstspruit 1 Carolina 1 Kempton Park 3 Klerksdorp 3 Potgietersrust 1 Standerton 1 Ventersdorp 1 Volksrust 1 Warmbaths 1 Wolmaransstad
Tuberculosis (Porcine)				1 Potgietersrust

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South African Veterinary Medical Association



54th ANNUAL CONGRESS

ONDERSTEPOORT,

AUGUST 25th, 26th and 27th, 1959

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The President, Dr. H. P. Steyn.

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PROGRAMME

of the

54th ANNUAL CONGRESS

Faculty Buildings, Onderstepoort,

August 25th, 26th and 27th, 1959

TUESDAY — August 25th:

9 a.m. Convocation of meeting.

9.05 a.m. Welcome to guests and delegates and members attending Congress.
Dr. R. A. Alexander, Director of Veterinary Services.

9.10 a.m. Official opening of Congress by Dr. W. J. Edens, Agricultural Attaché, Embassy of the United States of America.

10 a.m. Reply on behalf of Association: Dr. A. M. Diesel.

10.10 a.m. Presidential address (Dr. H. P. Steyn).

10.30 a.m. Opening of Trade Exhibition by the Vice-President (Dr. R. du Toit).

11.15 a.m. Radio-isotopes in Biological Research:
Dr. W. R. McMurray.

12 noon The treatment of tuberculosis in man and animals with Isoniazid: Dr. H. Kleeberg.

2 p.m. Symposium on ticks:

(i) Ticks: Their biology and their distribution: Dr. G. Theiler.

(ii) Hyalomma and tick toxicosis: Dr. W. O. Neitz.

(iii) The practical application of some of the new synthetic insecticides in the control of ticks: Dr. L. C. Blomefield.

(iv) The development and practical application of organic phosphate insecticides: Dr. P. Thorold.

(v) The development and mechanism of insecticide resistance in the blue tick (*Boophilus decoloratus Koch.*): G. B. Whitehead.

(vi) A short review of the nature of dipping materials used in tick control in cattle from 1893 to 1939: Mr. P. M. Bekker.

WEDNESDAY — August 26th:

8.30 a.m. Prednisolone in the treatment of traumatic synovitis in the equine: Dr. J. Quinlan.

9.10 a.m. Torsion of the stomach in the dog: Drs. M. Hearn and B. Keep.

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9.30 a.m. Bloodvessel diseases in animals with special reference to a case of iliac thrombosis in the dog: Dr. J. F. du Plooy.

9.50 a.m. Bovine teat surgery: Drs. C. Howell and C. Maree.

10.10 a.m. The differential diagnosis of nervous symptoms in the bovine: Dr. J. D. Smit.

11.05 a.m. Some clinical laboratory tests useful in practice: Demonstration.
Drs. J. M. M. Brown and W. D. Malherbe.

11.30 a.m. The diagnosis of Brucella infections in sheep and goats: Dr. G. C. van Drimmelen.

12.10 p.m. The pH of meat: practical methods of determination and its significance, especially in the inspection and judgment of meat: Dr. L. W. van den Heever.

2 p.m. Preliminary observations on a recent epizootological blue-tongue survey: Dr. P. G. Howell.

2.20 p.m. Symposium on distemper:
(i) A few pertinent questions relative to immunisation: Dr. K. E. Weiss.
(ii) Differential diagnosis: Dr. Campbell Dickson.
(iii) Advances in treatment: Dr. U. von Backström.

4.10 p.m. Report by Dr. R. A. Alexander on the XVIth International Veterinary Congress, Madrid.

THURSDAY — August 27th:

8.30 a.m. A few remarks on Veterinary Organisation in a number of European countries: Dr. M. C. Lambrechts.

9 a.m. Business of the Association.

This issue of the Journal contains all those papers which were received in time for prior printing. Other papers presented at the Conference will be published in the next issue. — Editor.

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WELCOME TO GUESTS, DELEGATES AND MEMBERS

As host to the Congress, the Director of Veterinary Services was asked to welcome the visitors.

Dr. Alexander expressed his pleasure at seeing so many present. He stated that the Honourable the Minister of Agriculture was not able to be present but had sent his best wishes and was represented by Dr. Penzhorn. Neither the Secretary for Agriculture (Technical Services) nor the Secretary for Agriculture (Economics and Markets) was able to be present but both were represented.

Among those specially welcomed were, Dr. du P. le Roux (Secretary for Health), Dr. Edens (Agricultural Attache of the United States) who was to open the Congress, Mr. Whitehead of African Explosives, Dr. McMurray, of the C.S.I.R., and Mr. P. M. Bekker.

Dr. Alexander then expressed his pleasure at seeing so many of our old stalwarts present such as Mr. Chase, Dr. G. de Kock and Dr. Scheuber.

Dr. H. Nelson (M.O.H. of Pretoria and an Honorary Associate Member) was assured that he had made so much progress in veterinary science that he was now considered one of us.

The Directors of Veterinary Services of the Federation and of Nyasaland had not been able to attend but had sent messages of goodwill. Mr. Falconer was present representing the Bechuanaland Protectorate, Mr. D. Ross of the British Veterinary Association, and Mr. Gray, the Director of Veterinary Services of Northern Rhodesia.

Dr. Alexander went on to express his pleasure that so many municipalities had sent their veterinarians to the congress officially. This showed an appreciation of the part the veterinarian played in the field of public health.

The representatives of the firms exhibiting and the press were also welcomed.

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OPENING ADDRESS

by

William J. Edens,

United States Agricultural Attache, before the 54th Annual Congress of the South African Veterinary Medical Association at Pretoria, August 25, 1959.

I am deeply honoured and privileged to be with you on the occasion of your 54th Annual Congress. This occasion brings together South Africa's best thinkers and leaders in the profession of Veterinary Medicine.

There could be no more appropriate place for the meeting of this Congress than here at Onderstepoort; and I am confident your deliberations will be of great value to animal health of the Union.

Man has depended on animals as a source of food, clothing and shelter since the beginning of his time. Domestic animals and their care have always been closely related to the welfare of people, and in this commercial, scientific age concern for the care and health of livestock is more important than ever. Kinds of feed, balancing of rations, breeding for adapted and efficient growth, animal health and sound livestock management have come to be common farm terminology and recognized factors essential to a useful and profitable livestock industry.

Financially, the commercial livestock producer cannot overlook any of these factors. The farmer who loses a cow valued at £50 has lost the equivalent of a return on £1,000 invested for one year at 5 per cent. The loss of a sheep from illness in between shearing times is about the same as a loss of the return on £250 invested for one year. And the worst part of such a loss is that it comes off of the net farm return for the year and not off of the gross return.

History records that veterinary medicine developed contemporaneously with the domestication of animals, and the first authentic record of animal physicians is found in the laws of Assyria as laid down by Hammarabi in 2200 B.C. — almost a thousand years before Moses. In the centuries that followed, the developing science, like its counterpart in the human field, experienced periods of popularity and progress as well as of suppression before it finally became established as a respected profession. Records of formal education in veterinary medicine trace back to about 1740 in France, 1852 in the United States, 1854 in Canada and 1919 in South Africa, or ten years after Onderstepoort was established.

At present veterinary medicine is as modern and fast-changing as the headlines in our daily newspapers. Many of the drugs

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which the veterinarian uses and prescribes to-day were unheard of before World War II. Within the past two decades, whole new families of antibiotics have been added to his weapons for fighting disease. New treatments and surgical techniques are now saving lives and money in situations previously considered hopeless. And recently, veterinarians have been called to meet the new problems of space age.

In the Military Service the DVM's are working side by side with their more publicized fellow scientists, the phycisists, biologists and rocket engineers. The DVM now measures the effects of nuclear and thermonuclear energy on the health and foodstuffs of both man and animal, prepares defences against biological warfare, develops techniques for using animals in aviation medicine and other research. Air Force Veterinarians pointed out not long ago that animals are a vital link in the chain of events that prepares air crews for flight in vehicles of the future. When man finally rockets into space, I am sure veterinarians will have made major contributions toward that achievement.

About half of the total cash income received by farmers in South Africa is derived from the sale of livestock products. For the past two years the proportion has been 55 per cent in the United States. Consequently, whatever affects farm livestock vitally affects the agricultural economy of our countries. The veterinary profession plays a vital role in helping farmers maintain the health of livestock and carry on a profitable livestock enterprise. But the most convincing sign of what the veterinary profession means to the livestock industry — as in any business — is its economic value.

In 1955 the Veterinary Service Committee of the American Veterinary Medical Association made a study to determine what a veterinarian is worth in dollars to the community in which he is located. Treatment of pets was not included in the study and many other services were ruled out which time here does not permit enumeration. The final conclusion was that for every dollar a livestock owner spent for veterinary services, he received $5.78 return on his investment. With an estimated annual loss from animal and poultry diseases and parasites of nearly $3 billion in the United States, you can imagine what the loss would be without the services of veterinarians.

The present Veterinary Medicine programme of South Africa originated with and is closely tied to your Veterinary research and training laboratory, Onderstepoort. The work pursued here by Dr. Ray Alexander and his associates is of vital importance to the livestock industry of the Union. The important role played by an institution as Onderstepoort, in animal research, the prevention and stamping out of livestock diseases, and guarding the health of both man and beast, often is not thoroughly appreciated by the public.

The training of veterinarians to serve general farmers, commercial livestock producers and other animal owners who have

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vast amounts of capital invested in domesticated animals is a major contribution by Onderstepoort. Without it, research and the programme of controlling livestock diseases would soon become ineffective.

With five area veterinary field service sections, a concerted effort is being made by the Division of Veterinary Service and local veterinarians to provide farmers with vaccines and professional services in safeguarding the health of their animals. Onderstepoort is the only laboratory in South Africa which provides vaccines against diseases. This is a service indispensable to modern livestock production. The Service is to be congratulated on providing mobile laboratories for field services, where quick on-the-spot diagnosis of diseases can be made, and immediate action taken to treat, prevent or control diseases.

With no real shortage of veterinarians and an excellent veterinary training programme graduating 20 to 30 doctors of veterinary medicine annually to take care of professional turnover and the increase in animal production, livestock producers in South Africa are in a strong position to maintain better animal health and reduce losses caused by diseases.

In the United States the Federal Bureau of Animal Industry and State Veterinary Services co-operate in eliminating and controlling livestock diseases. Hand in hand the Federal Government and State Governments have moved to stamp out or effectively control many of the major livestock diseases known to you — such as foot-and-mouth disease, tuberculosis, tick fever, brucellosis, Scabies, hog cholera, New Castle disease, and others.

During the past five years Congress has been making available approximately $20 million a year to help stamp out brucellosis from cattle. State contributions have reached over $17 million a year for this work. The goal of complete modified certified status for the entire United States is now set for 1965.

The programme to eradicate tuberculosis was begun in 1917. To-day the percentage of infection in all counties in the United States is running somewhat below 0.2 per cent. Foot-and-mouth disease has been stamped out. The last outbreak was in 1929. Some diseases have not been so effectively handled. They are hog cholera, anaplasmosis, New Castle disease, fowl leucosis, and others. All are the objects of much research.

The United States Department of Agriculture's animal disease and parasite research is conducted in three major Federal installations, and a National Animal Disease Laboratory is now in the process of construction in Iowa. This is a $14 million laboratory and it will become a centre of the Department's infectious disease research.

The national professional organization of veterinarians in the United States is the American Veterinary Medical Association. Approximately three-fourths of the 20,000 veterinarians in the United States are members of the AVMA. This association main-

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tains headquarters offices in Chicago and has a central staff of about 30 people in addition to the editorial staffs, which produce the Journal of the AVMA and the American Journal of Veterinary Research.

The United States Department of Health, Education and Welfare has a small staff of veterinarians located at the Communicable Disease Centre of the Bureau of State Services. Most of these veterinarians are working in States as State Public Health Veterinarians and in Research. Their efforts are primarily directed toward diseases of animals transmitted to man, which have come to be known in later years as the zoonoses. The Army and Air Force in the Department of Defence both have staffs of veterinarians whose principal functions are to inspect foods and to cooperate on research relative to food products and animals used in military research projects. Veterinarians employed in the United States Department of Agriculture number about 2,000 and their functions are principally regulatory and research.

Veterinary medicine is more than just a job. Like most medical careers, it is a way of life, requiring a purposefulness and a dedication beyond that imposed by careers dealing with ordinary material things. The veterinarian stands on the front lines of the constant battle for better public health. He is a guardian of human lives through the safeguarding of our animal food supply; the protector of a nation's vast investment in food animals by maintaining their health. He is often a pioneer — always a scientist — using his skill and his knowledge for the public welfare. The Veterinary Service in any country must be thorough, and it is most worthy of financial support by the people through their local and central Government; a support in keeping with a strong veterinary science training programme, safe health standards, and a progressive livestock industry.

Dr. Diesel replied on behalf of the Association and stressed the brotherhood of veterinarians the world over.

OPENING OF THE TRADE EXHIBIT

The Vice-President, Dr. R. M. du Toit, opened the Trade Exhibit. He thanked the exhibitors for their presence and assured them that the exhibit added greatly to the value and interest of the congress. Not only did these firms keep us up to date by displaying the latest in drugs and instruments but they also helped materially in supplying scientific literature and abstract services which were of the greatest value. Dr. du Toit wished the exhibitors a very pleasant and worthwhile stay in our midst.

Dr. Langbridge replied on behalf of the exhibitors and handed the President a cheque for £100 as a donation from the Medical Exhibitors' Association to the Association's benevolent fund. This generous gesture was greeted with loud applause.

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SIGNAL HONOUR AWARDED TO PROF. R. A. ALEXANDER

The President announced that Prof. Alexander had been awarded an outstanding honour during the XVIth Annual Veterinary Congress in Madrid this year. In commemoration of the Congress, the Faculty of Veterinary Science of the University of Madrid had decided to award a gold medal and elevation to Honorary Professorship to six eminent veterinarians as a merited recognition of their contributions to veterinary science. Prof. Alexander was one of the recipients. The President congratulated him on this great honour on behalf of the Association. (Prolonged applause).

In reply Prof. Alexander thanked the President for his remarks and the members for the enthusiastic reception they had accorded the announcement. He went on to say that he regarded the award not so much as a personal honour, but as an honour to South Africa, to Onderstepoort, to the Faculty and to the profession in this country as a whole.

NUUSBERIG

Hulde en erkentlikheid is aan dr. P. S. Snyman, aftredende Adjunk-direkteur van Veeartsenydiens, betoon tydens 'n plegtige byeenkoms by die Krokodilbrugingang van die Kruger Nasionale Wildtuin op die 11de September 1959.

Tydens die funksie is 'n gedenkplaat onthul by die draadheining wat aan die suidelike oewer van die Krokodilrivier opgerig word. Voortaan sal genoemde versperring teen Bek-en-klouseer en ander veesiektes bekend staan as „Die Snymanheining."

Nagenoeg vyftig lede van sy veldpersoneel was teenwoordig en is daar van 'n leier en veldsman afskeid geneem onder omstandighede waaronder die meeste hom die beste leer ken het. Daar is veral gewag gemaak van die rol wat dr. Snyman vervul het aan die voorpunt van Bek-en-klouseer-veldtogte en die vertroue wat hy deurgaans behou het in heinings om hierdie gevreesde siekte die hoof te bied.

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RADIO-ISOTOPES IN BIOLOGICAL RESEARCH

W. R. McMurray, M.Sc.(Natal), D.Phil.(Oxon.),
Radioactivity Division,
National Physical Research Laboratory,
South Africa.

Introduction:

With the advances in man's knowledge of the atom, there developed, almost simultaneously, applications of that knowledge. Some are spectacular like nuclear weapons and nuclear power. Others, though less spectacular, may prove to have as much potential for useful utilization for the good of mankind. Considerable research has been conducted during the last decade in applying atomic energy to biological problems of a pure and applied nature. Valuable results have been obtained across a wide front from basic research in microbiology, botany and zoology to applied research in food production and processing. Thus we have gained new varieties of crops; pest control problems have been solved; animal management has been simplified by elucidating the dynamic reactions of living processes; the application of fertilizer has been made more effective in supplying the mineral needs of the specific crop and so on.

The widespread use of radioactive and stable isotopes in the study of biological problems is a development of the last 15 years. It must nonetheless be remembered that many of the techniques and principles were pioneered before the 2nd World War. Thus the effects of radiation upon the reproductive system of animals were noted before 1910 but it was not until 1928 that H. J. Muller was able to demonstrate that radiation caused Mendelian mutations. The first application of this knowledge to the development of new varieties of important crops occurred in 1940. We now have radiation produced varieties of grain which are rust-resisting, ripen earlier, give higher yields and have stiffer straw.

The use of isotopic tracers, so-called tagged atoms, also has a long history. De Hevesy used the tracer technique to study the chemistry of lead in 1914 and pioneered isotope tracer applications in biology when he determined the absorption of lead by plant roots in 1923.

The value of radioisotopes in research arises from the ease and accuracy with which they can be identified and measured in extremely small quantities using the highly-sensitive methods of detection now available. This makes it possible to study some processes that could scarcely be studied in any other way or else

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only by very expensive methods. In fact, it has been said that the more complex and recondite the system under investigation, the greater is the likely value of radioisotope techniques compared to conventional methods. The advances in research made possible by the use of radioisotopes have been compared to those that came to biology and medicine when the microscope became available. Consider the outstanding examples afforded by the recently acquired understanding of the processes involved in photosynthesis of green plants, or, by our knowledge about milk secretion in cattle — both vitally assisted by radioisotope techniques.

One important achievement resulting from the use of isotopes is that now for the first time, it has become possible to follow the movement of substances into or out of a system which is in dynamic equilibrium, and to identify movement which is opposite to the apparent overall direction. These techniques are providing information on the dynamics of growth processes, the true course of metabolic reactions and the replacement rate of elements and compounds in tissues and organs of the animal body.

Radioisotopes:

Before we continue, let me explain briefly about isotopes. Take the example of Carbon. The carbon atom exists in a variety of forms which are chemically identical but differ only in weight. Each one, in fact, contains a different number of neutrons in the nucleus of the atom. Some of these isotopes of the element carbon exist in nature. These are stable isotopes. The others are produced artificially in a nuclear reactor and these are radioactive. Carbon-14, the radioisotope of Carbon with 14 heavy particles in its nucleus (8 of them neutrons), is also produced in the atmosphere by the action of cosmic radiation on atmospheric nitrogen. When carbon-14 undergoes radioactive decay, it emits a beta ray which is readily detectable by a sensitive counter. This emitted radiation is what makes radioactive isotopes so easy to trace. They may be buried deep inside the animal body or several feet underground, but even there, their emitted radiations can still be detected with a suitable counter. Actually, the beta rays from carbon-14 have very little penetration. It is fortunate that most radioactive isotopes also emit very penetrating gamma-radiation.

Ideally, the tracer atoms or radioactive isotopes (or just radioisotopes) should behave exactly like the stable atoms that they are intended to trace. While it is true that the various isotopes of an element have similar chemical properties, they differ in that reaction rates are somewhat dependent on the mass of the isotope — the only distinguishing characteristic between isotopes. This isotope effect is greatest for light weight elements, it is just detectable in the case of carbon and must certainly be considered in investigations involving heavy isotopes of hydrogen.

No other method can approach the sensitivity of the isotope method in detecting the presence of, and tracing the path of a

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given element. Actually, this is possible in one of two ways. Either we start with a radioisotope and trace the path of the radioactivity from insertion to ultimate destination — hence the name *"tracer,"* or we start with a stable isotope and then activate the products with neutron irradiation. The presence of the stable isotope in the product can be deduced from the radioactivity which is produced. This method is known as *"activation analysis."*

The demand for radioisotopes has led to the growth of a considerable industry to provide *reactor produced radioisotopes* of almost every element in a convenient form. Although we in South Africa have to import all our radioisotopes from America or England (mostly the latter), this is not really a large handicap to radioisotope research in this country.

Except for the important elements oxygen and nitrogen, useful radioisotopes are now available for most of the elements which enter into biological processes. In the case of oxygen and nitrogen certain investigations have been able to make use of a stable isotope as tracer. There are, altogether, some 130 radioactive isotopes listed in the catalogues of isotope production centres such as Amersham (connected with Harwell) in England.

Often it is necessary to trace only a certain kind of molecule or only a part of a molecule rather than particular atoms. To meet this need, a growing assortment of hundreds of organic molecules have been synthesized with radioactive replacements for one or more of their atoms. These *tracer* compounds are commercially available and include proteins, sugars, amino acids, vitamins and hormones.

Radioisotopes in Botanical Research:

The most extensive use yet made of isotopes in agriculture has been concerned with investigations of *fertilizer uptake*. The Radioactivity division of the National Physical Research Laboratory did a number of original studies in South Africa using phosphorus-32 in phosphates. The Western Province Fruit Research station has an active team investigating problems of this nature with reference to fruit trees. The radioisotope technique makes it possible to determine how and when to apply the fertilizer for optimum utilization.

Photosynthesis was still a mysterious chemical process in 1940. The isotope oxygen-18 established that water played a prominent role with the release of oxygen. The use of carbon-14 assisted the discovery of the other steps in the process as described to the American Association for the Advancement of Science in 1954. As in many life processes, the steps in photosynthesis are very rapid — it has been demonstrated that many compounds may be formed within a few seconds.

Nuclear Radiation has given a great stimulus to the development of new varieties of crops by the process of *radiation induced mutations*. Besides improved varieties of grain, an example of

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local interest is that of peanuts. A new variety developed in North America yields 30% more peanuts per acre, has a size and shape better adapted to mechanical harvesting and has better resistance to common leaf-spot disease. We in the Transvaal, require a peanut plant which is resistant to the disease rosette. I am personally supervising the physics of seed irradiations conducted by the National Physical Research Laboratory for this and other purposes.

Human and Animal Biology:

Of the 92 elements, 15 or 20 are known to be essential to the life process. Sodium for instance is important in body fluids and tissues and it is easily traced by means of the gamma-radiation emitted from the radioisotope Sodium-24. An intravenous injection of radioactive sodium chloride illustrates again *the dynamic quality of life processes.* Only a few seconds after injection in one arm, the sodium 24 gamma-rays are detectable in the other arm. Another example: injected radiocarbon-labelled-acetate is found to be converted into carbon dioxide in the exhaled breath. The radioactive carbon dioxide can be detected almost immediately after injection, reaching a peak in about 15 minutes.

Metabolism studies have concerned most of the elements known to be important. Some part of metabolism studies is motivated by the hope of finding differences between the needs of cancer cells and normal tissue and so possibly discover a method of cancer control. In a typical protein metabolism study, glycine was labelled with carbon-14 and injected into normal rats and rats with liver tumours. The rats were then sacrificed at various intervals so that metabolism was stopped at various stages of completion. The tumours and normal tissues were then chemically analysed for the amino acids which lead to the eventual building of body protein. The radioactivity of any fraction demonstrated the presence of parts of the original glycine molecules. In such a way it was possible to produce a complete "flow sheet" of glycine metabolism. As with most such metabolism studies, the same general scheme was found for both normal and cancerous tissue.

Hormone studies are being assisted by radioactive tracers. Since a small quantity of labelled hormone when injected will mix with the same substance produced by the glands, it is possible to find out how the body treats its own regulators. In this way the body control of the insulin hormone is being studied with insulin labelled with Iodine-131. In fact, additional information is obtained by labelling the substances acted on by Insulin. This is a two-way tool which permits effective study of similar problems in cause and effect.

The normal pattern of blood cell manufacture and circulation is affected by some of man's most important diseases. 65% of the body's iron is in the haemoglobin of the circulating red cells and some of the remaining 35% is in the blood-forming organs. The

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radioisotope iron-59 can, therefore, be used very effectively in studying the production and make-up of blood both in sickness and in health. In one study of the anaemia which occurs frequently in cancer cases, the anaemia was shown to be due to shortened red cell life, not to reduced cell production as previously thought. Another study assisted blood transfusion services by using radioisotope tagging to test the effectiveness of methods of preserving stored blood supplies.

In cholesterol metabolism studies, tracer techniques are employed to give information as to the origin of substances where chemical tests merely demonstrate their presence. When cholesterol labelled with tritium (radioactive hydrogen) is fed to normal persons and to patients with arteriosclerosis, the tritium activity in their blood fractions is found to be different. The normal subject has a higher concentration of radioactivity in the free cholesterol (in the blood plasma) than in the total cholesterol. The patient with arteriosclerosis has the opposite situation. Such a test could lead to the early diagnosis of a tendency to this disease.

Animal Studies:

Animal studies must often serve as a basis for medical practice. This process is now being reversed in so far that some well-tried procedures in medical therapy are now being applied to valuable animals such as racehorses and prize bulls. Valuable breeding bulls with eye cancer have had their usefulness prolonged after treatment with the beta radiations from strontium-90. Eye applicators of this radioisotope are not uncommon specialist equipment in hospitals.

That ketosis in dairy cattle — a disease affecting the normal body checks and balances — can be effectively treated with the hormone ACTH has been shown by the use of radioisotopes. The cattle and sheep disease, bloat, is also being studied with the assistance of radioisotope techniques.

Radiation might also play a role in the *development of vaccines.* Irradiation of fungi has resulted in mutants which are being used to develop greater crop resistance to fungus-caused diseases. A similar possibility exists for work with organisms causing animal diseases. Variants of reduced virulence may prove of value as vaccines which induce immunity to more virulent forms of the pathogen. Another interesting application of radiation to the same end, lies in the ability of a very high dose of radiation to knock out the effectiveness of the micro-organism without affecting its antigenicity. Thus the British Radiation laboratory at Wantage has shown that the Foot-and-Mouth Virus, after exposure to a gamma-radiation dose of several million rontgens, is 60% more effective as an antigen whereas its infectivity is reduced to a very low level.

A major portion of the radioisotope applications in animal husbandry have concerned metabolism studies in poultry, cattle and sheep. Perhaps the best known of such studies relates to

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cobalt metabolism of the ruminant. Cattle and sheep require between 0.04 and 0.07 parts per million of cobalt in their diet whereas simple stomached animals (horses, pigs, etc.) require much less. Chemical studies of such minute quantities are extremely difficult whereas radiocobalt tracer studies are quite simple and also permit the investigator to determine the metabolic path of the element. It was shown in this way that deficiency in Vitamin B-12 is due to cobalt deficiency in the diet and that B-12 is synthesised by the animal from ingested cobalt. Oral administration is, therefore, the most effective way to provide cobalt where this element is lacking.

Mineral Metabolism in animals has been studied with a large variety of isotopes including the following: Phosphorus-32, sulphur-35, calcium-45, iron-55, cobalt-60, copper-64, zinc-65, strontium-89, molybdenum-99, iodine-131, cesium-134 and tantalum-182.

The use of iodine-131 in studying *thyroid activity* in cattle is leading to a better understanding of the relation between thyroid activity and environmental conditions. In particular, the thyroid uptake of I-131 is being used to assist in the selection of animals in the breeding of heat-resistant cattle. Other investigations have indicated that high milk yields are dependent upon the optimal functioning of this endocrine gland. If thyroid behaviour is related to productivity, calves that will be poor milk producers may be eliminated early from the herd.

With further reference to milk, a great part of our new knowledge about *the milk secretion processes* has been gained by using carbon-14 and other radioisotopes to follow the breakdown of organic metabolites and the extent to which these metabolites contribute to the formation of milk. It has been shown that the micro-organisms in the rumen of the cow act on ingested starch and cellulose to produce the principal end products — acetic acid, propionic acid and butyric acid. Analysing the milk and determining the amount of activity found in each milk component has shown which components arise from these three acids or from glucose. Some of the later milk formation studies have been assisted by an artificial cow. This artificial cow consists of a system of bottles, tanks, tubes, pump and an isolated fresh udder. They cannot yet do without cows but the information obtained from research of this kind has undoubted value in improving milk production by indicating correct choice of economic feeds, improved management practice and the choice of better stock for breeding.

Food-Borne Diseases and Food Processing:

There are at least 35 food-borne diseases which may be transmitted from animals to man through the handling or ingestion of meats and sea foods.

In an autopsy of over 11,000 human bodies in the U.S.A., 16% contained trichina cysts as a result of the consumption of partially cooked pork. Note, on the other hand, that only 300

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clinical cases of trichinosis occur in the U.S. per annum. Ionising radiation has been well established as a means of destroying helminths, such as trichina worms (and presumably beef tapeworm too) without affecting the quality of the meat.

The bovine strain of TB is also capable of infecting man. One survey concluded that nearly 12% of 1,200 TB patients were infected with the bovine strain. Preliminary studies on the tubercle bacillus showed that moderate dosages of radiation will prevent its growth.

Other diseases include salmonellosis from liquid egg and fish meal. A medium gamma-radiation dosage is effective in the destruction of salmonella bacteria.

Moreover — the ability of radiation to destroy insects and bacteria is being applied to the more effective processing, pasteurisation and sterilization of a wide range of foodstuffs. As an example, radiation sterilized meat has been stored for 5 years at about 40°F without suffering bacterial deterioration.

Entomology:

Entomologists, particularly those who specialise in farm pests, are making good use of radioactive tracers. With all pests, a *knowledge of the life cycle* simplifies the approach to effective measures of control. Consider such insects as worms which menace root crops. Research scientists have put radioactive cobalt tags on worms with a plastic, and have been able to follow them, out of sight, underground. It is even possible to determine their depth under the surface.

Insects as small as aphids can be labelled by feeding the aphids on plants fed on radioactive phosphorus. Similar labelling of grasshoppers has shown how they move through farmland. Flies were labelled by feeding them, after a period of starvation, on sugar and radioactive phosphoric acid. Some of these flies were traced 20 miles from the point of release, although the majority remained within a mile radius.

Other methods have been used for labelling of insects but the great advantage which can be derived from the use of radioactivity is the ability to detect the presence of the insect when it is not visible to the eye. There seems to be scope for these methods to be applied in the study of the habits of ticks.

A particularly interesting technique of pest elimination by *breeding to extinction* has been developed and applied with great success in the control of the American screw worm fly — a pest with which we in South Africa are not unfamiliar. In 1953, the entomological branch of the U.S. Department of Agriculture discovered that if screw worm pupae are irradiated with a certain dose of gamma-radiation, the resultant male flies are all sterile. Tests also showed that the female fly mates only once with no preference for a fertile over a sterile male. With this background it was decided to use the heavily infected Island of Curaçao for an experi-

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ment. Sterile males were released from an aircraft at the rate of 400 per square mile per week. Eggs laid in goats penned at various points across the Island gave a measure of the intensity of infestation from the proportion of infertile eggs. The experiment was so successful that in the 8 months following the release of the infertile males, not a single case of screw worm infestation was found in a domesticated animal and it was felt that the fly had been eradicated from the Island.

It is reported that the same technique is being applied to portions of the U.S. continent and that sheep bot fly and the tsetse fly may respond to similar treatment. The fact that the female screw worm fly mates only once is important to the success of the Curaçao experiment.

Health Hazards and Radiation Protection:

Radiation and radioisotopes have not been wholly beneficial to man. I need hardly mention the Atomic bombs over Japan nor the 100 radiologists who died before 1922 because of radiation-induced cancers. In fact, the first fatality, who happened to be Thomas Edison's assistant, occurred only one year after the discovery of these radiations. Now, too, we are concerned for ourselves and our progeny because of the strontium-90 and caesium-137 legacy of the nuclear weapon testing programmes. Other than the purely practical expedients of shielding or keeping one's distance, there is no method of protection against the damaging effects of these nuclear radiations once we have been exposed. Radioactivity must, therefore, be handled with enlightened circumspection. With 60 years of experience now behind us, we have obtained a very good idea of how much radiation dose man can absorb in a working life with suffering ill effects. An International body, the International Commission on Radiological Protection, has been in existence for many years and continuously reviews their recommended *maximum permissible exposure levels* in the light of our increasing understanding of the subject of the chronic exposure of man to small doses.

Conclusion

There can be no doubt that remarkable advances in biological research have already been made possible by the use of radioisotope techniques. I believe that we cannot afford to remain spectators of the achievements afforded to foreign scientists and that there is a large potential for the application of these methods to South African problems.

Obviously, the first step towards participation in radioisotope research is the training of qualified research workers — a process which takes years. The application of radioisotopes is the work of a specialist who must also specialise in the field of the application. I wish to quote from a book by Professor Comar, "Radioisotopes in Biology and Agriculture." In the preface he makes two points which I want to repeat here.

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1. "Radiation techniques, to the naive, may represent the panacea for investigational difficulties . . . to the cynic they may appear as gadgetry . . . As always the truth lies somewhere between the extremes . . .
2. "The really important contributions of these procedures in the biological field will be realised only in so far as they are utilized by investigators who are well grounded and have been working in the field of application. This is not to belittle the physical scientist, whose tremendous contributions is acclaimed by all. Nevertheless, it is the biologist who recognises the important problems in his own field, is familiar with the experimental material and must be counted upon to make the interpretations."

In the light of these remarks, I will be so bold as to recommend that our Atomic Energy Board be approached to establish a fellowship or fellowships that will attract suitably senior scientists to give full-time attention to the application of radioisotopes to biological problems of importance to South Africa. The Atomic Energy Board is empowered to stimulate research in, and the application of, radioisotope techniques and I expect them to be interested in sponsoring such research in the interests of South Africa. In such projects the National Physical and Chemical Research Laboratories will give full technical co-operation with the greater knowledge and experience that we have in radiation physics and radiochemistry.

In conclusion, let me reiterate, that the contribution of radioisotopes and radiation in biological research as applied to increasing the production and effective utilization of agricultural products, may turn out to be just as important to the future welfare of the human race as the more spectacular contribution of atomic energy.

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DISCUSSION ON "THE TREATMENT OF TUBERCULOSIS IN MAN AND ANIMALS WITH ISONIAZID"

By H. Kleeberg

(For paper see June, 1959, issue of this Journal).

DR. LAMBRECHTS pointed out that the use of isoniazid raised several problems in the control of tuberculosis in cattle. Our aim must be final eradication. Isoniazid would be invaluable in making possible the retention of infected but valuable breeding cattle and allowing calves to be raised from them free of infection. This would enable clean herds to be built up from infected ones. Treatment would have to be strictly supervised, as any laxity might lead to the spread of infection. The fact that the drug caused a negative tuberculin test in animals that were still infected was a matter of major concern and the drug must be strictly controlled to prevent malpractice.

DR. DU P. LE ROUX (Secretary for Health) informed the meeting that isoniazid would be placed on the schedule of potentially harmful drugs and its sale controlled. This would be done at the request of the veterinary authorities. He assured the Director of Veterinary Services of his Department's support. He recalled how Dr. Dormer had successfully protected babies in a tuberculosis hospital with the drug and mentioned the problem of resistant strains developing.

DR. WEYLAND also spoke of the danger of resistant strains and the fact that they might regain virulence.

DR. KLEEBERG, in reply, emphasised that he had kept 20 treated reactors in close contact with 200 clean cattle without any spread of infection. Open and generalised cases must be eliminated from the start. If they were to be retained and treated, reactors should be branded and never considered cured. Treatment should only be allowed in order to build a clean herd. Treated reactors should be eliminated as soon as it was economically possible.

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TICKS: THEIR BIOLOGY AND THEIR DISTRIBUTION

GERTRUD THEILER

Onderstepoort Veterinary Laboratories

There comes a time in every branch of science when it is advisable and profitable to sit back and take stock of accumulated facts and to reassess current practices in the light of recent findings. This meeting offers such an occasion in our study of ticks and their possible modes of control.

In applied Entomology it has been shown so often, as to be axiomatic, that the more that is known about the bionomics of an insect, the more intelligent is the approach to its control; so that in this science the days of "rule of thumb" methods are on the way out. In this symposium we hope to shew you that during the last couple of decades a considerable body of information has been built up on the ecology and the life histories of ticks; so that even though we can not hold out hopes for the end of all our troubles we can at least suggest a more hopeful approach to them.

Taxonomy:

It may seem a trite remark to make, but it bears repeating "that when we set out to study a tick problem we need to know with which species we are dealing." In ticks the genera are clear cut, and shew constant features, the species, however, are ill defined, with features which shew a large range of variations. The rearing of F_1 generations is bringing clarity into the taxonomic field and the extent of the deviation from the typical has been established for most of the economically important ticks, both for the adults and for the immatures. By degrees the lesser known ticks are also being studied. Feeding times and developmental periods are also fairly well known as a result of these F_1 rearings. But time has not permitted the study of the survival periods of the unfed larvae, nymphae or adults.

At the time when the Tick Survey material was identified some species were but ill defined. Thus, in the paper on the distribution of *R. appendiculatus,* the brown ear tick, the tick is recorded for the Brokenveld of the Karroo — under the covering remark "the question hence arises as to whether the tick is a true *R. appendiculatus* or whether it is a strain which has become adapted to the arid conditions obtained in the Fauresmith "broken-

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veld" or whether it is not a new species or sub-species. A further more detailed study will have to be made before a considered opinion can be expressed. In the meanwhile indications all point to the fact that the tick present is not the usual *R. appendiculatus*, but a brown tick with different environmental potentialities." Upon breeding, this ecologically aberrant tick shewed itself to be *Rhipicephalus pravus* — a tick favouring drier areas than does *R. appendiculatus* and spreading through Kimberley-Bechuanaland into East Africa. Another species which was shown by the Tick Survey to need revision is *R. capensis*. For this species I could get no clear-cut distribution picture, humidity/aridity preference or behaviour pattern. F_1 generation rearings have shewn that in some instances extremely heavily punctate *R. simus* had been identified as *R. capensis*. It would seem that the at present "catch-all" *R. capensis* may be divided into two or three species or sub-species. *R. simus* in the Union also shews a large range of variations, these in more tropical areas "run into" *R. senegalensis* and *R. longus*. Where the one stops and the others begin we do not as yet know. Much work still needs to be done on the group. *R. sanguineus* also shews a wide range of punctation pictures; for this species, as for *O. moubata*, workers are postulating the presence of more than one strain within the species, each strain having different adaptational and disease transmitting potentialities. In the same way we find that some of the other genera also are receiving attention. In the Union the Hyalommas, the Bontpoots, are easily differentiated into three species; in tropical and in North Africa, however, the species are still undergoing revision. Even our very familiar yellow dog tick, *Haemaphysalis leachi*, is being overhauled. Amongst the *Ixodes* the two species present on our domestic stock i.e. *I. pilosus* and *I. rubicundus*, and their environmental preferences are so well known that when I am told that *I. pilosus* occurs in parts of Kenya I question the identification. The "wild" *Ixodes* species, however, are being shewn to be a complex group and are still receiving much attention; as yet we know but little about them, for they never occur in great numbers and collections are few and far between.

Host Preferences:

The information gathered during the last two decades, though not as complete as we would like it to be, is yet adequate enough to allow us to draw some conclusions as to the host preferences of some of our ticks, knowledge which is as essential in our search for disease reservoirs as it is in our campaigns for tick control. Fortunately a large number of ticks are very host specific and are no menace to our health nor to our economy. On the other hand, the few ticks that infest our domestic stock mostly shew a wide range of adaptability to a large number of hosts, shewing somewhat similiar likes and dislikes as adults and as immatures; but frequently the immatures may choose quite a different set of hosts to those

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of the adults. *Boophilus decoloratus* is a simple problem, it is a one-host tick confining its attentions almost exclusively to herbivores; *I. pilosus*, as far as we know, should present no difficulty even though it is a three-host tick, for the immatures feed on the same hosts as do the adults — mainly cattle and sheep and bushbuck; in certain Karroo areas the adults may be found on dogs. In the genera *Rhipicephalus, Amblyomma* and *Hyalomma* the feeding patterns differ greatly and host preferences are widespread. *R. appendiculatus* will remain one of our headaches for a long time to come, for its immatures are catholic in their taste; *R. evertsi* also is a fairly universal feeder. *R. simus* immatures, however, confine themselves mainly to the small rodents, with the elephant shrews and the hares a very poor second. The immatures of *A. hebraeum* seem satisfied with the *Bovidae*, but may feed on a whole host of other groups of animals, with a fair percentage on birds. As to the *Hyalommas*, in 1947 I stated that, "the Tick Survey had not produced one instance of a hyalomma larva or nympha having been collected off our domestic stock, nor do they seem to infest mammals under field conditions. Their preferred hosts seem to be birds . . . This statement for the *hyalommas*, holds only for South Africa, for in Asia and Europe, they seem to behave differently." Material collected during the last decade has added many more records to the bird host list; it has also shewn that the *hyalommas* can and do feed on the *Cricetidae* (i.e. the Gerbilles) and the *Leporidae* (hares). Records off other hosts are but scant with but a few off *Bovidae*. Until quite recently I had no immatures listed from domestic stock, when Dr. Stampa brought in a sample of "literally thousands" off the face of a sheep in the Tantjiesberg, to follow up his finding by specimens off an Angora goat in the Jansenville district. As more collections come in we may get more specimens off domestic stock and our *hyalommas* will then be shewn in their feeding behaviour to approach that of the Asiatic and European species more closely.

Of importance is the finding that the small field rodents do not play the important rôle ascribed to them at first, but that more attention should be paid to the elephant shrews (*Macroscelidae*), the hares (*Leporidae*), and to the birds. At present great attention is being given to migrant birds and their tick burdens.

Tick Activity:

Although we have gone beyond the stage of accepting the statement "there are no ticks in Winter, (so no collections were made for the Tick Survey during this season") we still are not in a position to give definite active periods for each (economically important) tick, for each region in the Union. As yet time has not allowed for the tabulating and sorting of the dates of collection of the abundant material sent in. We can give an approximation as to the main period of activity for most of our adult ticks, e.g. *R. simus* early summer to autumn; *R. appendiculatus* late summer

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to autumn; *B. decoloratus* in waves throughout the year; *I. pilosus* throughout the year; *I. rubicundus* mostly April to June but may stay on until September; *Margaropus winthemi* also is a winter tick.

What the active periods are for the larvae and nymphae we as yet have no definite idea. *R. appendiculatus* and *A. hebraeum* we know are active in late autumn and winter but may appear already in March. In *I. rubicundus,* the only tick which has been systematically studied in the Union, according to S. Stampa, the larvae have an active period extending from March to mid-August with a peak in April-May; and the nymphae from July to November, with a peak during September-October.

It is agreed that most larvae aggregate at the tips of grass blades and bushes to await their hosts when they transfer *en masse.* The adults of the *Rhipicephalus, R. simus* and *R. appendiculatus,* also climb to the top of the grass or bush, but I have never seen an *R. evertsi, Amblyomma* or *Hyalomma* questing in this position. When sitting on the ground I have seen *Hyalommas,* as also *R. appendiculatus,* approaching me from about two yards away. It is generally agreed that ticks, whether as adults or as immatures, never travel any great distance and it is assumed that a burnt margin of a few yards round a camp is an efficient cordon against their spread under their own foot power. What the factor is that decides the seasonal activity we do not know. Humidity/aridity has been shewn to be the decisive factor for survival. From the data to hand it would appear that this plays a rôle in the seasonal activity of the immatures of most of our Karroo and grassland species. W. J. Gray at Mazabuka in Northern Rhodesia is studying the ecology of some of the tick larvae, but is not yet in a position to give an answer to the questions of locality preferences or of factors influencing larval activity.

Dispersal:

The actual dispersal — and hence distribution — of a tick is tied to that of its host or hosts (in so far as the tick cannot travel any great distance under its own power). In this way a tick with the same host at all its stages is tied entirely to the distribution of that host, e.g. *Aponomma exornatum* and the Leguaans, which can occupy only a small part of its potential area; whereas the tick with a wide range of hosts can occupy to the full its potential area of distribution, and will be present in large numbers, e.g. *R. appendiculatus.*

The smaller, short ranging animals will probably play a larger rôle in maintaining the local population, e.g. elephant shrews and *I. rubicundus* or field mice and *R. simus,* than will the wide ranging ones. The latter, however, play the important rôle in the dispersal of the tick and in establishing new foci, e.g. the sheep and the Vaal Ribbok for *I. rubicundus* and the Hyaena or Wart-

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hog for *R. simus*. As far as I can make out in South Africa no one tick appears to be tied to the distribution of its intermediate host(s). In those instances where birds often act as intermediate hosts, e.g. the *Hyalommas* and the *Amblyommas*, it is the birds that spread the tick furthest afield, often into lands completely unknown. We have evidence that the African *H. rufipes* has been carried to, and become established in, parts of Southern Russia.

Our interest in intermediate hosts is not only in the rôle they play as local tick-feeders and as local distributing agents, but also in their possible rôle as carriers and reservoirs of diseases. Hence the more detailed our knowledge, not only of host preferences, but also of the distribution map of each potential host, the more useful will that knowledge be. When at some future date the host-vector-disease relationship has been established, our chances of coping with disease outbreaks will thus be all the better.

Defence Mechanism:

Workers in other countries, notably A. D. Lees in England, have studied the cuticles of several groups of arthropods and have gained much information as to their structure and function. It has been shown that the tick cuticle has a waterproofing layer of wax which, although effectively preventing water loss, does not hinder the uptake of water. Resistance in unfed ticks to desiccation at low humidities is achieved by a dual mechanism: active uptake and the physical retention of water by the wax layer. In the different species these wax layers have different critical temperatures at which the wax undergoes an alteration (ranging from 32°C for *I. ricinus* to 75°C for *O. savignyi*) and at which its impermeability breaks down. Species having higher critical temperatures are more resistant to desiccation, at temperatures within the biological range. The *Argasidae* have an extra cement layer besides the wax layer and are hence also more resistant to abrasion than are the *Ixodidae*.

As the cuticle of the engorging tick stretches, it loses the power by the epidermal cells of actively taking up water, this added to the wax layer makes the engorged female resistant to the uptake and to the penetration of tickicides. Engorged females are notoriously the hardest to kill.

An interesting feature of the tick cuticle is that the abrasion of a limited area of cuticle temporarily inhibits the activities of all or nearly all, the epidermal cells, many of which underlie normal, undamaged cuticle. Evidently the epidermal cells are interconnected in some way, and the interruption in the function of one group of cells may influence the functioning of an adjacent group (in the still unengorged tick). This power of taking up water is regained within 24 hours. In the *Ixodidae*, since the wax layer is freely exposed, chloroform and detergents in which the wax will dissolve, have a marked action in increasing transpiration particularly in those species with low critical temperatures, e.g. the genus *Ixodes*. In the *Argasidae* the extra cement layer is very

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resistant to extraction and is only broken down by boiling chloroform. So that it would appear that by adding detergents to our tickicides we should be enhancing the value of the dip/spray for the *Ixodidae*, but not for the *Argasidae*.

The study of the tick cuticle indicates that it would be more profitable to dip/spray the unengorged tick rather than the engorged, and that tickicides with wax solvents or abrasives increase our chances of success. Work is continuing on new formulations and adjuvants.

Distribution

Macroclimate:

For the Union, in broad outlines, we have been able, from our knowledge of host preferences and from our distribution maps, to draw some conclusions as to the factors influencing the distribution and the factors limiting the seasonal activity of the various economically important species. Some have been shown to be influenced: (A) directly by decreasing humidity, e.g. *R. evertsi* and *B. decoloratus;* (B) by increasing humidity, e.g. the *Hyalommas;* (C) by the humidity/aridity in the tall grass veld associated with the presence and/or shade of bushes or trees, e.g. *R. appendiculatus* and *A. hebraeum;* (D) by specific conditions, apparently of even moisture, offered; (i) by Sourveld for *I. pilosus;* (ii) by *Rhus erosa* and by *Danthonia*-veld for *I. rubicundus;* (iii) by the shade of big trees in the deep sand veld of the Kalahari and of neighbouring areas, for *O. savignyi;* (E) by the even conditions in the nests of small veld rodents, which are its intermediate hosts, for *R. simus*.

At present we know just enough so that we can tell whether a tick introduced into a new area will have a chance of maintaining itself or not. We must, however, bear in mind that due to man's agricultural activities the climatic conditions affecting tick life are being changed and in many areas the balance for or against the tick are being altered; frequently only one small factor needs to differ slightly to affect the tick's survival or spread. A striking example of this is the very consistent manner in which *I. rubicundus* is following the incursion of the plants *Rhus erosa* and *Danthonia disticha* from the typical Karroo into the grassveld of the Southern Free State. *I. rubicundus* can also be used to illustrate another method of spread, besides the one we have just mentioned (where it followed its intermediate hosts in their migrations with the changing veld), i.e. it has been introduced by man on his sheep into completely new areas onto several farms (Kwaggaskop, Suikerboskop, Rietfontein and Spitzkop) in the Belfast area in the Transvaal, and onto several farms in the Dargle Valley, Lion's River district of Natal. These are areas in which the intermediate hosts, the *Pronolagus* hare and the elephant shrew, are already established, and where the microclimate is suitable for the survival of the immatures. That we have to take note of environmental changes is also illus-

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trated by the very slow spread of *R. evertsi* into little Namaqualand. We also need to watch the man-introduced species, e.g. *Haemaphysalis aciculifer,* brought in with cattle after the Rinderpest, and still scattered haphazardly in parts of the Union. It is not likely ever to spread very far, for it needs warm moist surroundings. *Argas megnini,* however, since first reported at the time of Union, has spread far and wide; dry area tick that it is, it threatens to take in the better part of the Union. It has also recently been introduced into South West where it undoubtedly will be solidly entrenched before long. The other introduction (if it is an introduction), *Hyalomma glabrum* (= *turanicum?*) does not look as though, at the moment, it will spread beyond its present range in the Karroo; in many ways it seems less resilient to climatic changes than either *H. rufipes* or *H. truncatum.*

Microclimate:

The distribution and the maintenance of a species within a large macroclimatic region is obviously dependent on the number of times the suitable microclimatic conditions occur within the region (e.g. for *R. simus,* the number of field mice nests or runways) as also the number of times the host comes within the microclimatic survival area of the tick, as also the lengths of the survival periods of the unfed phases of the tick. In South Africa we know but little as yet, of the exact conditions under which our ticks live; at which stage and phase (flat or engorged) they are hardiest to withstand climatic changes; whether these short or prolonged changes are sudden or gradual; what rôle dew formation plays, etc.

W. J. Gray, at Mazabuka, Northern Rhodesia, has been busy for the last few years, painstakingly observing the behaviour of larvae of a few species exposed to a wide assortment of microclimates. He is getting some interesting results, which, when correlated and assessed, will undoubtedly give us some lead as to where and when to change a vegetation picture so as to discourage the tick or to break into its life history by offering unsuitable conditions at a critical period in its development.

Control by Land- and Livestock-Management:

The idea of reducing tick infestations by interrupting, or by interfering with, the life cycle has been tried out in several countries.

In Africa veld-burning was one of the apparently obvious ways of breaking into the life cycle, but, haphazardly as it has been applied, it has shown no visible results. This lack of success could be ascribed to various reasons; either no account was taken of the life cycle of the tick(s) at the time of application, or the fire was not hot enough to kill the hidden tick or not effective enough to disturb or to destroy the tick's shelter. For veld-burning to be applied judiciously we need to know more about the ticks' hiding places and their accessibility to fire. Or, if our aim is merely partial control (and not near or complete eradication) then the

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burning could be carried out when larvae are actively questing at the tips of the grass-blades or bushes.

More information is needed before the times of application of fire, either to destroy the active larvae directly or to destroy the habitat, can be recommended. Veld-burning, however, does seem to offer a way of breaking into the life cycle of the tick.

Rotational grazing may offer a means of interfering with the tick's life cycle, especially with a one-host tick or with a tick having a small range of alternate hosts, if the tick has very definite seasonal periods of activity. The stock can be moved from a clean camp into an infested camp at the time of tick inactivity on the latter. This movement can be planned to fit in with the programme of land-management for pasture improvement. In some instances where ticks are confined to certain localised habitats within a larger area, the stock can be kept away from the infested areas during the time of tick activity, e.g. *I. rubicundus* occurs mostly on hill slopes and not on the flats and is active only in winter, so stock can be alternated between winter grazing in the flats and summer grazing on the hills.

In Australia strategic dipping is combined with rotational grazing. Dipping and stock movements are based on the actual and present behaviour of the tick, in this instance *B. microplus* (*australis*), a one-host tick.

In Russia, under conditions of intensive farming, in addition to rotational grazing there is added the idea of further interfering with the tick's life cycle by mowing and removing the hay or by disking or ploughing and by disturbing its resting-stage habitats and thus exposing the ticks to the inclemencies of the weather, be it the cold of winter or the heat of summer. In some instances all that is needed is the removal of scrub and bush in grass lands, thus doing away with the shelter both of the intermediate hosts, and of the tick itself, as is also being done for *A. americanum* in N. America. This method suggests itself for *R. appendiculatus* and *A. hebraeum*. Trees, left as shade for the stock, would not offer shelter for the ticks, in that the ground underneath is invariably trampled flat. As has been pointed out before, frequently an apparently small alteration will bring about the necessary changes to render the habitat unfavourable for the tick. It is obvious that a knowledge of the ecology of the tick(s) is a *sine qua non* for the the planning of its control, by starvation, by exposure or by direct destruction at the resting or questing phases.

In South Africa the difficulty is decidedly complex for, at one and the same time, we are dealing not with one species alone, but with several; each with its own preferences as to seasonal activity, habitat and hosts; each presenting its own problem. It would appear that if there were a change in approach, and if instead of attempting the eradication/control of all species present, we were to study one in detail, find out how best to handle it, and then having controlled it we were to go on to the next, we might reach the

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desired end much sooner. With the advent of the tickicide resistant strains, it would appear only logical that the *ad hoc* treatment against ticks in general should be replaced by the species treatment. Each treatment should be based on the particular habits of the tick concerned.

Much useful information is accumulating but further ecological work still needs to be done to help in planning stock and veld management programmes.

Parasites and Predators:

The results of the work done in America and in Asia on the parasite *Hunterellus hookeri* was so disappointing that no one has had the courage to try it again. Birds and field mice, from what data has been obtained, play but a minor rôle in keeping down tick numbers.

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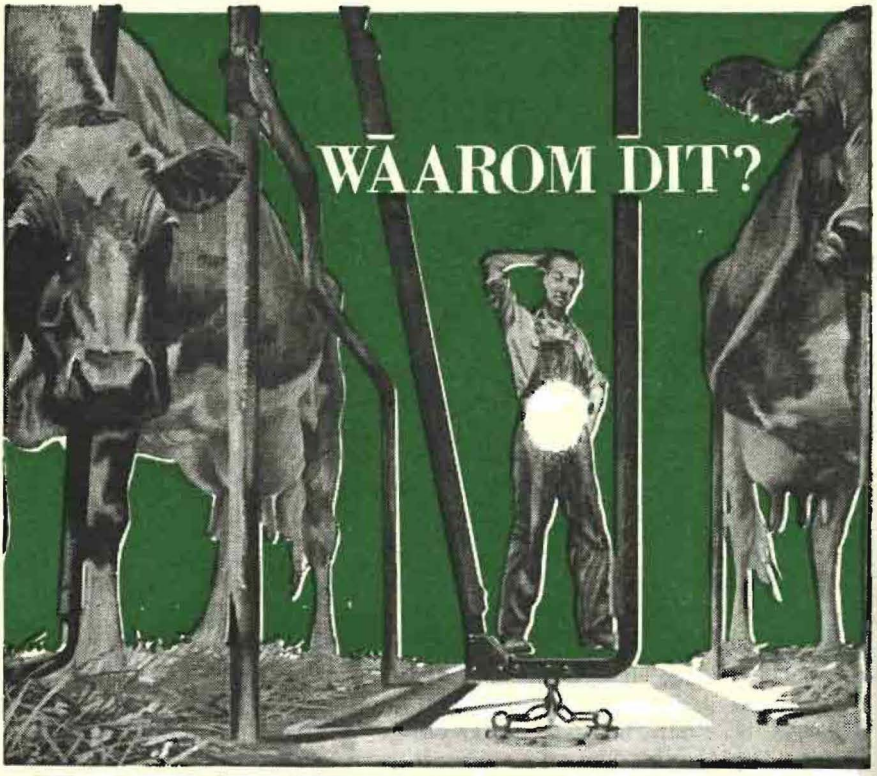
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METIBIOTIC

(*METICORTEN-asetaat + penisillien-dihidrostreptomisien*)

...SAL U KOEIE HELP OM MELK TE GEE

- **vollediger ontstekingswerende effek**
- **vinniger beheer oor infeksie**
- **enkele-dosis-besuiniging**
- **kleiner produksieverlies**
- **minder uierbeskadiging**

SCHERING CORPORATION V. S. A.

SCHERAG (EDMS.) BPK., POSBUS 7539, JOHANNESBURG

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THE PRACTICAL APPLICATION OF SOME OF THE NEW SYNTHETIC INSECTICIDES IN THE CONTROL OF TICKS

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In introducing this subject it is extremely difficult to know where to begin. So much field work has been done and so many new insecticides and formulations have come onto the market with their various proprietary names, that the uninitiated finds himself at a loss to choose the right insecticide for the control of his particular tick problem. It is my intention to deal only with three insecticides, viz. BHC, DDT. and Toxaphene, and for the sake of simplicity, I shall refer to them as such, in the wettable powder or suspension and emulsion forms.

I shall first deal with each insecticide's merits and demerits in the control of ticks, then with combinations of such insecticides with each other and with Arsenite of Soda and, lastly, with the practical application of such insecticides in the field by means of plunge dipping, machine spraying and hand spraying.

A. The Individual Insecticides.

1. B.H.C.

(a) *Blue Tick Control*: Before the investigations by the writer, B.H.C., used as a wettable powder, was found to give excellent results even when used at the then recommended concentration of 0.005% gamma at weekly intervals. It was soon evident, however, that the various BHC. washes in the dipping tanks of the Eastern Cape were not controlling the ticks and it was obvious that, in the absence of a simple tank-side test, the insecticidal concentration of the dip washes was allowed to fall below the recommended strength, which we now know was in itself inadequate for efficient control of all species of ticks. Thereafter, observations carried out at certain dipping tanks revealed that BHC., used at a concentration of 0.01% gamma at weekly intervals, did not control the blue ticks and it was established by Whitnall and his co-workers that those blue ticks were resistant to BHC.

Subsequent experiments on the farm showed that the blue ticks resisted concentrations of 0.025% gamma and, although at that stage a concentration of 0.1% did give the desired control,

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it was later found that even the latter concentration was not entirely effective.

Thus, for the control of blue ticks in those areas of the Union where the tick has developed a resistance, which includes practically the whole East Coast for distances up to 100 miles inland, BHC., in any form, is not recommended by itself but rather in combination with some insecticide that does offer the desired control.

Where, however, the tick has not developed a resistance, BHC. will give a satisfactory control at concentrations of 0.02% gamma or more.

(b) *Bont, Brown and Red Tick Control*: Weekly treatments in BHC. at concentrations of 0.02% gamma or more will give satisfactory control of all the abovementioned ticks except in the case of the brown tick and red tick under the following circumstances:—

(i) When these ticks have been allowed to pack in clusters in the ears and under the tail respectively, in which case several consecutive weekly dippings or even hand-dressing is necessary to bring them under control.

(ii) During the hot humid months when the period of attachment of the adult stages becomes so short as to allow full engorgement between treatments. For a complete control it is necessary to treat at 5-day intervals during such periods.

2. D.D.T.

(a) *Blue Tick Control*: This insecticide has been studied both as a wettable powder and as an emulsion and both exert excellent control of blue ticks when used at the recommended strengths at weekly intervals.

It must be mentioned here, however, that on one farm where DDT. Emulsion had been used under the control of the owner over a period of ten years with satisfactory results, a DDT. resistant blue tick developed. These ticks resisted a concentration of 0.3% para para of the wettable powder and there is little doubt that even higher concentrations will be resisted. Since the above observation, other workers have encountered similar experiences on other farms.

In view of the fact that DDT. as an emulsion must be used at a higher concentration for the control of other ticks (Bont, Brown and Red) and even higher still as a suspension, its use alone would entail great expense and is therefore restricted. Because of the very good control, DDT. exerts on the blue ticks, its use at a concentration of not less than 0.15% para para in the case of the emulsion and 0.3% para para in the case of the suspension in combination with another insecticide which is effective against the other species of ticks is recommended.

(b) *Bont, Brown and Red Ticks*: As already indicated, DDT., in economic concentrations, does not control these ticks and therefore, except where expense need not be considered, it should be

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used in conjunction with an insecticide which does effectively control these ticks.

3. TOXAPHENE.

(a) *Blue Ticks*: As in the case of BHC., Toxaphene, when first studied in this country, apparently gave a very satisfactory control of the blue tick, but later it was found that here and there in herds known with certainty to have been dipped weekly in 0.2% Toxaphene suspension the odd blue tick was found in an engorged state on those parts of the animal's bodies that are always thoroughly immersed in the dip wash during the act of dipping, i.e. the bellies. This very definitely indicated some resistance to Toxaphene on the part of the blue tick and in more recent years, this indication has been proved to be a fact.

Unlike the case of BHC., the writer has not encountered a severe build-up of resistance to Toxaphene although other workers maintain that such has in their experience been the case.

Therefore, for practical purposes, it is recommended that weekly treatments with Toxaphene at a concentration of not less than 0.2% be used for the control of blue ticks except in those localities where a resistance to this insecticide has been encountered in which case Toxaphene should be used in combination with some other insecticide which is effective against the blue tick, e.g. DDT.

(b) *Brown, Bont and Red Ticks*: Weekly treatments with Toxaphene either as a suspension or as an emulsion at a concentration of 0.2% has given a very good control of all the abovementioned ticks and, in the opinion of the writer, it is the insecticide of choice for the control of the bont tick.

Like BHC., the control is insufficient in the case of the brown and red ticks during the hot humid summer months as evidenced by the somewhat free engorgement of these ticks during that period. This can be overcome by resorting to shorter intervals between treatments during such periods but for all practical purposes and in the absence of disease carried by such ticks, the seven-day interval is considered satisfactory and recommended.

B. COMBINATIONS OF INSECTICIDES.

As will be seen above, combinations of insecticides are often necessary when all ticks, some of which show a resistance to one or other of the insecticides, are to be controlled. It would be useless to use DDT., at the concentrations recommended for blue tick control, when bont, brown and red ticks also constitute a problem. Similarly, BHC. would be inadequate when the blue ticks are resistant to that insecticide. Therefore, when it is desired to control all ticks, including the BHC. resistant blue tick, it would be rational to employ a combination of the abovementioned two insecticides and it is recommended that they be used in the following minimum concentrations:—

BHC. — 0.02% gamma.

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DDT. — Emulsion 0.15% or suspension 0.3% para para. Similarly when Toxaphene resistant blue ticks are to be contended with, a mixture of Toxaphene and DDT. may be used and the following minimum concentrations are recommended:—

Toxaphene — 0.25% either in suspension or emulsion.

DDT. — 0.15% para para as an emulsion or 0.3% para para as a suspension.

Unfortunately, as is no doubt discussed by another contributor to this symposium, certain formulations of the component parts of the abovementioned combinations are not compatible with the result that certain physical changes may render the mixture less effective or even toxic and therefore the veterinarian is advised to be very sure of his ground in this regard when recommending combinations of insecticides. Mention should be made here in regard to the writer's findings when Arsenite of Soda was combined with BHC. Three farms were selected. The dipping tank on one being filled with 0.02% gamma BHC. suspension plus 0.16% Arsenite of Soda, the second with 0.16% Arsenite of Soda alone and the third with 0.02% gamma BHC. suspension alone.

It was found that the best results were obtained with the BHC. alone while Arsenite of Soda alone was the least effective. Where Arsenite of Soda and BHC. were combined, the bont, brown and red ticks tended to cluster as so often happens when arsenic is used alone and a partial engorgement develops within such clusters giving the impression that the formation of such clusters gives the ticks therein some measure of protection against the BHC.

However, despite these findings, and in view of the fact that the Arsenite of Soda concentration may be easily tested at the tank-side, it is recommended that combinations of arsenic and DDT. be used where ranching is practised and in Native areas wherever the resistant blue tick is encountered. Finally, it must be explained that all three of the synthetic insecticides mentioned exert their control of the blue tick against the larvae stage, whereas control over the moulting larvae, nymphs and adults is insufficient.

Therefore, where these insecticides are used in weekly treatments, the cattle so treated can be expected to show fully engorged blue tick females on the day of the third dipping but thereafter only larvae that have attached between treatments will be found on close examination.

B. The Practical Application.

Formerly, arsenic solutions were used almost exclusively and, apart from very isolated instances, the only method of application was by plunge dipping. With the advent of the newer synthetic insecticides, however, owing to various factors associated with these insecticides, application by means of machine and hand-spraying were studied. The former method has appealed to the public and is being adopted on an ever-increasing scale.

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1. PLUNGE DIPPING.

Because only arsenical solutions were used in the past, large capacity dipping tanks were advocated with a view to giving cattle a longer swim and therefore better contact with the insecticide. With the advent of the synthetic insecticides it was found that such large dipping tanks were undesirable for the following reasons:

(a) The price of the synthetic insecticides is very much in excess of arsenic and therefore the filling of such large capacity tanks involves heavy expense.

(b) When emptying prior to spring cleaning and replenishing, the wastage is excessive.

(c) The amount of water and insecticide added after dippings is less in proportion to the total volume and this results in a higher proportion of fresh to old insecticide in the tank.

Therefore, it is recommended that dipping tanks of capacities of from 2,500 gallons to 3,000 gallons, depending on the size of the herds to be treated, be employed for use of the synthetic insecticides. It has been shown that herds of 500 head can be suitably dipped in a 2,500 gallon tank without having to replenish during operations, while a 3,000 gallon tank is suitable for herds of 1,000 head of cattle and more.

(A plan of the type of tank recommended is available).

The essential features in regard to the dipping tank recommended are as follows:—

1. High walls especially in the first half of the tank to eliminate wastage due to splash and consequent unnecessary loss of dip wash.

2. Kick-plates situated on a downward slope at the entrance to ensure a thorough plunge and immersion of the head and ears.

3. A footbath is recommended because when dealing with expensive synthetic insecticides it is essential to keep the dip wash as clean as possible for optimum results. Such a footbath must be kept clean and must be refilled with clean water prior to and even during dipping if large numbers of cattle are to be dipped. Failure to do so will defeat the object and render such a footbath a disadvantage rather than an advantage.

4. A short, narrow bottom, firstly to reduce the total capacity of the tank and secondly to facilitate thorough plunging which is always necessary in the case of suspension dips just prior to each dipping in order to bring back into suspension all the insecticide which has sedimented since the previous dipping. Unless such plunging is diligently carried out, much of the insecticide will remain

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at the bottom of the tank with resultant inefficient tick control owing to an understrength dip wash. Furthermore, in the absence of thorough plunging the insecticide will be trapped under the mud, gravel and other debris usually found in a dipping tank, resulting in tightly packed parallel strata of such insecticide as is so often encountered when cleaning out a dipping tank. To illustrate this point the following figures are given in respect of one dipping tank studied:—

Date	Tank Level in Gals. Before Dipping	Tank Level in Gals. After Dipping	Water Added Gals.	50% DDT. W.P. Added Lbs.	Chemical Test Result %	No. of Cattle Dipped
8.12.49	2,760	2,740	2,760	166	0.176	64
15.12.49	2,710	2,680			No test	65
22.12.49	2,650	2,620			0.136	64
29.12.49	2,550	2,510			0.132	64
6. 1.50	2,590	2,540	80	5	0.114	64
12. 1.50	2,500	2,460			0.110	63
19. 1.50	2,410	2,380			0.108	62
26. 1.50	2,340	2,300			0.088	76
*2. 2.50	2,550	2,500	250	15	0.190	76

*Tank thoroughly Plunged.

From these figures it will be seen that despite a filling and replenishment rate of 6 lbs. of 50% DDT. wettable powder per 100 gallons of wash, the DDT. concentration fell from 0.176% para para to 0.088% para para.

Thereafter, with thorough plunging and without any change in the replenishment rate the concenration was brought up to 0.190 para para. The difference can only be ascribed to the sedimented insecticide being brought back into suspension by the thorough plunging of the bottom of the dipping tank. These figures further show that approximately 60 head of cattle passing through a dipping tank were unable to agitate the bottom of the tank sufficiently to bring sedimented insecticide into circulation and this shows the inefficiency of common practice of passing 20 head through a tank before starting the main dipping operation. While dealing with the question of a fall in the insecticidal concentration of a dip wash, it must be remembered that a process of selective removal of insecticide by the cattle takes place during the course of both plunge dipping and machine spraying. Such removal varies according to the insecticide itself and the formulation in which it is employed. Thus in the case of a 40% Toxaphene wettable powder, a dipping tank was filled and subsequently replenished with 5 lbs. per 100 gallons to give a theoretical concentration of 0.2% Toxaphene. The following figures will illustrate the loss resulting from selective removal bearing in mind that the tank was thoroughly plunged before each dipping:

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Date	Tank Level in Gals. Before Dipping	After Dipping	Water Added Gals.	Dip added 40% Toxaphene W.P. Lbs.	Chem. Test Results % Toxaphene	No. of Cattle Dipped
30. 3.51	2,540	2,470		127	0.19	124
5. 4.51	2,540	2,450	70	3¼	0.18	155
12. 4.51	2,600	2,540	150	8	0.17	157
19. 4.51	2,600	2,540	60	3	0.17	
26. 4.51	2,500	2,420			0.18	165
2. 5.51	2,600	2,540	180	9	0.16	158
10. 5.51	2,470	2,400			0.14	158
17. 5.51	2,590	2,500	190	10	0.15	154
23. 5.51	2,470	2,380			0.15	154
30. 5.51	2,300	2,220			0.14	150

From these figures, it will be seen that, despite a well maintained replenishment rate, the dip wash concentration fell from the theoretical 0.20% Toxaphene to a constant of approximately 0.14%.

While on this subject it might be advisable to discuss the question of selective removal in the case of machine spray dips. Here again the insecticide is removed to a greater or lesser extent depending on the insecticide and formulation.

Dealing again with the same 40% Toxaphene wettable powder as discussed above, a spray dip sump freshly filled with spray wash at a concentration of 0.20% Toxaphene and plunged thoroughly throughout spraying operations gave a chemical test of 0.07% Toxaphene after 879 head of cattle had been sprayed. In the case of emulsions and miscible oils a similar behaviour was shown in that, in the case of Toxaphene, the figures in regard to one widely used preparation were 0.220% Toxaphene initially and 0.182% after 699 head had been sprayed, while in the case of BHC. emulsion the figures were 0.192% gamma and 0.120% gamma respectively after 630 head had been sprayed. On the other hand a good emulsion of DDT. showed no loss whatsoever.

Therefore it must be stressed that close attention must be paid to the recommended replenishment rates for the various proprietary formulations on the market as these have been based on the exhaustion rates encountered in the field, and by following such recommendations an efficient tick destroying concentration can be maintained.

Finally, in view of the fact that it is seldom possible to test the newer insecticides at the tank, it is essential that the dipping tank is calibrated so that accurate measurements of the volume of water or dip wash in the tank can be assessed and recorded before and after each dipping, to enable one to calculate how much water and therefore how much insecticide must be added. By following such a procedure it has been possible to maintain a satisfactory

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concentration of insecticide in dipping tanks over periods exceeding 2 years.

2. SPRAY-DIPPING.

With the advent of the new organic insecticides, we were faced with various problems that prompted workers to look for some means other than plunge dipping for the application of such insecticides.

Such problems included (1) Sedimentation of insecticide in the case of suspensions, (2) Breaking of emulsions and consequent inefficient control of ticks, or poisoning of stock, (3) Chemical changes of the active ingredient with loss of effectivity and (4) The physical and chemical effect of such substances as dirt, faeces, urine, sweat, etc., which tend to render the insecticide less effective. Again, apart from the use of certain proprietary testing outfits for proprietary formulations, it is not possible to test dip wash concentrations in the field, and it was felt that to mix a fresh wash of known concentration on the occasion of every treatment would be a decided advantage. Lastly, with the possibility of the blue tick developing a resistance to insecticides being ever present, the change-over to another insecticide would be far more convenient and less expensive in the case of a spray dip than in the case of a plunge dip. For the above reasons, it is little wonder that both veterinarians and stock owners are inclined to view the modern spray race as the practical answer to the use of the synthetic insecticides. However, despite the problems that spray dipping overcomes enumerated above, there are some disadvantages and some essentials associated with this method of application.

(a) *Disadvantages*:

(i) First and foremost, the spray dip must be powered by a mechanical unit and here one has something that can, at times, break down.

(ii) Contrary to general anticipation, the spray dip in the long run requires as much water as a dipping tank.

(iii) Although the wetting powers of a spray dip can be developed to a high degree of efficiency, complete immersion is effected by plunge dipping and therefore optimum wetting is obtained.

(iv) In areas where there is a constant interchange of cattle and the animals have become accustomed to plunge dipping a spray dip is a definite disadvantage.

However, taking it all in all, and provided the essentials mentioned below are provided for, spray dipping will have the advantage over plunge dipping when dealing with our newer insecticides.

(b) *The Essentials for a Good Spray Dip*:

(i) *Delivery Pressure*: In all spray dips studied, it has been found that the higher the delivery pressure, the better the wetting

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of the cattle, provided fogging does not occur. However, the delivery pressure should be inversely proportional to the rate of delivery and in the average spray dip the delivery pressure should be somewhere between 25 and 30 lbs. per square inch.

(ii) *Nozzle Pressure*: Where large-bore piping (3 ins. down to 1½ ins.) is used, the pressure at the nozzle furthest from the delivery will be only slightly less, if at all, than the delivery pressure. However, friction in the pipes and at the bends does tend to reduce the pressure at the nozzles but the larger the piping the more constant the pressure throughout. Therefore, to ensure that adequate size piping is used, it is recommended that the difference between the delivery pressure and that of any of the nozzles should not exceed 3 lbs. per square inch.

(iii) *Rate of Flow*: Although a large flow was obtained in an inefficient spray dip and *vice versa*, a good practical flow at 30 lb. per square inch delivery pressure should be in the vicinity of 12,000 gallons per hour. However, in view of the inconsistent results obtained in the various spray dips studied, it is recommended that the minimum required should be 10,000 gallons per hour at 30 lbs. per square inch delivery pressure. Again, it must be emphasized that the rate of flow is inversely proportional to the pressure. In other words, the greater the volume of wash passing through the nozzles, the less the pressure necessary to thoroughly wet the animals passing through.

(iv) *Number of Nozzles*: In the spray dips so far observed, the number of nozzles and other spray points has varied from 47 to 74, but all other factors being equal, the greater the number of nozzles the better the wetting. However, much depends on the type, capacity and situation of the nozzles. A number somewhere in the vicinity of 45 to 50 should meet the case.

(v) *Arrangement of the Nozzles*: These should be so arranged that all parts of the bodies of different sized cattle, including calves, will be thoroughly wetted during their unhindered passage through the spray dip. Special attention should be paid to the ears and top line of the neck.

(iv) *Type of Nozzle*: The essential feature of a good nozzle is that it should allow the free passage of fluid without hindrance in the form of coarse droplets. Fogging resulting from devices used to break up the jets is to be avoided.

(vii) *Length of Spray Dip*: Spray races of 36 ft., 22 ft. and 18 ft. long have been studied and observations have indicated that any length in excess of 18 ft. was superfluous. Unfortunately, it was not possible to study a shorter race but it has been claimed since that spray dips of 12 ft. and shorter have given satisfactory results.

(viii) *Filtration of Return Flow*: This is one of the most important aspects of spray dips. No matter how efficient the spray dip is in all other respects, it will rapidly be rendered inefficient as a result of nozzle clogging if the filtration system is faulty.

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Various types of filtration devices have been studied and it has been found that where the return flow is allowed to fall onto a smooth, perforated metal sheet slightly inclined upwards against the flow, the best results were obtained. The flow spreads fan-wise up the metal sheet and all dirt is washed to the edge of the perforated sheet where it accumulates and can be easily removed by hand from time to time during spraying. The wash, with its movement up the perforated sheet automatically cleans the centre of the fan and passes through the perforations.

A rough surface, such as wire gauze, traps dirt, grass, hair, etc., in its mesh and sooner or later becomes clogged, overflows and allows unfiltered dip wash to enter the spray system with resultant clogging of the nozzles.

The aforesaid perforated sheet must have a 6—9 inch protective edge around its perimeter to prevent any flow over the edge into the sump. The perforations should be of such a size as to allow the passage of fluid rapidly, yet solids too large to pass through the nozzles should be retained. 100 to 120 perforations per square inch gave excellent results.

(ix) *Rapid Return Flow*: A rapid return flow is essential for the sake of economy. When the sump becomes empty and the pump sucks air, spraying will cease. When such a stage has been reached, all wash in the spray dip, draining race and pipes must be regarded as "dead" wash. Therefore, the sooner the spray wash is returned to the sump the less "dead" wash there will be.

It has been observed that in spray dips constructed without a view to a quick return of the wash, 200 gallons and more have been found to be "dead," whereas in a well constructed dip the "dead" wash was brought down to 45 gallons, and it is felt that an even lower figure is possible.

Although it is difficult to suggest gradients because of the various types of dips studied, it is recommended that these should be such as to ensure as rapid a return of the wash as possible without constituting a danger to the cattle.

In all experiments undertaken, even in the case of efficient spray dips, there appeared to be insufficient wetting of the top line of the neck, under the tails and in the in the ears, despite efforts to remedy these inadequacies by dint of extra spray points, rearrangement of the nozzles, etc.

However, although in the better type of spray dips the tick control was not absolute, sustained spray dipping at weekly intervals with efficient concentrations of insecticides, gave sufficient control of ticks for us to regard spray dipping as a satisfactory substitute for plunge dipping.

3. Hand Spraying.

It has been found in the field, that many owners possess such small numbers of stock that it is neither economical nor practical for them to erect plunge dips or spray dips. Such owners are

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usually found on commonages and peri-urban areas and they have up to now depended upon commonage plunge or spray dips or adjoining farmers for the treatment of their cattle. Too often complaints have been received from such owners that the plunge and spray dips they are forced by circumstances to use are apparently insufficiently supervised with the result that the ticks on their cattle are not controlled. Therefore, there appears to be a good case for the treatment of such small herds by hand spraying.

Hand spraying diligently carried out, will give as good, if not better, results as plunge dipping or machine spraying, and therefore some mention of this method of treatment must be made.

In the experiments undertaken, the spray wash was mixed in a 20 gallon drum which could be plunged during spraying operations in the case of suspensions, and two good stirrup-type hand pumps were used. There are several types of good hand pumps with two nozzle leads on the market, which could be used with advantage. The nozzles should be of the trigger-type connected to the pump by high pressure hoses of sufficient length to enable the furthermost animal in the race to be sprayed without moving the pump or the drum of spray wash.

The cattle were run into the race 5 at a time, tied and sprayed from both sides starting with the tail and working up to the ears and face in the case of each individual animal. The worker on the one side of the race would commence with the animal at the front of the race while the worker on the other side would start at the back of the race. In this way the one worker would not interfere with the other operator overmuch.

In order to obtain thorough wetting 2 to $2\frac{1}{2}$ gallons of dip wash should be used per beast and after the workers have attained sufficient practice it will take 4 minutes per beast to do a thorough job. Thus for a herd of 25 head, approximately 60 gallons of dip wash would be required and with efficient workers and efficient spray pumps the work can be completed in approximately 2 hours.

It can be argued that the treatment could be effected with less work and in less time but, in the experiments undertaken, the object was to attain a wetting no less than that effected by plunge dipping. In fact the ears were held in one hand while they were thoroughly sprayed. Similarly the tail was held and manipulated while spraying the anal region and brush. The wetting was therefore very much better than that usually obtained in machine spray dipping and the practical results were correspondingly better. In view of the fact that none of the sprayed wash is saved, the question of selective removal of insecticide falls away.

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ORGANO-PHOSPHATE INSECTICIDES

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East London

Resistance to insecticides by the various arthropods inimical to man is of such an order to-day that when a new preparation is formulated and appears promising, the sceptics among us immediately look for the next one. The problem now is a double-edged one. Not only must we find an effective killer, but it must also be " resistant proof ". In the field of veterinary entomology in South Africa we have just begun to use the group of insecticides known generally as the Organic Phosphates.

These substances which are esters, amides or other derivatives of phosphoric and thio-phosphoric acids and are not to be confused with inorganic phosphorus and its compounds, were first of all developed by Schrader in Germany about 1942. Since 1946, they have been widely used throughout the rest of the world due to resistances developed against the chlorinated hydrocarbons. The group contains some of the most toxic substances yet used in agriculture. This toxic hazard must of necessity be accepted where it is essential to control resistant populations spreading disease. However, it is no less essential that by careful selection and sufficient experimental trial the toxic hazard be reduced to an absolute minimum, i.e. as far as possible made " farmer proof ".

For the control of ticks, particularly the resistant Blue Tick, and the sheep Blowfly, there are available in the Union in the group of Organic Phosphate Insecticides, four compounds; Asuntol, Diazinon, Malathion and Delnav. These have all been selected for their insecticidal activity and comparatively low toxicity for stock and at the concentrations used for the control of ectoparasites they are most unlikely to cause toxicity. The action of the Organic Phosphates is not limited to external application only, but, as in the case of Korlan and Warblefly control, can be fed to the animal and exert its effect systemically. A later and very interesting development is that some of the Organic Phosphates, such as Neguvon and Asuntol, have proved to be potent anthelmintics and are already in general use in Australia.

The toxic effects of the Organo-Phosphates all follow the same pattern (Radeleff *et al.*) of affecting the nervous system by depressing or inhibiting the enzyme cholinesterase and some also, such as Mipafox, produce demyelination of the peripheral nerves.

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Briefly the physiology of the reaction (Jolly) is as follows: Acetylcholine is formed at the end of many nerve fibres of the central nervous system, the autonomic nervous system and many motor nerves. The release of acetylcholine is caused by the nerve impulse whereby it is enabled to cross the synaptic junction. The acetylcholine is then immediately broken down to acetic acid and choline by the enzyme cholinesterase associated with the blood cells.

Cholinesterase (Barnes) also attempts to hydrolyze the Organo-Phosphate compounds just as it does the choline esters. However, when hydrolyzed, the phosphatic group remains attached to the enzyme thus preventing its action on acetylcholine.

The toxicity of the organo-phosphatic compounds is related to the affinity they have for cholinesterase and this affinity appears to be related to the alkyl groups attached to the phosphate; greatest toxicity being exhibited by an isopropyl group, less by an ethyl group and least by a methyl group. The result of this inhibition of cholinesterase is that acetylcholine accumulates at the synapses and the receptor cells of the cholinergic system are over-stimulated.

Symptoms of Poisoning

After absorption of a toxic dose of one of the organic phosphates, ruminants exhibit the following symptoms—"Muscarinic": increased flow of saliva, which becomes excessive and watery, lachrymation, dyspnoea with exaggerated respiratory movements, apparent colic with audible peristalsis and in some cases diarrhoea and head movements to the flank, the eye shows a drowsy appearance and is slightly glazed and the gait becomes stiff legged and aimless; then "nicotinic", such as muscular fasciculations and twitching of the eyelids appear and extreme weakness particularly in the hindquarters, causing the animal to go down on its side, turn its head over on the flank, then stagger up again. Eventually the animal becomes exhausted, is recumbent on its flank, shows weak paddling movements of the fore limbs and dies as a result of paralysis of the muscles of respiration or suffocation due to the oedema and fluid in the lungs.

Post Mortal Examination

This is usually negative except for a marked oedema of the lungs with fluid and foam in the trachea and bronchi. There may also be a slight gastro-enteritis.

Diagnosis

At present there is no satisfactory chemical test. In the living animal exhibiting symptoms, a blood test can be carried out for the presence or absence of cholinesterase. However, the test has certain limitations — blood must be used fresh or preserved in heparin and even heparinised and refrigerated blood, after 8 to 10 hours, gives variable results with the Lovibond comparator test. Other

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methods are essentially laboratory techniques. Diagnosis, therefore, rests mainly on clinical symptoms and anamnesis.

Differential Diagnosis

Heartwater must be ruled out as an animal in extremis from organic phosphate poisoning shows a large amount of froth and fluid exuding from the nose and mouth and paddles with the forelegs.

Treatment

The only form of treatment, until the recent development of the Oximes, was the administration of Atropine. However, unless it was given early on and maintained, results were not 100% effective. Since the introduction of the Oximes, 2 PAM (2 Pyridoxine-Aldoxime Methiodide) and DAM (Diacetyl monoxime) treatment with them alone or combined with Atropine has been very effective.

The action of Atropine is to reduce the severity of the muscarinic symptoms, but has no effect on the nicotinic symptoms of muscular weakness and paralysis. It has, however, been demonstrated (Grob) that the Oximes may reverse the cholinesterase inhibition and neuromuscular block, but have no effect on the muscarinic symptoms. In Australia the following treatment has been recommended (Gordon):—

CATTLE: Combination of Atropine and 2 PAM, Atropine 12.5 mg in 10 c.c. distilled water injected slowly intravenously followed by 37.5—50 mg injected subcutaneously, at the same time 50 mg/Kg of 2 PAM in 100—125 c.c. of physiological saline injected subcutaneously.

SHEEP: Two or three doses of 4—6 mg/Kg of Atropine. Gordon states that an initial dose of 2 mg/Kg, one-third intravenously and two-thirds subcutaneously has proved effective.

In the treatment of humans poisoned by Parathion in Japan, 2 PAM has been most effective (Namba and Hiraki). (These authors estimate that since the use of Parathion in agriculture in Japan over 6,000 cases of poisoning have occurred).

Experiment

CALF (1) 10—12 weeks old, dosed Neguvon/Asuntol @ 80 mg/Kg.
(2) 3— 4 weeks old, dosed Neguvon/Asuntol @ 70 mg/Kg.
(3) 3— 4 weeks old, dosed Delnav @ 100 mg/Kg.
(4) 4— 6 weeks old, undosed control.

Within an hour all three dosed animals were showing symptoms, lachrymation, salivation, weakness, stumbling gait, twitching eyelids, muscular trembling. Calf (2) was very staggery and continually collapsed and struggled up then collapsed and lay on its

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flank. All three showed dyspnoea and exaggerated respiratory movements. 1½ hours after dosing all three were treated:—

CALF (1) 2 PAM 30 mg/Kg in 1% solution intravenous;
(2) 2 PAM 15 mg/Kg in 1% solution intravenous;
(3) Atropine Sulphate 30 mg subcutaneously.

Calves 1 and 2 showed an almost immediate response and within five to ten minutes were grazing. Calf 3: The symptoms only started abating after half to three-quarter hours. Six hours later it appeared normal, but was thirsty and showed diarrhoea, eighteen hours after dosing it was recumbent showing colic, grunting, marked salivation and paddling of the forelegs and expired shortly after being seen.

BLOOD CHOLINESTERASE TEST — LOVIBOND COMPARATOR METHOD

	Before Dosing		1 Hour after Dosing			18 Hours after Dosing		
Time:	40″	60″	40″	60″	80″	60″	80″	100″
Calf (1)	75	100	37.5	62.5	100	25	25	25
Calf (2)	100	—	75	100	—	12.5	12.5	12.5
Calf (3)	100	—	50	75	100	0	0	0
Calf (4)	100	—	87.5	100	—	87.5	100	—

The above experiment exemplifies results in the treatment of induced poisoning in some very young calves and some cases in cattle and goats which had ingested dipping fluid. A drawback in the use of 2 PAM is its expense.

The most effective treatment, therefore, would seem to be a combined therapy with Atropine and 2 PAM at a minimal dosage which so far appears to be at 15—20 mg/Kg.

Atropine should be given at double the therapeutic dose in cases of acute poisoning and should be repeated as necessary, intravenously for immediate effect as subcutaneously it takes ± half an hour to act.

Animals that have been sprayed require washing, those that have ingested material should be given purgatives and carnivores given emetics. The patient should be kept as quiet as possible and prevented from moving about.

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THE DEVELOPMENT AND MECHANISM OF INSECTICIDE RESISTANCE IN THE BLUE TICK, *BOOPHILUS DECOLORATUS* KOCH

G. B. WHITEHEAD

The blue tick, *Boophilus decoloratus* Koch, is indigenous to Africa where it is an ectoparasite on a variety of mammalian species. It has become particularly well adapted to domestic bovines and in some areas of South Africa it can be found in large numbers on cattle. Apart from the general debilitating effect large populations of the blue tick can cause, the blue tick is of economic importance because of the pathogenic organisms of which it is a vector. The diseases transmitted by the blue tick are anaplasmosis and piroplasmosis of cattle, spirochaetosis of cattle, horses and other animals and tick bite fever, a rickettsial disease of man. Failure to control the blue tick can result in enormous losses in livestock and for this reason control measures are systematically and conscientiously applied.

Under laboratory conditions blue tick larvae are particularly sensitive to relative humidity and a hundred per cent hatch of eggs is only obtained at a relative humidity greater than 75%. Larvae readily succumb to desiccation but will survive for a considerable time at high humidities. The effect of atmospheric humidity on the distribution of the blue tick has been clearly indicated by Theiler (1949).

The tick is present along the south and east coasts of the country and is limited inland to areas where the average annual rainfall is above 15 inches. Blue tick populations are heaviest in the east where the annual rainfall is highest and temperatures most equitable.

Measures for the control of the blue tick have been in practice since the turn of the century. The practice of dipping cattle in dipping tanks containing sodium arsenite was eventually adopted throughout most of the tick-infested areas of the country. It was established that sodium arsenite at a concentration of 0.16% As_2O_3 was effective in controlling most species of tick at a dipping interval of seven days. This practice was particularly effective against the blue tick but because of a continuous source of reinfestation from indigenous fauna strict control measures had to be maintained.

Sodium arsenite preparations continued to be effective for the control of the blue tick until 1939. Following on numerous com-

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plaints from farmers in the East London district investigations revealed that the blue tick had acquired a resistance to sodium arsenite (du Toit and others, 1941). Almost simultaneously the presence of arsenic resistance in blue ticks was demonstrated in the Bathurst district (Whitnall and Bradford, 1947). With the disclosure of the fact that the failure to control the blue tick was a result of a resistance to sodium arsenite it became apparent that the blue tick population along the coast from Port Elizabeth in the south through Natal in the north was resistant to sodium arsenite.

The observation of arsenic resistance in the blue tick along the entire eastern coastal belt in a very short time after the first disclosure of resistance in the East London and Bathurst areas does not of necessity mean that arsenic resistance was built up rapidly in a few years after a long period of successful control. It may have arisen gradually and been undetected over many years in this area.

It was shown (Whitnall and Bradford, 1949) that gamma BHC was exceedingly effective against the arsenic resistant blue tick and this material soon became widely used in areas where the arsenic resistant tick had become established. The first indications of resistance to BHC in the blue tick were observed only eighteen months after the field introduction of BHC (Whitnall and others, 1952). The BHC resistant tick eventually covered the same geographical area as was originally covered by the arsenic resistant strain. Ticks that were resistant to BHC also retained a resistance to sodium arsenite.

DDT was then introduced for the control of the arsenic-BHC resistant strain and although it appeared to exert insecticidal action mainly on larvae, it achieved an effective field control. DDT was used for more than five years before DDT resistance first developed (Whitehead, 1956).

At the present time several organo-phosphorus preparations have been successfully introduced for the control of the arsenic-BHC-DDT resistant tick. To date no signs of resistance to the organo-phosphorus group have been observed.

The availability of the various strains of insecticide resistant blue ticks afforded a good opportunity for a complete laboratory evaluation of insecticide resistance and the interdependence between the various types of resistance.

Using both the adult fully engorged female tick and unfed larvae in laboratory tests the resistance to a variety of insecticides was investigated (Whitehead, 1958, 1959). The fact that arsenic resistant ticks were originally controlled with BHC, suggests that there is no relationship between arsenic resistance and resistance to BHC. Although the ticks resistant to arsenic and BHC eventually became resistant to DDT it is obvious that resistance to arsenic and BHC bore no relationship to DDT resistance. In Table 1 the effect of a number of insecticides on blue tick larvae of different strains are given as measured by the concentration of insecticide

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required to produce 50% mortality (LC 50) in 24 hours. Results of insecticide tests on adult female ticks are presented in Table 2. Tests for response to insecticides using adult ticks, although in many

Insecticide	Strain of blue tick larvae		LC 50 (% concentration of insecticide)	Increased tolerance compared with the most sensitive strain
Gamma BHC	Ferndale	1956	0.2395	8 x 10^2
	Frankenwald	1956	0.000298	1
Toxaphene	Ferndale	1956	0.829	9 x 10^2
	Frankenwald	1956	0.000870	1
Chlordane	Ferndale	1956	30156*	7 x 10^6
	Frankenwald	1956	0.00426	1
Dieldrin	Ferndale	1956	26971*	2 x 10^7
	Frankenwald	1956	0.00123	1
Aldrin	Ferndale	1956	71031*	3 x 10^7
	Frankenwald	1956	0.00179	1
pp'DDT	Ferndale	1956	0.00345	1
	Frankenwald	1956	0.00491	1.43
	Transvaal	1958	0 02154	6.25
	Frankenwald	1958	0.01045	3.03
Malathion	Ferndale	1956	0.08199	1
	Frankenwald	1956	0.1034	1.26
Diazinon	Ferndale	1956	0.000756	1.02
	Frankenwald	1956	0.000742	1
Delnav	Frankenwald	1958	0.000291	1.21
	Ferndale	1958	0.000232	1
	Nagaza Park	1958	0 000315	1.35
	Transvaal	1958	0.000270	1.16
Asuntol	Frankenwald	1958	0.000346	1.38
	Ferndale	1958	0.000531	2.12
	Nagaza Park	1958	0.000713	2.85
	Transvaal	1958	0.000254	1
Korlan	Frankenwald	1958	0.001880	2.87
	Ferndale	1958	0.000654	1
	Nagaza Park	1958	0 002926	4.5
	Transvaal	1958	0.003421	5.25
Sevin	Frankenwald	1958	0.00404	1
	Ferndale	1958	0.00566	1.4
	Nagaza Park	1958	0.00487	1.2
	Transvaal	1958	0.00586	1.45
Pyrethrum	Frankenwald	1958	0.00000946	2.41
	Ferndale	1958	0.00000962	2.45
	Nagaza Park	1958	0 00007240	18.3
	Transvaal	1958	0.00000395	1

*By extrapolation

TABLE 1.—The effect of a number of insecticides on several strains of blue tick larvae.

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respects more convenient to manipulate, are considerably less accurate and can only be used to show gross changes in insecticidal response. Because it is difficult to assess whether a treated tick is alive or dead and because of the fact that some insecticides are known to afford control of the blue tick by affecting the viability of eggs produced by the treated female, results of tests with the adult female have been assessed on the percentage hatch of eggs produced. The results of the effect of various insecticides on the viability of eggs produced by treated female ticks are presented in Table 2.

Insecticide	Percent Concentration	Per cent. of adult female ticks which produced viable eggs after treatment of the following strains of adult female blue ticks.					
		Frankenwald 1956	Ferndale 1956	Allandale 1956	Frankenwald 1958	Nagaza Park 1958	Transvaal 1958
Sodium arsenite	0.32 As_2O_3	0	56	32	3	20	0
Gamma BHC	0.01	8	—	—	93	33	—
	0.125	0	92	98	—	—	—
Toxaphene	0.125	36	97.5	98	90	—	—
Chlordane	0.25	4	100	96	79	—	—
Dieldrin	0.125	0	—	98	—	—	—
	0.25	—	94	82	75	—	—
Aldrin	0.25	8	98	88	74	—	—
pp'DDT	0.5	0	6	98	—	—	—
Dilan	0.5	8.5	24	90	—	—	—
Malathion	0.125	88	93	94	100	—	—
	1.0	12	8	10	15	—	—
Diazinon	0.031	0	2	0	15	—	—
Delnav	0.025	—	—	—	10	14	10
Asuntol	0.025	—	—	—	10	13	—
	0.05	—	—	—	5	—	7
Korlan	1.6	—	—	—	40	60	25
Sevin	0.05	—	—	—	73	60	72
	0.1	—	—	—	40	57	50
Pyrethrum	0.00625	—	—	—	52	90	62

TABLE 2.—The effect of a number of insecticides on several strains of adult female blue ticks.

Pattern of insecticide resistance in the blue tick

Metcalf (1955) in a study of insecticide resistance in the housefly, *Musca domestica*, came to the conclusion that insecticides could be grouped in accordance with resistance developed to them. The development of resistance to one insecticide in a group resulted in an automatic resistance to all insecticides in the same group, even though these materials had never been used in the field. An

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examination of the results in Tables 1 and 2 suggests a pattern of resistance in the blue tick very similar to that in houseflies.

Resistance to sodium arsenite displays no cross resistance to any other insecticide investigated. BHC resistance developed subsequently in the arsenic resistant strain and automatically shows a cross resistance to all the chlorinated cyclic hydrocarbon insecticides such as toxaphene, chlordane, dieldrin and aldrin. BHC resistance does not confer a cross resistance to DDT which, although chlorinated, is basically a straight chain aliphatic compound.

It is of interest to note that over the period during which this work was carried out, the Frankenwald tick initially sensitive to all insecticides, eventually developed a resistance to the BHC group of insecticides. This came about after two years of the field use of toxaphene at Frankenwald. The Frankenwald 1958 ticks were thus highly resistant to BHC, toxaphene, chlordane, dieldrin and aldrin, all of which arose as a result of field treatment with toxaphene, but to no other insecticide (Tables 1 and 2). Resistance to the BHC group in the East London district arose from the field treatment with BHC.

DDT resistance developed in ticks that were resistant to arsenic and BHC (Whitehead, 1956). However, many ticks resistant to arsenic and BHC which are not resistant to DDT still exist (e.g. the Ferndale, 1956, strain). The strain resistant to BHC only (Frankenwald, 1958) were also not resistant to DDT. DDT resistance (Allandale, 1956, strain) were cross tolerant to Dilan which is a mixture of nitroparaffin analogues of DDT.

This phenomenon does not occur in the pattern of cross resistance in flies and suggests a mechanism of resistance in the tick which may be different from that in houseflies. Resistance to DDT surprisingly conferred a substantial cross resistance to pyrethrum which was confirmed by extensive tests on both adult and larval ticks (Whitehead, in press).

No resistance to any of the organo-phosphorus insecticides was detected and all strains examined reacted similarly to malathion, diazinon, Asuntol, Korlan and Delnav. The carbamate insecticide, Sevin, was also equally effective against all the strains.

From these results it is possible to group the series of insecticides in accordance with resistance displayed to them. Resistance to any one insecticide in a group will result in an automatic resistance to all insecticides in the same group. Although resistance to several groups can develop in time, resistance to each group is independent.

In the blue tick the grouping of insecticides according to the tick resistance or sensitivity is as follows:—

1. Sodium arsenite.
2. The chlorinated cyclic hydrocarbon group which includes BHC, toxaphene, chlordane, dieldrin and aldrin.
3. The DDT group which includes dilan and pyrethrum.
4. The organo-phosphorus group which includes malathion, diazinon, Asuntol, Korlan and Delnav.

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5. The carbamates of which only Sevin has been examined.

The separation of the organo-phosphorus group from the carbamate group in ticks is at present tentative as no resistance to these two groups of insecticide has yet appeared. However, in houseflies, organo-phosphorus resistance does not confer a cross resistance to the carbamates (Metcalf, 1955).

The development of resistant populations of the blue tick

Busvine and Harrison (1953) suggested that the development of resistance in insect populations depended on three factors, namely:

(i) The frequency of occurrence and effectiveness of resistant genes in natural populations of insects.
(ii) The intensity of selection, i.e. the magnitude of the population exposed to the insecticide and the proportion killed.
(iii) The number of generations per year.

It appears that these factors also contribute to the development of resistance in the blue tick populations. The response to an insecticide by individual ticks varies greatly. The distribution of the response of ticks to an insecticide within a population approaches normality if the applied doses are increased logarithmically. This fact is illustrated in Diagram 1.

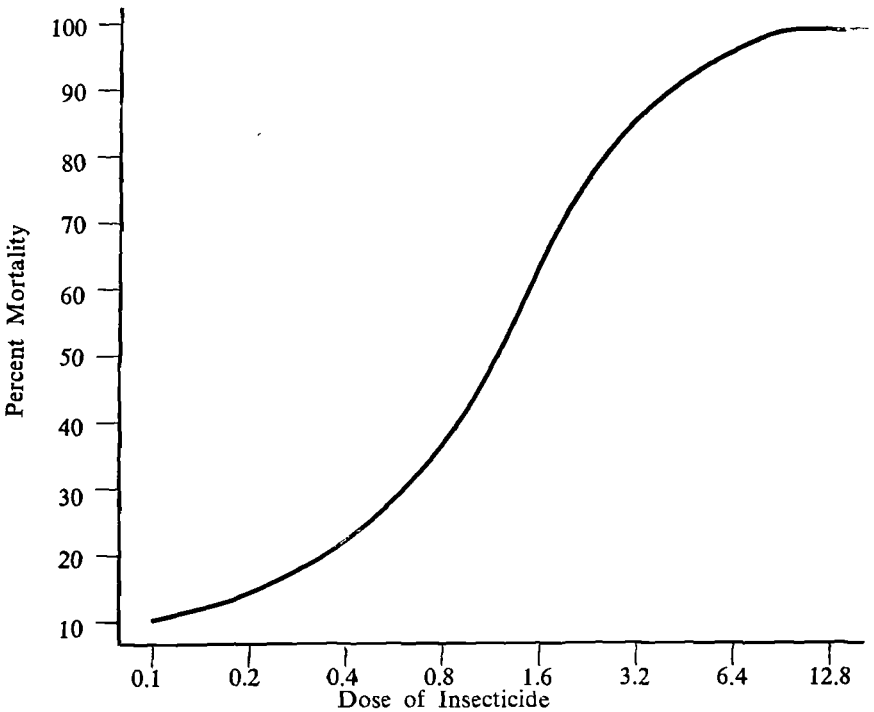


DIAGRAM 1.—The distribution of insecticide response in a population of ticks.

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It will be observed from the diagram that if a tick population were treated with a theoretical insecticide dose of 6.4% a large proportion of the population would be killed but a small proportion would survive. By doubling the dose the surviving proportion would be reduced even further but still a few would survive. Theoretically it is thus not possible to kill all individuals of a population despite the use of a high concentration of insecticide. The survivors which are the most resistant to the particular insecticide, would give rise to progeny many of which would also be capable of surviving insecticide treatment at that concentration. The more frequently the population was treated the more vigorous would be the selection of the resistant individuals. Eventually a population of ticks would arise which would be unaffected by low concentrations of the insecticide. The new population would show individuals differing in response to the insecticide and would give a similar type of distribution yet be much more tolerant to the insecticide.

It becomes obvious that the more frequently a tick population is treated and the more quickly it breeds and the more thoroughly the whole population is exposed, the more rapidly a resistant population will develop.

The blue tick being a one host tick reproduces fairly rapidly. Its rate of reproduction is greatest in areas of high humidity and temperature. Where the tick is more abundant the more necessary treatment becomes. Furthermore, the intensity of selection is very great in respect of the blue tick as in a programme of 7 day dipping each tick can be treated three times, at larval, nymphal and adult stage. It is because of these characteristics and the dipping practice that resistance to a number of insecticides became apparent. It is likely, for the same reasons, that resistance will develop to other insecticides after varying periods of time.

The rapidity with which a resistant population will develop is difficult to predict, because of the many unknown factors. By some very interesting recent work on a similar aspect of insecticide resistance in several species of mosquitoes, Davidson (1958) explained the comparatively slow development of DDT resistance, compared with resistance to BHC and dieldrin on a genetical basis. Breeding experiments with these species of mosquitoes showed that the inherited characteristic of BHC and dieldrin resistance was semi-dominant. In experimental cross-breeding of dieldrin-BHC resistant and sensitive *Anopheles gambiae* and recrossing of the progeny, a population was developed containing sensitive, hybrid and resistant individuals in a 2:4:2 ratio. The resistance of the hybrids was intermediate between the resistant and the sensitive strain but the hybrids were still sufficiently resistant to survive field application of dieldrin or BHC. Breeding experiments with *Anopheles sundaicus* known to have developed resistance to DDT relatively slowly showed that the inherited characteristic was recessive. Cross breeding the sensitive strain with a DDT resistant strain and breeding from the progeny resulted finally in a population

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containing only resistant and sensitive individuals in a 2:6 ratio. It is obvious from this information that interbreeding of DDT resistant and sensitive individuals in a mosquito population will result in the development of a resistant population far more slowly than the cross breeding of dieldrin-BHC resistant and sensitive individuals.

Having established the inherent nature of resistance in mosquitoes, Davidson concluded that the higher the selection pressure the sooner any resistance in a population would appear. In practice selection pressure would be highest with the most efficient insecticide or at higher dosages or as a result of a combined attack on all stages of the insect.

Cross breeding experiments with different strains of the blue tick would be very difficult to execute but the similarity in the difference in time taken to develop resistance to BHC and DDT in the blue tick and in mosquitoes suggests that the mechanism could be similar, i.e. the hereditary characteristic of DDT resistance is recessive and that of resistance to the chlorinated cyclic hydrocarbon group is semi-dominant. By the same assumption it may be concluded that the inheritance of arsenic resistance is also recessive. This type of investigation of the mode of inheritance of the resistant characteristic might allow a prediction to be made as to the length of time a newly introduced insecticide for blue tick control would remain effective.

Distribution of resistant strains

Resistance to sodium arsenite, BHC and DDT in the blue tick was in each case first reported from the East London district. At the present time arsenic and BHC resistance exists in ticks from a narrow belt along the coast from Mossel Bay to Natal. DDT resistance in the blue tick is still localised in the East London area but is now making its appearance up and down the coast. Inland the blue tick is sensitive to all insecticides except in a few areas around Pretoria and Johannesburg where BHC resistance has developed (Bekker, 1953), (Whitehead, in Press).

The development of resistant strains along the eastern coast suggests that climatic conditions have contributed to this development. The climatic conditions along the coast are relatively humid and equitable with frost-free winters which are conditions which favour the development of large populations of the blue tick. In consequence control measures are more conscientiously applied on farms in the coastal belt than inland where the cold dry winter conditions exert a considerable controlling influence on the development of blue tick populations. Thus the high reproductive potential of the blue tick along the coastal belt and consequent frequent treatment could have led to the more rapid selection of the resistant individuals. There is no reason to assume that resistance to any insecticide would not develop in ticks from inland but a longer time lapse for resistance to develop would be expected because of the

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reduced rate of survival and consequent reduced frequency of insecticidal treatment. Resistance to any insecticide may be expected to develop eventually in tick populations from inland areas as has been the case in the Pretoria district and at Frankenwald.

Mechanism of Insecticide Resistance
Sodium Arsenite

The mode of toxic action of arsenic has not been completely resolved. There is, however, some evidence that the action of arsenic is associated with the thiol group (-SH). Voegtlin and others (1923) demonstrated that the trypanocidal activity of arsenicals could be reversed by the addition of amorphous glutathione, cysteine and other related -SH containing compounds. An association between arsenic toxicity in insects and sulphydryl compounds was suggested by Fink (1927) who found that glutathione content of ten different species of insects decreased following the injection of both arsenic trioxide and arsenic pentoxide.

Lyman and Barrow (1937) indicated that the rôle of glutathione was that of a regulator of the rate of reversible oxidation-reduction reactions. Barrow and Singer (1943) pursued the idea of the regulatory function of glutathione further when they found that seventeen out of thirty-two enzymes examined were inactivated when treated with -SH deactivating agents and furthermore were reactivated by the addition of reduced glutathione. This suggested that glutathione was essential to a number of enzyme systems. It thus becomes apparent that the immobilisation of glutathione by arsenicals could disrupt the oxidation-reduction and many other enzyme systems depending on the -SH grouping and thereby bring about a serious state of intoxication.

Assuming arsenic toxicity is associated with -SH containing compounds it is conceivable that resistance to arsenicals might be a result of an increased level of the particular -SH containing compound or compounds. Forgash (1957) showed that the difference in response to arsenite in the sexes of *Periplaneta americana* was related to the amount of glutathione in various tissues. The greatest reduction of glutathione occurred in the fat body, and the female roach which was more tolerant to arsenic than the male, contained approximately eight times more fat than the male.

With the availability of arsenic resistant and sensitive strains of the blue tick it was possible to investigate the level of several -SH containing compounds on tick eggs and larvae.

An examination of tick eggs midway between oviposition and eclosion showed a sulphydryl level in eggs from an arsenic resistant strain twice as great as that in an arsenic sensitive strain (Thompson and Johnston, 1957). Further work on this aspect of the mechanism of arsenic resistance revealed that both cystine and cysteine occurred in greater quantity in unfed arsenic resistant larvae than in arsenic sensitive larvae of approximately the same age. Likewise determinations of glutathione and free sulphydryl in larvae

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showed significantly greater quantities of both compounds in the arsenic resistant strain as compared with the arsenic sensitive strain (Harington, personal communication).

It appears from these findings that increased levels of -SH containing compounds can be associated with arsenic resistance in the blue tick.

DDT

As with arsenicals, little is known of the mode of toxic action of DDT. The mechanism of resistance to DDT in houseflies has, however, been fairly well investigated. It was shown (Perry and Hoskins, 1950), (Sternberg, Kearns and Bruce, 1950) that DDT was broken down in flies to the non-toxic 1, 1-dichloro-2, 2-bis (p-chlorophenyl) ethylene (DDE) and that the breakdown was far greater in the resistant strain of flies than in the sensitive strain. It has been shown that the breakdown of DDT to DDE was as a result of an enzymatic reaction. The dehydrochlorinating enzyme, called DDT-dehydrochlorinase was successfully isolated from DDT resistant flies but could not be detected in the sensitive strain (Sternburg and others, 1954).

The DDT resistant blue tick is also automatically resistant to Dilan, which is similar to DDT but has no chlorine atoms on the alkyl chain and therefore cannot be dehydrochlorinated. In DDT resistant houseflies Dilan is effective against the DDT resistant strains (Metcalf, 1955). This and the fact that there is a cross tolerance to pyrethrum conferred by DDT-resistance in ticks, suggests that the dehydrochlorination of DDT in DDT-resistant ticks may not be the mechanism of resistance. However, work still being undertaken shows no formation of DDE after 24 hours of exposure of resistant or sensitive larvae to deposits of DDT. After 24 hours all of the DDT sensitive strain were dead whereas all the DDT resistant strain remained alive. Where the two strains had been exposed to DDT for five days there was a slight conversion of DDT to DDE in the DDT resistant strain but no conversion in the sensitive strain which were all dead within 24 hours of treatment. This fact suggests that DDT could immediately form a transient complex with an enzyme in the tick tissue which would immobilise it immediately and render it innoxious and that the conversion to DDE according to the Michaelis-Menton equation for enzyme reactions takes place at a much slower rate corresponding to an ordinary elimination process. Aspects of this work are still being investigated.

gamma BHC

Little is known of either the mode of action of or the mechanism of resistance to gamma BHC in insects. Work on houseflies has established that BHC is broken down to a number of unidentified products. The rate of breakdown appears to be greater in the resistant strain (Bradbury and Standen, 1953). Sternburg and Kearns (1956) maintained that a conversion of BHC to penta-

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chlorocyclohexene was the first stage in the breakdown of BHC in flies and found that the rate of formation of this compound was greatest in the BHC resistant strain. Our preliminary attempts to detect pentachlorocyclohexene in BHC treated tick larvae have so far been unsuccessful but the possibility of the existence of the same breakdown mechanism in ticks as occurs in houseflies cannot be discounted.

It will be noted that the most logical explanation of the mechanism of resistance to the three insecticides is entirely different. In the case of arsenicals, resistance to arsenic has resulted in a selection of a strain with a higher level of -SH compounds. With BHC and DDT there appears to be a breakdown of the insecticide within the tissues to a non-toxic product. The enzyme system responsible for the breakdown of DDT to DDE has been shown to be ineffective against BHC. This is a possible explanation for the reason that there is no cross resistance between resistance to sodium arsenite, DDT or BHC.

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DISCUSSION

DR. DU TOIT (opener) thanked the speakers and pointed out that in many species of ticks the build up of the ensuing summer's population largely depended on the immature forms which flourished during the late autumn and early winter months. With the advent of cold weather and the disappearance of the easily noticed adult ticks, most farmers tended to relax control measures, thus allowing of a large stage build up of immature forms.

The question of resistance to tick infestation was also a matter that required study. He recalled an occasion where 20 head of cattle were moved from an area with few ticks to a heavily infested area. Within a few hours the new arrivals were covered with ticks whereas the local animals only had the odd tick.

MR. GRAY gave a very interesting account of work being done at Mazabuka which will be quoted at length, as follows:

"I would like to quote a statement made in Dr. Theiler's most enlightening paper: 'In South Africa we know but little as yet of the exact conditions under which our ticks live.' We have taken this as the focal point of our observations.

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Briefly, these have been directed to the engorged female, eggs and unfed larvae of *R. evertsi* in three distinct habitats:

(1) A short-grass area, covered, in the main, by *Sporobolus marginatus* (about 6 inches high).
(2) A medium height grass area, consisting of an almost pure stand of *Chloris gayana* (3 to 4 feet).
(3) A tall-grass area of *Hyparrhenia* — Spp. (6 to 8 feet).

These areas are quite separate, but adjacent and occur on a black or dark grey clay soil, rich in alkaline earths, akin to the 'sub-tropical black clay soils of the Transvaal' of van der Merwe. They are literally adjacent so that the macroclimate is the same in each case.

In the early stages of the observations, engorged females, collected from cattle, were marked with paint, dropped at random in these and other habitats, allowed 'free range' and observed *in situ* over long periods. In due course a fairly constant pattern emerged. When dropped on to grass, the female seeks to move downwards through the vegetation to ground level. If the cover is sufficient, there may be very little lateral movement and the female may find a convenient site to oviposit only a few cm. distant from its arrival point on the ground. Where cover is sparse, the lateral movement may be considerable; up to a distance of 11.9 metres has been observed.

Ovipositing sites depend on the nature of the ground and its cover. On areas where growing vegetation is sparse, but on which there is accumulated debris or litter, females oviposited beneath the litter but generally at ground level. The depth of litter overlying any female varied from time to time, depending on wind and rain — a depth of 3 cm. was not infrequently noted. On bare ground, with loose soil, ovipositing females have been found to a depth of about 2 cm. below ground level. On bare ground, with the soil 'baked' hard, females have been observed to die before finding a suitable ovipositing site. Some such deaths have occurred within eight hours, due, it is thought, to heat exhaustion (desiccation). On tufted grasses, females were found ovipositing either deep into the crown or at the base of the plant, from 1 to 2 cm. below ground level.

Once a fairly clear picture of this pattern emerged, females, collected from cattle were no longer marked and 'dropped,' but, instead, put into small, white cotton mosquito netting bags and placed in sites which looked suitable at and below ground level to a depth of 1 to 2 cm., in the short-grass area, the medium-grass area and in the tall-grass habitats.

No precise observations on pre-ovipositing or incubation periods have been made, because it was found impossible to do so without disturbing the female. Instead, the period between fall of female to eclosion has been noted. The shortest period observed was more than 19, but less than 23 days — this occurred during December and January. The longest period, more than 78, but less than

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83 days, was during April/July. Both these occurred in the *Hyparrhenia*.

Larvae remain at or close to the site of eclosion usually in tight cluster formation for some time before ascending the vegetation. The shortest time observed occurred in the short-grass area and was about 4 days; the longest, more than 16, but less than 20 days, occurred in the tall-grass habitat. Ascending the grass, the larvae generally settle in small to large clusters at or near the tips of leaves and stems and to leeward on the underside of a bent blade of grass at or near the highest point. With age and/or rising temperatures or falling humidities, the larvae tend to move away from the vegetation tips to such sites as leaf axil, between leaf and stem, within a leaf curl or into the hollow tip of a stem.

R. evertsi larvae have remained on the upper vegetation in the tall-grass area for periods of up to 149 days during March to July.

With a view to obtaining some idea of the temperature and humidity close to the ground within the three types of habitat, measurements are made daily, between 08.15 and 08.30 hours. Relative humidity is taken at 1-2 cm. above ground by means of an aspirated hygrometer. Maximum temperatures are taken at ground level and at 1-2 cm. above ground level and, in the case of the short-grass area, at 1-2 cm. below ground level as well.

The general pattern emerging so far shows the short-grass area to be least favourable and the tall-grass area most favourable to the engorged female, ovipositing and eclosion rates.

Observations based solely on visual estimates suggest that females placed at about 2 cm. below ground in the short- and medium-grass area, oviposit a greater number of eggs and the eclosion is greater than in those placed at ground level in these two habitats.

In the short-grass habitat, the temperature generally is higher and the humidity is generally considerably lower than in either of the other two habitats. That may be a factor causing the conditions to be less favourable than in the other two habitats."

DR. STAMPA stated that toxic effects caused by some organic phosphate insecticides developed very rapidly and in such cases treatment with atropine gave good results. With other compounds toxic symptoms developed more slowly and in these instances PAM was the antidote of choice.

DR THOMAS asked about the possibility of a cumulative action especially when organic phosphates were used both as dips and as anthelmintics.

DR. THOROLD replied that after a single exposure absorption could continue for up to two weeks. The cholinesterase level in the blood frequently did not return to normal before 3 to 4 weeks. It therefore appeared that a period of 3 weeks should be allowed between treatments.

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INTRA-ARTICULAR AND INTRA-THECAL PREDNISOLONE IN THE TREATMENT OF TRAUMATIC INFLAMMATION OF SYNOVIAL STRUCTURES IN EQUINES

JOHN QUINLAN

It is intended that this paper should be confined to practical experience on the use of prednisolone (Δ^1-dehydrocortisone) in the treatment of traumatic injury to joints, tendon-sheaths and bursae. I will deviate from this aim only to give a cursory review of the relevant literature available in veterinary journals. However, the advancement of our knowledge on the physiological necessity of a healthy adrenal gland, and its hormones, as indicated by Addison (1855), and its important role in the endocrine system, deserves close study by the veterinarian.

The inter-relationship between the pituitary gland and the sex-glands with the suprarenal concerns the maintenance of life, the processes of reproduction, metabolism and growth. In addition to this inter-relationship it has been demonstrated that cortisone has independent individual functions; with one of these we are concerned, namely its anti-inflammatory action; its anti-allergic action is outside the reference in this paper. Further, I will confine my discussion almost entirely to its action on synovial inflammation.

When injected into a synovial structure, undergoing the phenomenon of inflammation, it decreases synovial fluid, the cellular reaction, capillary growth, fibrin deposits, oedema and evidence of fibrosis. (Hench, et al. 1949). In fact it entirely transforms the local syndrome associated with these inflammatory conditions.

Cortico-steroid therapy is well established in medical science. A vast literature is now available on its value both in systemic and local conditions. In fact, in view of its value in human medicine, it is astonishing that almost 10 years have elapsed since Thorn (1949) and Hench (1949) demonstrated its effectiveness in humans by intra-articular injection, before it was introduced extensively in veterinary practice. Probably the cost, even until recent months, has been responsible for our tardiness in keeping parallel with our medical confréres.

I have used cortico-steroids in several equine diseases in which there was cause for anxiety. My experience is that they rarely fail to produce improvement in general well-being: malaise and anorexia are relieved. The clinical picture is rapidly changed.

It is suggested that the cortico-steroids by their anti-inflammatory action remove the barrier to the spread of micro-organisms from an acute, septic, local inflammatory lesion. To overcome

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this disadvantage, if, in fact, it does exist, they can be combined with antibiotics. I have not found that they have anything but a beneficial effect on the treatment of septic synovitis where stress and pain are important factors. I use them in company with antibiotics.

The work of Kinsell and Jahn, 1955, has demonstrated the value of combined therapy in patients with acute infections.

The cytology of destruction and subsequent repair, following the use of Cortico-steroids, leaves no doubt that the local improvement in inflammatory lesions of joints, tendon-sheaths and bursae outweighs suggested disadvantages (Sprague, 1951, Calkins and Bauer, 1955).

The most common diseases of racehorses are traumatic injury to the joints, tendons, tendon-sheaths, ligaments and bursae of the fore limb. The shoulder and elbow joints are rarely involved: The knee, fetlock, flexor tendons, check ligament, sesamoidean tendons, the carpal sheath, digital sheath, bursa podotrochlearis, and the sheaths of the extensor tendons are most frequently affected.

The joints, tendons, ligaments, sheaths and bursae of the hind limb are less frequently affected by traumata in the thoroughbred. However, gonitis, coxitis and tarsitis are not uncommon; neither are strains of the ligamentum tarsi plantare. The sheaths and bursae most frequently affected are those of the hock and the digital sheath.

The cause of the frequency of disease of the limbs in thoroughbreds is usually faulty conformation, incorrectly balanced rations, mineral imbalance and racing two-year-olds on going which is usually firm.

Prior to 1957 I had treated these various pathological conditions of the limbs resulting from trauma by the recognised methods of therapy, which in each case depended on the severity of the lesion. These methods included rest and bandaging, cold treatment, cooling pastes and lotions, followed by warm treatment, Preissnitz bandages, antiphlogistine packs, liniments, Bier's hyperaemia, blistering and firing: I have also used inducto-therapy and ultra-short wave therapy. The use of the Faradic current is more recent in equine practice. It is of great value in preventing adhesions and muscular atrophy. It has a very useful rôle in equine practice.

Firing of horses should be avoided if at all possible. The operation rather flavours of ancient than modern surgery, in which the patients' comfort is a *sine qua non*. It is looked upon as crude by our medical confréres and cruel by many knowledgeable owners and trainers of thoroughbred horses, even though the operation is done under local or general anaesthesia. There is little doubt that it reduces speed by the formation of fibrous tissue around joints and tendons, thereby somewhat limiting normal physiological movement. Besides it is painful to horses for a few days following the operation. However, a long-acting local anaesthetic relieves this

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pain. There is always stiffness and pain on movement for several days after deep, needle-point firing which is usually desirable to reach the pathological area. Horses are greatly worried by flies in this country unless a fly-proof stall can be provided. The irritation may cause them to gnaw the fired area unless they are properly controlled. In fact, firing of horses cannot be undertaken until all precautions are taken to give the greatest possible comfort and protection from irritation.

I am sure most veterinarians in equine practice have been looking for therapeutic measures to replace actual cautery. Prednisolone has, in part at least, supplied the answer.

However, it is no substitute for firing in the older chronic lesions where extensive pathological change has taken place, usually as a result of continued work when horses should have been rested and treated. When irreversible pathological changes have become established marked favourable influence cannot be expected. Early diagnosis and early treatment are followed by rapid, sometimes dramatic, improvement. However, even in lesions showing chronic change intra-articular cortico-steroids can be combined with actual cautery with satisfactory results. In fact, they can also be supplemented with other forms of therapy in acute and sub-acute conditions with excellent results. The cortico-steroid reduces the stiffness and pain and allows the limb to function, which prevents atrophy and adhesions and therefore helps recovery. Local treatment, with pressure, ultra-short wave therapy, electric heat and Faradic treatment makes ideal supplementation to cortico-steroid therapy in selected areas.

The intra-articular use of prednisolone, with adjuvant therapy as described, reduces the necessity for firing in the majority of horses. It has the advantage that it shortens the rest period necessitated by firing in a large percentage of cases. This is of great importance in the case of racehorses, where economy must be considered, as, in fact, it must be considered in all horses.

I make no claim that prednisolone is a panacea for traumatic synovial inflammations. It could not be when their aetiology is considered. The weakness in conformation which acted predisposingly in the first case cannot be removed. The weakness remains to act as a predisposing cause to recurrence. Predisposition caused by incorrect feeding can be overcome by suitably balanced rations and the rest interval necessary to reach normality in bone density.

I have used prednisolone in a number of horses that have been put back to work within a month and have given no further trouble. I have used it in other cases that have worked and raced for various periods, but the condition recurred and firing had to be resorted to. This does not mean that firing will always prevent recurrences. I have used cortico-steroid treatment successfully in horses entered for handicap races so that they could be maintained in training with only very short periods of rest, 4—5 days, and run in those races. There is no doubt that prednisolone is a boon to the

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veterinarian who is concerned in equine practice. It is particularly useful to me since my work is confined entirely to equines, mostly racehorses, where quick recovery and continuation of work are so important.

Although there is limited literature on therapy with corticosteroids in orthopaedic practice it is apparent that it is being used increasingly (Cosgrave, 1958), (Smith, 1956), (Uvarov, 1959), (Bailey, 1958).

It has been shown by Hagedorn, 1957, in humans, that prednisolone used intra-articularly, produces the same effect as hydrocortisone in less than half the dosage. It is free from the local exacerbation of symptoms that follow the local use of hydrocortisone. In my experience these findings are supported. Since using prednisolone I have never had a case of acute symptoms, following intra-articular injection, that I experienced with hydrocortisone and prednisone. However, these exacerbations were transitory, not lasting longer than 12—18 hours. Kendall, 1958, has shown that prednisolone has been effective where hydrocortisone has failed. He states, further, that prednisolone is more rapid in action, that there is a reduction in the incidence of local exacerbation and that the duration of effects are longer. My observations on equines support these findings.

Recently I have used prednisolone with hyaluronidase, with encouraging results, in bursal enlargements where refilling of the sac is so common. However, my numbers are insufficient to make a definite conclusion as to whether the added hyaluronidase improves the treatment. It is customary to combine hyaluronidase with diluted prednisolone in cases of widespread painful periostitis such as sore shins, a troublesome condition in two-year-olds.

The following table summarises the work I have done on inflamed, non-septic, synovial structures.

Joints, Sheath or Bursa Treated	Number Treated	No Recurrence**	Recurred*
Carpus (Knee)	43	29 (67.4%)	14 (32.6%)
Metacarpo-phalangeal Joint (Fetlock)	56	39 (69.6%)	17 (30.4%)
Tarsus (Hock)	6	4 (66.6%)	2 (33.3%)
Sheath of M. extensor carpi radialis	3	2 (66.6%)	1 (33.3%)
Digital Sheath (Fore)	2	2	
Digital Sheath (Hind)	1	1	
Olecranon Bursa	1	1	
Pre-Patellar Bursa	1		1
TOTAL	113	78 (69.0%)	35 (31.0%)

*These recurrences were in chronic cases, or after a short period of preparation or racing. Some horses had actually won in between treatment and recurrence.

**These are cases followed up that have gone into training and raced for a long period without recurrence.

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These cases, with the exception of 12, were acute or sub-acute. Shortly after commencing intra-articular prednisolone therapy I realised one could not anticipate the dramatic response in chronic lesions with irreversible change that could be expected in acute and sub-acute conditions. I discontinued treating chronic cases with prednisolone and used actual cautery with drainage alone or in combination with an intra-articular cortico-steroid. My observations are contrary to those of Smith, 1956, who states, in discussing intra-articular prednisolone, that his best results have been in horses that have been lame for a long period.

I have not yet had an opportunity to treat arthritis of the shoulder, elbow, hip, pedal or stifle joints by intra-articular injection. Shoulder and hip-lamenesses are not easy to diagnose, that is, it is difficult to say exactly what structures are involved. In cases of this sort, with questionable anatomical aetiology, I use rest, with intra-muscular prednisone and an analgesic, with remarkably good results. In fact, I have seen some extraordinarily rapid recoveries in horses that had been lame for long periods, when rest alone seemed useless. In such cases it is now used as a routine measure.

Four recent cases are: A four-year-old racehorse that had hip-lameness with marked atrophy of the gluteal muscles, for several weeks and had been rested in his stall without result: Within two weeks he was quite sound.

The second case was a two-year-old colt with shoulder-lameness of several months' duration, with atrophy of the superficial pectoral muscles. After two weeks treatment there was marked improvement; within a month the colt was sound.

The third case was a four-year-old in the same stable with the same history. One week after treatment he was sound and went into training and raced. The condition recurred; a further course of treatment gave a complete cure.

The fourth case was a six months old foal with hip-lameness, with marked atrophy of the gluteal muscles. This foal was so long lame with no improvement that the case was thought hopeless; within a month after treatment he was sound. I regard these results as not entirely coincidental.

I have used prednisone in combination with antibiotics in the treatment of septic bursitis of the bursa podotrochlearis with excellent results. This is a condition which in my experience, almost always required major surgical intervention after which the horse was frequently valueless for racing. That there is no objection to the use of prednisolone in severe infections, when combined with antibiotics, is now well recognised.

For general systemic use 200 mg. of prednisone is used intra-muscularly in adult horses: smaller horses get a smaller dose according to their weight.

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Dosage

I use 75 mg. Prednisolone acetate for each intra-articular and intra-thecal injection. Aseptic precautions are of course, observed. In 113 injections, carried out under stable conditions, I have not experienced a case of sepsis.

Technique

Thoroughbred horses in racing condition are, as a rule, not the easiest of patients. However, I have had no difficulty beyond an occasional restless horse.

The hair is cut with a scissors over the site of injection and a local anaesthetic used. For this injection a twitch is frequently necessary. For the intra-articular injection no restraint is necessary beyond having an assistant prevent the limb being lifted. All injections are done with the limb on the ground. In this way distended joints, sheaths and bursae are more easily felt. The selected site is the most prominent part of the fluctuation and where the synovial cavity is closest to the skin.

The fetlock is penetrated from the lateral aspect, dorsal to the lateral sesamoid, between the suspensory ligament and the metacarpal bone. The digital sheath is penetrated at the same level between the suspensory ligament and the deep flexor tendon. It can also be penetrated below the fetlock between the volar annular ligament and the proximal annular ligament. The sheaths of the extensor carpi radialis and common extensor present no difficulty. They are usually fluctuating elongated enlargements which are penetrated at the lowest extremity.

The knee joint can be penetrated conveniently on the lateral aspect in two sites, depending upon whether the radio-carpal joint, the inter-carpal joint or the carpo-metacarpal joint is involved: The latter joints are, of course, communicating. The radio-carpal joint is penetrated on the lateral aspect in the angle between the radius and the accessory carpal bone, just dorsal to the insertion of the lateral ligament of the accessory carpal bone to the radius. When pressure is exerted on the anterior aspect of the radio-carpal joint a fluctuating area can be seen and felt at this site.

The carpo-metacarpal joint is penetrated on the lateral aspect posterior to the lateral ligament at the level of the joint where anterior pressure produces a protrusion of the site in the distended knee. I find it rarely necessary to introduce the needle into the anterior aspect of the joint. In this case the needle is introduced in the most prominent part of the fluctuation between the tendon sheaths. The carpal sheath is penetrated on the lateral aspect just dorsal to the knee in the hollow between the lateral digital extensor and the ulnaris lateralis. The stifle joint is penetrated between the middle and lateral patellar ligaments. It can also be penetrated between the medial and middle ligaments. When draining the stifle joint there are two capsules to be drained, the femoro-patellar and the femoro-tibial. They are, of course, communicating, but may need separate drainage.

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The hock is penetrated on the medial aspect at the most prominent part of the fluctuation, either anterior or posterior to the saphenous vein. It can also be penetrated on the lateral aspect where the capsule protrudes between the tibia and the deep flexor tendon. The tendon sheaths of the hock present no difficulty. They are penetrated at the most prominent point of the fluctuation. A 20 gauge needle 2 inches long is used. It is always introduced ventro-dorsally to avoid accidents in case the limb is suddenly raised. The synovia is drained under pressure to the last drop. In acute conditions, with marked swelling and high-grade lameness, blood-stained synovia is frequently present, almost always. It is not infrequent to find blood-stained synovia, even in cases where horses show only a low or middle-grade lameness if they are drained within 48 hours. This indicates that there has been some tearing of the joint capsule or tendon sheath.

The prednisolone is then injected through the same needle. The joint is flexed and extended several times following injection. It is then enclosed in an aseptic pressure bandage which is left in position for 48 hours; in the knee and hock 3-4 days.

The horse is confined to the stall on the day of injection. The following day 15 minutes walking is given morning and afternoon, and the day after 30 minutes walking morning and afternoon. After this period he has a long walk daily.

I advise that walking exercise only be given for a month, where this is possible; after a month the horse is usually ready to begin a slow preparation.

When horses are resting after treatment they are usually given supporting therapy by compression bandage and electric heat. The results are frequently dramatic.

When the bandage is removed after 48 hours the joint, tendon-sheath or bursa appear almost normal. In very acute cases heat and pain are reduced; lameness is greatly improved. Within 7—10 days only very acute cases are not going sound. The sub-acute cases are invariably sound. However, the precaution is taken to give them 30 days' rest, except in the exceptional cases mentioned.

I have occasionally repeated the treatment, within 7—10 days, where response was not satisfactory. However, it is rarely necessary. I have treated the same horse several times for recurrences over a period of 19 months. During this period he was prepared for racing, won a good race and was second in two of our biggest handicaps. During the treatment with prednisolone there was no indication of the usual changes associated with chronic arthritis.

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DISCUSSION

PROF. HOFMEYR expressed the disappointment of the meeting in the fact that Prof. Quinlan had not been able to attend and present the paper and reply to the discussion.

Prof. Hofmeyr stated that he was sure we all agreed that "firing should be avoided if at all possible" provided we had an adequate substitute. He pointed out that the duration of treatment mentioned by Prof. Quinlan was minimal compared to accepted standards with firing and stated that he would have liked to have had that point discussed with the author. Referring to the table summarizing the results it would be seen that permanent recoveries were listed as two-thirds and breakdowns as one-third of the total. It was not clear exactly what was considered a "breakdown" or how long the cases were followed up.

DR. STEYN pointed out that using human practice as a comparison was not always justifiable. He mentioned this because **Prof. Quinlan had** stated that these remedies had been applied in human practice for ten years and only now were they being used by veterinarians. In human practice the criterion of success was often the relief of pain and discomfort. In small animal practice our standards were somewhat similar but in equine practice nothing short of restoration to full functional recovery satisfied the owner or the practitioner. Judged by these standards cortico-steroid therapy often failed dismally. This was apparent even from the figures quoted by Prof. Quinlan. Dr. Steyn continued: "We know, too, that the beneficial effect of the cortico-steroids is based primarily upon their so-called *anti-inflammatory* effect, *not* upon their *healing* effect. In other words, one can expect the rapid disappearance

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of inflammatory changes and with it, the disappearance of the clinical symptoms of pain in those cases in which the injury within the affected synovial structure is slight. In cases where the injury is more extensive and in which permanent recovery is dependent upon the normal healing of the damaged tissues, cortico-steroid therapy must fail. The best that one can expect in these more serious cases is a temporary relief of the pain symptoms.

Unfortunately, the degree of lameness and the amount of observable local reaction is very often a poor guide to the extent of damage which has been sustained. For this reason it is very difficult at present to diagnose accurately before treatment which cases are really suitable for treatment with the corticoids and which are not.

When the chances of failure with any particular type of treatment are as high as 33%, that treatment must be considered a bad treatment. More especially is this the case when the treatment is expensive.

I do not wish to suggest that cortico-steroid therapy should be condemned, but I do state emphatically that it should be used with caution and only in selected cases. As indicated earlier, the selection of suitable cases presents considerable difficulty, and one only learns which cases are likely to be most suitable for treatment after a fair amount of experience.

As an illustration of the type of difficulty one may encounter, I quote a few examples of recently treated cases:

A colt developed a pronounced thoroughpin of the off-hind without lameness. This case was treated with prednisolone and after three intra-synovial injections at weekly intervals, a perfect recovery resulted. The colt has won one race since treatment.

A few months later another colt of the same age as the former, i.e. a two-year old, was presented with a pronounced thoroughpin of the near-hind and a very slight distension of the deep flexor sheath of the off-hind. In this case, too, there was no lameness. I felt confident of a good result with prednisolone, but after two treatments, the near-hind was much worse. The off-hind, the slightly affected sheath, responded well with one treatment and a second treatment was not necessary.

Clinically the only difference between these horses was: the first case suffered from no conformational defect, whereas the latter suffers from bad conformation.

Similarly, two horses in one stable were treated for arthritis of the knees and fetlocks. One of them recovered spectacularly, the other improved but was not fit for racing. Again the main difference was one of conformation.

One cannot conclude from these four cases that horses with bad conformation should not be treated. I believe that it is the degree of injury which has preceded treatment which is of vital importance, and if one is able to obtain a reliable history in any particular case, it is of more value as a guide to prognosis than

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mere conformation. Always, however, one must remember that conformation must be of vital importance in any long-term prognosis.

There are one or two statements made by Prof. Quinlan which I feel it is my duty to challenge.

The first of these is his statement about firing. I agree with him that firing should be avoided if there is any other treatment which might give equally good results. I also agree that firing may grate upon one's aesthetic sense, but this can be overcome to some extent by the use of sedatives, etc., post-operatively. I, however, heartily disagree with his statement that firing reduces speed by fibrous tissue formation.

May I quote a few examples? I shall confine myself to only a few horses which I have fired, not mentioning a number which I know were fired by colleagues and which could be used to strengthen my argument.

First of all, there was Radlington. This horse ran second in the Durban July twice and on the second occasion, at least, the race was run in what was then a new record time. In fact, Radlington, although second, broke the former record.

A less known, but nevertheless very good horse, Darwin, was fired by me. He only reached the top division after he had been fired.

Sea Lord suffered from a badly sprained suspensory ligament and certainly would not have raced again had he not been fired. I do not believe this horse was as good *after* he had been fired as he was before, but, nevertheless, he won some thousands of pounds after having been fired, including a number of what might be called our lesser classics. And it was his disability which affected him, not the firing.

I could list a very large number of lesser lights, but the foregoing will serve to illustrate my point. None of these horses is still racing — that is why they are mentioned by name.

The second statement with which I disagree is the advice that cortico-steroids should be used in the presence of infection, e.g. in septic bursitis. I agree that usually the combination of one or more antibiotics with a corticoid reduces the danger of a flare-up of the infection, but it is a dangerous procedure to adopt as one might have the ill luck to encounter a resistant infection. In such an event, the result may be disastrous. Once the infection has been brought under control there is no objection to the use of corticoids if this was still considered necessary but in all probability that necessity would have passed."

DR. IRWIN: It is useful to distend the capsule before aspirating the synovia. We use an elastocrepe bandage, leaving only one small area of the joint capsule uncovered. We also find it easier to keep a tube gauze pressure bandage on the hock than the conventional elastocrepe roller bandage.

DR. BOSWELL: I agree with Dr. Steyn that we get better results in acute than in chronic cases but one can never tell in chronic

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cases; some do surprisingly well while in others the results are very disappointing. We have had several instances where horses have done very well after firing. Although firing does appear somewhat barbaric, I do not think we can discard it, at least until we find something better.

PROF. HOFMEYR: The effects of prednisolone treatment and firing are in certain ways opposite. Prednisolone reduces inflammation while firing produces it. This difference is not always fully appreciated. We use X-ray examination as a routine. Bony tissue can only be resorbed by hyperaemia which is produced by firing. Where there is excess bone formation I cannot see that prednisolone will have any effect. Another difficulty is that our assessment of joint pathology is mainly clinical and subjective. A very interesting investigation has been made by Eggers in Vienna. He has studied the cytology of the synovia. Normal synovia contains 100 to 300 lymphocytes per c.m.^{3} In acute synovitis this figure is increased up to tenfold. In normal synovia the relationship between young and mature lymphocytes is 30 to 70. By assessing the relative percentages one can assess the age of a lesion. Mesothelial cells increase in chronic aseptic inflammations. Neutrophils are only found in septic conditions where the figure may be as high as a million per c.m.^{3} This work promises to give some objective criteria in judging the age and nature of injuries.

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Pfizer RESEARCH REPORT

Antibiotics

— THE FACTUAL APPROACH

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We at Pfizer share the belief of most independent authorities that the real criterion of the value of any antibiotic is its effectiveness and its economy *in practice.*

When micro-organisms are to be selected for comparison, they should be confined to those which are associated with actual disease conditions met *in the field,* otherwise the results will be of academic interest only.

Whilst "in vitro" tests might serve some purpose in indicating the *probable* usefulness of a group of related antibiotics, there is agreement between most authorities that they can achieve no more useful purpose than that.

The real Test

The real test is what antibiotics will achieve "in vivo" as an example of which we quote from a paper entitled "The value of bacterial sensitivity testing in the treatment of urinary tract infections (a study in correlations)", *Swanton and Lind in Antibiotics Annual 1956/57 pp. 191 to 195.*

"... In the case of the tetracyclines the organism need be sensitive to only one of the three tetracyclines in order for therapy to be effective using any one of them."

"... It is apparent from the "in vitro" studies that testing sensitivity of certain strains of bacteria to this group of broad spectrum antibiotics becomes of simple academic interest but offers little practical value."

The practical answer

Under South African conditions, in veterinary practice, Terramycin has proved itself over and over again to be most effective at *normal* dosage levels at *minimum cost* in the treatment of all those infections in which the use of the tetracycline group of antibiotics is indicated.

It has always been the earnest endeavour of Pfizer to place the most convenient dosage forms at the lowest possible price in the hands of the profession, and at the same time to make no claims other than those which can be *fully* substantiated in clinical practice in this country.

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SOME VASCULAR DISEASES IN THE DOG WITH A NOTE ON POSSIBLE TREATMENT

J. F. DU PLOOY

Although the vascular system of the dog is relatively disease-free, there are however, a few conditions which are of importance. As we become more proficient in the use of the E.C.G. and such tests as the Cholesterol and Lipid tests can be done, and correctly assessed, vascular conditions will play a more important role in small animal practice.

Heart-worm Infection:

While of no consequence in this country, Heart-worm infection constitutes one of the most serious canine disease problems in the Southern States of America. Minnick[1] reports the prevalence among hunting dogs as follows: 68—74% in 3—5 year-olds and almost 100% in dogs over 5 years. The adult Heart-worm *Dirofilaria immitis* is usually found in the right ventricle and in the pulmonary artery of dogs.[2] Microfilaria swarm in the bloodstream and are transmitted by blood-sucking arthropods.

Symptoms: Early tiring, gasping for breath and collapse on exertion, coughing, convulsions and dropsy.

Treatment: Sodium Arsenamide in doses of .1 cc./lb. body weight administered intravenously for 15 days. Dead worms may serve as emboli in the smaller vessels.

Neoplasms:

There are only two specific tumours of blood vascular tissue (1) Haemangioma (Benign), consisting of newly-formed vessels and (2) the Malignant Haemangio-endothelioma composed essentially of growing endothelium.[3]

Incidence — Froehner[4] found two haemangiomas among 643 tumours of dogs, and Feldman only 2 among 81. In a total of 1,706 neoplasms (1,501 canine and 205 feline) observed at the Angell Memorial Hospital, 64 were of the blood vascular system, 54 in dogs and 10 in cats. 41 were haemangiomas and 23 haemangio-endotheliomas, affecting mostly the spleen 31, liver 11, tongue, rectum, anus omentum, etc. Sites of metastatic involvement were brain, liver, adrenal, lymph nodes, mesentery, lung and heart.

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History and Symptoms

Typically the patient is presented with a history of sudden weakness. Pallor is marked. Sometimes the animal is already in shock and there may be a history of injury. Occasionally the owner may report a progressive downhill course, with weight loss, thirst and dizziness. Examination often discloses a palpable well-defined mass in the anterior abdomen, with tenderness and weakness. If a vascular tumour is suspected the patient must be handled cautiously, or fatal bleeding may be started. I have diagnosed vascular tumours of the spleen on numerous occasions by palpation and examination of the mucous membranes alone. As all these patients were aged, permission was not granted for splenectomy. A 3-year-old Corgi was presented in a state of severe shock with the history that it had been kicked. On examination it was obvious that the dog was haemorrhaging internally and suitable treatment was instituted. The animal succumbed 10 minutes later, and on autopsy it was found that a vascular tumour of the spleen had ruptured, leaving the spleen in two distinct halves. Since all the symptoms cited are non-specific, diagnosis is based on history, physical examination, laboratory reports (Haematocrit) and the clinician's experience. Meier cites a case of death in a dog due to rupture of a myocardial haemangio-endothelioma of the right ventricle.

Treatment

Splenectomy is curative in the benign type and usually curative in the malignant type if done early. If the patient's condition does not permit immediate surgery, treatment consists of transfusion and fluids and vitamin B12 to combat dehydration and anaemia. As far as skin lesions are concerned, wide excision or electrical removal may obviate recurrences.

ARTERIOSCLEROSIS — ATHEROSCLEROSIS:

For the sake of clarity we will regard Arteriosclerosis as the thickening of the arterial wall with connective tissue and/or calcium deposition, Atherosclerosis as the dimunition of the size of the vessel due to fat deposition on the intima.

Arteriosclerosis is associated with hypertension and chronic renal disease and atherosclerosis with hyper-cholesteraemia. Some observers regard these conditions as identical. Atherosclerosis is no longer generally regarded as a degenerative phenomenon associated with ageing, and therefore inevitable and irrevocable, but rather as a pathological disorder which is apt to become manifest in later life. There is no agreement however as to the pathogenesis of this disorder, and in particular, whether it is due to genetic or environmental factors. Of the lipids indicated in atherogenesis, the role of serum-cholesterol has been extensively studied because of the early discovery of cholesterol in atheroma, the frequent occurrence

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of atherosclerosis in clinical conditions associated with elevated serum-cholesterol and the experimental production of atherosclerosis by feeding cholesterol to susceptible animals. Premature or accelerated atherosclerosis occurs frequently in three clinical conditions, diabetes, hypertension and hypercholesteraemia. Other facts that may be implicated in atherogenesis are phospholipids, triglycerides and total lipids.[5]

According to Ratcliffe[6] social pressure has become a major factor in the rising frequency of arteriosclerosis at the Philadelphia Zoo. This is due, he suggests, to an imbalance in adrenal secretion and to inactivity. If this is true in zoo animals, it could also play a big rôle in dogs, especially in the lap dog varieties. The most striking increases in the frequency of the disease have occurred since 1946, " 10 years after diets were improved (but remained low in fat) and in association with decreases in mean age," they said. Thus they maintain that in the development of this disease, factors other than diet and age may have a major influence.

Kuo *et al*[7] maintain that lipaemia may be one of the factors that precipitates or aggravates acute arterial insufficiency in arteriosclerotic patients. Satisfactory control of lipaemia is therefore indicated in patients with severe coronary and peripheral vascular diseases.

After 10 years of observing cardiac diseases in the dog, Detweiler[8] has concluded that cardiovascular disease among dogs is far more common than has been generally realised, and that the incidence and importance of arteriosclerosis in the dog have not been established. Much research is needed on the possible relation between chronic renal disease and cardiovascular diseases; the incidence and underlying causes of spontaneous hypertension in the dog remain to be clarified. The incidence of arterial hypertension, in some cases of which an association with chronic renal disease can be shown is estimated to be .9% or higher.

According to McGill[9] *et al* two forms of necrotizing arteritis relating to diet and renal insufficiency occur in dogs. The type produced by high fat diet combined with renal insufficiency characteristically occurs in the large elastic arteries and the endocardium.

In the type produced by renal insufficiency alone, the most consistent lesion is necrosis of the walls of the small muscle arteries.

THROMBOSIS:

Due to the fact that the coronary arteries are end arteries, thrombosis in them is extremely dangerous. If the thrombus is slow forming and the anginal pains pass over after a few minutes, treatment with appropriate drugs can prolong life, but if the thrombus forms rapidly, death supervenes rapidly. In arteries other than the coronary arteries, a collateral circulation is set up to prevent infarction and necrosis unless the lumen of the artery is totally obliterated by the thrombus. Using this principle of collateral circulation, Professor Thompson[10] has performed an operation for

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the treatment of sclerosis and thrombosis of the coronary arteries by opening the pericardial sac and aspirating the fluid. He then dusts four grams of sterilized talc into the pericardial cavity in the vicinity of the diseased artery. This causes adhesions of the pericardial sac to the myocard and a collateral circulation is set up from the vessels of the pericardial sac. So far constrictive pericarditis has not resulted.

A new operation of the coronary arteries was performed in the dog by Bailey.[11] It consists of the partial removal of obstructive matter from diseased coronary arteries. After identification of the artery, a half inch incision is made into the distal end. A curette with a lumen in its shaft, is introduced into the artery through the incision, and worked towards the proximal portion until the passage of the curette through the obstructive matter was confirmed by the flow of blood through the lumen of the shaft. Without stopping the heart or lung, the surgeons removed the occlusion by working the curette towards the incision. They then sutured the adventitial tissue over the wound. Important to the success of this procedure, was the administration of adequate quantities of heparin to the patient both during the operation and for several days thereafter.

A four-year-old Dobermann was presented with the history of suddenly having gone acutely lame after having slightly dragged the off-hind leg for three days. On examination, the cause of the lameness could not be discovered until it was realised that the pulse was absent. That fact plus the coldness of the affected limb prompted a diagnosis of iliac thrombosis, and the possibility of an operation was suggested. The owners would not hear of it and insisted that the animal be destroyed in their presence. On autopsy a thrombus was found in the right external iliac artery. It completely occluded the vessel and extended for about 5 centimetres down the iliac artery and 2 centimetres into the abdominal aorta. As the thrombus was not very hard and only lightly attached to the intima, I am of the opinion that Bailey's operation could have been used with success in this case.

The favourable results achieved with dicumarol and heparin in the treatment of coronary thrombosis has been attributed to their action as anti-coagulants. It appears however that the beneficial effects may be due in large part to the action of increasing coronary blood flow, that is vasodilation.

Nalefsi and Gilbert[12] have demonstrated that dicumarol and heparin resemble the xanthines in their action on coronary vessels. " Miltrate " and " Comoudin " are modern drugs used in the treatment of thrombosis.

EMBOLISM:

Embolism may be caused by a thrombus, fat or air.

Thrombo-embolism often results from detached portions of growths on the heartvalves in chronic bacterial endocarditis.

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Treatment consists of oxygen by mask, atropine and papaverine intravenously.

Fat embolism has been the cause of death in two cases in my experience. Both animals had severely fractured femurs and damage to the blood vessels. Open reduction was resorted to and the fractured ends plated, a lot of bone marrow was cleared away and damaged vessels ligated. The next day both animals exhibited signs of cyanosis, dyspnoea and restlessness, and subsequently died. No signs of embolism were visible in the lungs, and it is probable that embolism of the coronary arteries was the cause of death, because, according to Geza De Takáts[13] fat carried from the site of injury through the peripheral veins to the right heart is capable of passing through the capillaries of the lung and thus enters the general circulation. This depends on the amount of mobilised fat and on the blood pressure of the pulmonary artery. The fat may pass through the kidneys into the urine or it may cause embolic occlusion of any organ, e.g. heart, brain, kidney, etc.

PATENT DUCTUS ARTERIOSUS:

Saunders[14] describes the technique for surgical closure of Patent Ductus Arteriosus. After thoracotomy the left lung was compressed, the Aorta and Pulmonary artery were identified by following the course of the recurrent nerve and the position of the ductus was determined. The pericardial sac was incised and the ductus freed from surrounding tissues and prepared for ligation. Before permanently ligating the ductus the most important consideration is the actual rôle of the ductus. In most cases it is simply a prenatal structure that had not receded normally. If this is not the case then the ductus serves as a compensatory structure. In septal defects or transposition of large vessels (Tetralogy of Fallot) the ductus may persist to increase pulmonary circulation. The so-called "Blue Baby" operation creates an artificial ductus to serve the same purpose. If a compensatory ductus is ligated, death or severe circulatory embarrassment will follow. The role of the ductus can only be determined at the time of operation, and this is done by temporary occlusion of the ductus. If the structure plays a compensatory role cyanosis will soon become manifest. Saunders concludes that life expectancy of the dog was considerably lengthened by the surgical correction of the condition.

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DIE DIFFERENSIALE DIAGNOSE VAN SENUWEE-AANDOENINGS BY ONS HERKOUERS

J. D. SMIT
Onderstepoort

'n Hele verskeidenheid van aandoenings met senuweesimptome kom by ons herkouers voor. Dikwels is dit moeilik of selfs onmoontlik om met 'n kliniese ondersoek die etiologiese oorsaak van die toestand vas te stel. In baie van die gevalle kan die korrekte diagnose alleen gemaak word nadat 'n deeglike lykskouing uitgevoer is. 'n Ondersoek van die brein word gewoonlik beskou as 'n tydrowende taak wat nie die moeite loon nie en word dan nagelaat. Nogtans kan 'n breinondersoek dikwels 'n mens in staat stel om 'n korrekte diagnose binne 'n kort tyd te maak. Verskeie siektes kan alleen met 'n breinondersoek gediagnoseer word of anders vergemaklik dit die diagnose.

As voorbeeld kan ons hartwater noem. Deur die bereiding van smere op so 'n manier dat 'n groot aantal kapillêre vate in die smeer voorkom, kan hartwater maklik en betroubaar gediagnoseer word. In dié verband moet ek wys op die belangrikheid van deeglike kleurmetodes. Dikwels word verkeerde diagnose gemaak omdat die smeer onderkleur is. Die diagnose word baie vergemaklik indien die groepe van kapillêre vate met 'n lae vergroting gesoek word en dan oorgeskakel word na olie. Die diagnose van hartwater kan ook gemaak word deur die maak van intimasmere, maar dit is moeilik en gee geensins 'n betroubare diagnose nie. Deur 'n sistematiese ondersoek van groot getalle niere van positiewe gevalle is vasgestel dat in ± 50% van gevalle 'n positiewe diagnose gemaak kan word deur die ondersoek van niersnitte. Die parasiete kom dan in die kapillêre van die glomeruli voor.

Teneinde die oopmaak van die brein uit te skakel, is smere gemaak van die nekmurg, maar ook hier kan alleen in omtrent 50% van gevalle die diagnose bevestig word. Breinsmere, en veral hippocampussmere, bly dus die enigste betroubare diagnostiese metode. Breinsnitte kan ook gebruik word, maar dit is tydrowend en nie naasteby so betroubaar as smere nie.

Gedurende die laaste paar jaar het ons gevind dat daar 'n groot vermeerdering in die voorkoms van gevalle van breinrooiwater is. In tipiese gevalle van breinrooiwater kan die diagnose alleen gemaak word deur middel van breinsmere. Dieselfde metode word gebruik as by hartwater. In baie gevalle is beide bloed en miltsmere nega-

tief en is die hele P.M. negatief vir die gewone rooiwater. Breinsmere toon egter 'n massale besmetting. Feitlik elke rooisel bevat parasiete. Dit lyk of daar 'n agglutinasie van besmette rooiselle in die kapillêre vate plaasvind. Die vaat raak dus verstop en vertraag die normale bloedvloei met die gevolg dat senuweesimptome ontwikkel.

Soortgelyk hieraan vind ons ook gedurende die laaste paar jaar 'n toename in gevalle van draaisiekte of die breinvorm van Theileriose. Die gevalle het eers net in Pretoria en Rustenburg voorgekom, maar onlangs is gevalle selfs tot in Pelgrimsrust vasgestel. Die uitstaande simptoom is die neiging om altyd na een kant te draai, verder is die dier gewoonlik blind, veral in die een oog. In die laaste stadia verloor die dier sy balans of lê in ekstremis half verlam. Die diagnose kan alleen bevestig word deur 'n ondersoek van die sentrale senustelsel. Die mees opvallende letsel is 'n trombose van die bloedvate van die breinvliese en/of die brein. Veral die kleinere vate is aangetas.

Die dele van die brein wat deur hierdie vate voorsien word toon dan 'n fokale nekrose. Die letsels mag selfs in die rugmurg voorkom. Die diagnose word gemaak deur smere te maak van die inhoud van die bloedvate met die trombose. Die trombus bestaan in hoofsaak uit makrofage waarin die Koch se liggaampies gevind word. Ook in die aangetaste dele van die brein kan die selle met Koch se liggaampies gevind word. Smere is die maklikste metode, maar breinsnitte toon gewoonlik ook die Koch se liggaampies.

Veral die gevalle met rugmurgletsels kan maklik verwar word met rugmurgabsesse. Rugmurgabsesse is natuurlik 'n belangrike oorsaak van senuweeaandoenings by beeste. Die gevalle kom veral onder jong beeste voor, en meesal in die lumbale deel van die rug. Volgens prof. Schulz is die predisponerende oorsake hoofsaaklik: ouderdom, fosfaattekorte, ruwe hantering en toevallige etterprosesse iewers in die liggaam. By laasgenoemde speel 'n swaar bosluisbesmetting waarskynlik 'n rol. In lammers kom rugmurgabsesse ook voor, meesal as 'n komplikasie van wondbesmetting na die afsny van sterte.

Onlangs het verskeie gevalle van 'n purulente encephalitis en/of meningitis a.g.v. preisznocardbesmettings by skape voorgekom. Met die skynbare algemene toename in preisznocardbesmettings deur die land mag meer sulke gevalle voorkom. Dit kom ook soms voor as 'n komplikasie van 'n swaar *Oestrus ovis* besmetting met sekondêre bakteriese besmetting van die sinusse en verspreiding van daar na die brein. Naverwant hieraan is ook die gevalle van middeloorontstekings wat dikwels by skape en varke voorkom. Dit is gewoonlik 'n purulente infeksie van die middeloor. Die patogenese is nog nie altyd duidelik nie. In sommige gevalle, veral in varke, kom dit waarskynlik deur die Eustachiaanse buis van die keel na die oor. In ander gevalle, veral in skape, kom die besmetting waarskynlik van buite, moontlik as komplikasie van bosluisbesmetting of irriterende middels. Die toestand mag groot afmetings onder 'n

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kudde skape aanneem. In sommige gevalle sprei dit ook na die brein of veral in varke mag dit die been (mastoied) aantas. In varke is die simptome taamlik kenmerkend, die diere lê op die aangetaste sy en as jy hulle omdraai spring hulle onmiddellik terug. Belangrik is dat die uitwendige oor dikwels geen letsels toon nie. Die middeloor moet dus ondersoek word. In die vark kan dit maklik gedoen word deur die gehoorbeentjies met 'n mes uit te lig nadat die brein verwyder is.

Die voorkoms van lintwurmblase in skape is natuurlik welbekend. Ek kan egter daarop wys dat dit soms groot afmetings mag aanneem. In een geval is bv. meer dan 'n honderd skape in 'n kudde aangetas. In baie van hierdie gevalle was 'n akute eosinofilêre encephalitis en meningitis die vernaamste letsel. Die migrerende parasiete was verantwoordelik vir boorgange wat besmet geraak het, geen blase was nog gevorm nie. Vandaar die suksesvolle behandeling deur sulfa en antibiotiese middels.

Traumatiese letsels van die brein moet ook in gedagte gehou word. Veral in beeste mag horingbreuke aanleiding gee tot bloedings op die brein, veral tussen die breinvlies en been.

Gewasse kom ook voor in die brein, breinvliese of been. Verskillende tipes mag voorkom, wat veel mag verskil in kwaadaardigheid. In hoofsaak is egter die druk wat uitgoefen word op die brein as gevolg van die beperkte skedelruimte. Dit is merkwaardig hoe die dier hom kan aanpas by die verlies in brein weefsel deur drukatrofie indien die proses nie te snel plaasvind nie. Mens staan verstom oor die klein persentasie van brein wat oorbly, en selfs dan wys die dier nie opvallende senuweesimptome nie.

Hidrocephalus interna kom ook dikwels voor en mag verantwoordelik wees vir senuweesimptome. Die oorsaak is nie altyd maklik vas te stel nie. Belangrik is die gevalle wat voorkom by skape as 'n komplikasie van bloutong. Wanneer dragtige diere wat ten volle vatbaar is tussen die 4de en 8ste week van dragtigheid bloutong opdoen, of geënt word, mag hierdie gevalle voorkom. Dit kan vergelyk word met wat gebeur by die vrou by Duitse masels. Sommige lammers word dood gebore, ander is swak, blind en mag 'n variasie van senuweesimptome toon. In lammers wat 'n paar maande oud word is die hidrocephalus interna baie opvallend. In een uitbreek was tot 60% van die lammers aangetas.

Ek kan nie nalaat om hondsdolheid te noem by die differensiale diagnose van senuweeaandoenings nie. Veral by beeste kan dit maklik verwar word, veral met breinrooiwater. Ek moet die belangrikheid van die hippocampus by die histologiese diagnose beklemtoon. Dit is die enigste deel van die brein waarop 'n betroubare histologiese diagnose gemaak kan word.

Selfs 'n siekte soos bloednier gee soms senuweesimptome. Veral in diere met gedeeltelike immuniteit kan soms baie opvallende senuweesimptome voorkom. Dit stem dan baie ooreen met hartwater en word klinies dikwels daarmee verwar. Die diere mag vir

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dae in 'n koma lê en gallopbewegings maak, selfs die knip van die oë kom voor.

Verskillende vergiftigingstoestande gaan gepaard met senuweesimptome, veral is daar verskeie gifplante hiervoor verantwoordelik. Ek wil egter nie hierop ingaan nie. Interessantsheidshalwe wil ek net noem die gevalle van blindheid wat mag volg op tjienkerintjievergiftiging. Opvallend is die skynbare gesonde oog. Die blindheid is die gevolg van beskadiging van die optiese senuwee en bloedings in die retina. In Oos-Transvaal het ek heelwat gevalle teëgekom a.g.v. *O. saundersii.* Van die swamvergiftigings moet ek wys op die belangrikheid van *Diplodia Zeae.* Die gevalle kom baie meer voor in warm dele en in besonder op die Springbokvlakte. Op die hoëveld, waar die winters koud is, kom gevalle selde voor, nieteenstaande die feit dat vogtigheid daar meer geskik is.

Loodvergiftiging is ook baie belangrik. Senuweesimptome kom gewoonlik voor en mag verwar word met verskillende toestande, bv. hartwater, lamsiekte, ens. 'n Breinondersoek is hier ook tot groot hulp. Die brein het 'n opvallende vuil bruin kleur en 'n swaar kongestie van veral die breinvliese. Die diagnose kan met 'n groot mate van sekerheid op die voorkoms van die brein gemaak word indien die nodige ondervinding van dergelike gevalle bestaan.

Hierdie voordrag moet beskou word as 'n oorsig van ondervindings opgedoen oor die loop van jare by die hou van lykskouings en die probleme om 'n korrekte diagnose te maak.

DISCUSSION

DR. ADELAAR (Opener) mentioned numerous conditions causing nervous symptoms in cattle including bacterial and viral diseases as well as poisonings and deficiency states.

DR. SHONE (S. Rhodesia) described an outbreak of Phalaris staggers in which 60 sheep were lost. The symptoms were constant nodding of the head and a peculiar rocking horse-like action when the animals were disturbed. Post-mortem examination revealed no specific lesions. The condition could be prevented by dosing cobalt or by applying it to the soil but not by the injection of vitamin B_{12}. This was the only outbreak to date although Phalaris was widely planted in Southern Rhodesia.

DR. VAN DER LINDE mentioned an outbreak of carbolineum poisoning in cattle in which the symptoms were depression, blindness and unsteadiness. He also described two outbreaks of rabies in which the symptoms were atypical even for the "dumb" type. They consisted of aimless turning movements, constant licking, rubbing and moaning without aggressiveness, bellowing or salivation in the early stages. The animals died in coma with profuse salivation.

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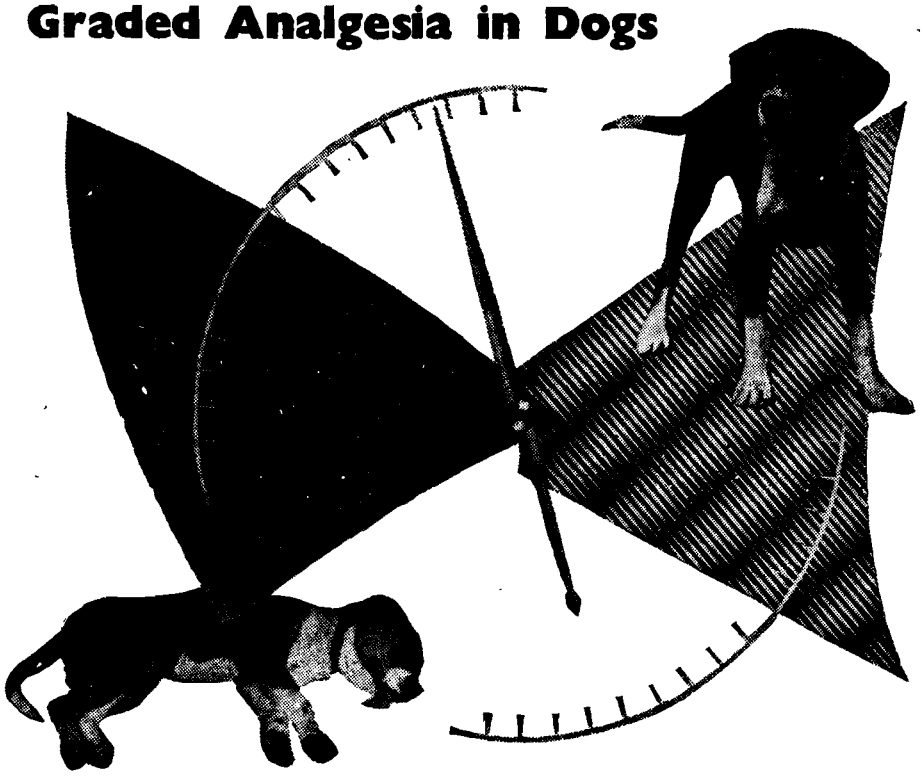
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DIAGNOSIS OF BRUCELLOSIS IN SHEEP AND GOATS

G. C. VAN DRIMMELEN
Onderstepoort

Abstract: A preliminary report on the diagnostic procedures applied in brucellosis of sheep and goats; with special reference to the genital brucellosis of Merino sheep in South Africa.

Diagnosis of the different forms of *Brucella* infection in sheep and goats has recently become a very pressing problem in South Africa. There exists an infertility in flocks of all breeds, which appears to be primarily or wholly due to genital brucellosis. The spread of this disease has to be combatted at present by guarding against the introduction of infected stock into clean flocks and by building up clean flocks from uninfected young stock.

Although sheep are easily infected with *Brucella* organisms they frequently do not suffer severely from the disease. Relatively few abortions, relatively mild lesions and a high rate of spontaneous recovery lead to a comparative disregard of the infection. Only the flocks of karakul sheep, in which the handling of new-born lambs and their pelts has resulted in frequent human infection, have attracted attention to brucellosis.

The recent discovery of widespread infection in the semen of infertile Merino rams is, however, at present causing great concern. This condition calls for a reliable diagnostic technique, firstly to establish the presence of the infection in a flock and secondly to distinguish individual carriers of infection in order to build up or protect clean flocks.

Material

An infectious disease is best diagnosed by isolation of the causal organisms. This is an expensive process in ovine brucellosis and not likely to succeed with the present technique in more than 10% of cases. After outbreaks of abortion, we have found evidence of *Brucella* infection of sheep and goats in S.W.A., the Northern Cape, the O.F.S. and the Transvaal Bushveld. Our earliest cases investigated were received from Otjiwarongo, Omaruru, Outjo, Marienthal and Prieska.

A direct diagnosis by means of microscopical tests on specimens is the second best method of diagnosis. One foetal specimen from Prieska, one from Kroonstad and lately one from Potchefstroom

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permitted microscopical diagnosis of *Brucella* infection in contaminated foetal material. Such material is difficult to obtain and more difficult to deliver at a diagnostic laboratory in a satisfactory state to permit proper examination. This examination should be carried out on the spot, as the technique is simple and inexpensive.

Concerning the genital infection of rams the greatest advance has been made possible by the valuable and very practical contribution of van Rensburg and de Vos.[2] These workers devised an apparatus for semen collection, which is so effective and reliable, so economical to operate and so simple in construction, that it will soon be a regular component of the armamentarium of every veterinary practitioner in sheep farming areas.

Semen smears of rams in many districts have been found to contain *Brucella* organisms. Infection has been diagnosed mainly in the Karroo and the Northern Cape districts; but also isolated farms in the O.F.S., Natal and Transvaal.

For the rest we have been committed to indirect diagnosis by means of blood serum samples submitted from all over Southern Africa, where epidemics of abortion or cases of infertility have occurred.

An individual allergic test would be a great help. Unfortunately, experience in mediterranean countries has proved these tests to indicate all previous contact with the antigen and not only the carrier state. It is therefore useful and at the end of an eradication campaign, and may be considered in uninfected areas of this country to prove freedom from brucellosis.

Methods

Isolation of the causal organism is done from the foetus, foetal membranes, colostrum and the semen. Occasionally testicular lesions excised at P.M. have yielded cultures of *Brucella* organisms.

For microscopical examination smears are made from the same materials and from cultures.

Staining: The staining methods in use are (all smears are dried in air and fixed by gentle heat):—

(1) Stamp, McEwen, Watt and Nesbit[2] modified Ziehl-Neelsen acid-fast staining method:

Slide flooded with 3.0% Carbol Fuchsin — 10 mins.
Decolorized with 4% Acetic Acid — 1-10 secs.
Washed and counter-stained with 1.0% Methylene Blue — 2 mins.
Washed and dried in air: (*Brucella* = RED).

(2) Hansen's staining method:

Slide flooded with 3 parts saturated alcoholic Methylene blue freshly mixed with 10 parts 0.04% KOH — 5 mins.
Washed and flooded with 1.25% Safranin (Geigy) dissolved in H_2O (at 37°C) — 1.3 secs.
Washed and dried in air: (*Brucella* = BLUE).

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(3) Koester's staining method[3]:

Slide flooded with 2 parts saturated alcoholic Safranin freshly mixed with 5 parts N/$_1$KOH (= 5.6%) — 1 min.

Washed and flooded with 0.1% H_2SO_4 — 10 secs.

Washed and flooded with 1% Carbol Methylene Blue — 2-3 secs.

Washed and dried in air — (*Brucella* = RED).

(4) Gram's staining method:

Slide flooded with 2 parts saturated alcoholic Safranin

Poured off and flooded with 0.7% KI — 2 mins.

Washed with Acetone-Alcohol — 1-10 secs.

Washed and counter-stained with 1% Safranin — 1 min.

Washed and dried in air

(*Corynebacterium* = VIOLET).

(*Brucella* = RED).

Serological tests employed:—

(1) Agglutination in physiological saline with *Br. abortus* antigen standardised to O.I.E. sera ([5]).

(2) Agglutination with other antigens.

(3) Agglutination in 5% saline ([6]).

(4) Blocking antibody tests ([7]).

(5) Coombs antiglobulin tests ([8], [9], [10]).

(6) Complement fixation tests.

(Opsonocytophagic tests and Flocculation tests have not yet been satisfactorily carried out in the present work).

Results

Our earliest cases available for investigation were received from veterinarians in S.W.A. A number of strains were isolated; the majority of which behaved in such atypical manner on culture, that we were unable to type them with the techniques at our disposal. *Br. melitensis* and *Br. abortus* variants were, however, determined and eventually some were freeze-dried.

Figure 1 gives a schematic presentation of the most frequent findings in definitely positive and negative tests on serum.

Final results obtained after completing all tests are demonstrated in specimen reports shown in figure 2.

By means of these tests it has been found possible to diagnose brucellosis in many flocks from which only suspected sera as tested by the old routine had been received. (See Table 1).

A number of vaccinated sheep which had received identical treatment, showed great variation in their reactions. (See Tables 2 and 3).

A number of rams suffering from genital brucellosis or exposed to infection[12] have also been tested repeatedly. (See Table 4).

Results on a few reactors kept for investigation, and on contact animals placed with them are shown in Table 5.

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FIGURE 1.

BRUCELLOSIS

DIAGNOSTIC TESTS

TITRE :	5	10	20	40	80	160	320	640	
				TUBE AGGLUTINATION					
	—	+	+++	++++	+	—	—	—	= Pos. (Prozone)
	++++	++++	++++	+++	+	—	—	—	= Pos.
	++	—	—	—	—	—	—	—	= Neg.
				5% SALINE TUBE AGGLUTINATION					
	++++	+++	++	+	—	—	—	—	= Pos.
	+++	++++	+	—	—	—	—	—	= Pos.
	+	—	—	—	—	—	—	—	= Neg.
				BLOCKING ANTIBODY : AGGLUTINATION INHIBITION					
(	—	—	—	—	—	—	—	—	) No agglutination
	—	—	+	+	++++	++++	++++	++++	= Pos.
	++++	++++	++++	++++	++++	++++	++++	++++	= Neg.
				COOMBS ANTIGLOBULIN AGGLUTINATION					
(	++	+	—	—	—	—	—	—	) neg. agglutination
	++++	++++	++++	++++	++++	++++	+++	+	= Pos.
	+++	++	+	—	—	—	—	—	= Neg.
				COMPLEMENT FIXATION					
	•	++++	++++	++++	+++	+	—	—	= Pos.
	•	+	—	—	—	—	—	—	= Neg.
TITRE :	5	10	20	40	80	160	320	640	

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FIGURE 2.

SPECIMEN RESULT OF COMPARATIVE TESTS IN OVINE BRUCELLOSIS

— = negative at lowest dilution.
1 = reaction at lowest dilution (1: 2.5).

No.	Agglut.	5% Sal.	Block	Coombs	Cmpl. Fix.	Diagnosis
	Titre / Reading	Titre / Reading	Titre / Reading	Titre / Reading	Titre / Reading	
6	10 / 442 — — —	10 / 442 — — —	1 / 133333	5 / 441 — — —	— / 444444	Positive
8	10 / 442 — — —	10 / 432 — — —	— / 444444	10 / 432 — — —	160 / 4 + + + + +	Positive
15	1 / 31 — — — —	5 / 22 — — — —	80 / 1 + + + + 1	40 / 422221	160 / 4 + + + + 1	Positive
22M	— / — — — — — —	— / — — — — — —	1 / 133334	— / 11 — — — —	10 / 414444	Suspicious
39M	5 / 431 — — —	1 / 31 — — — —	1 / + 22222	80 / 222222	— / 444444	Positive
51M	— / — — — — — —	— / — — — — — —	5 / + 13333	1 / 21 — — — —	— / 444444	Suspicious
68	1 / 21 — — — —	1 / 41 — — — —	1 / 133333	5 / 321 — — —	— / 444444	Suspicious
85	— / 21 — — — —	1 / 2 — — — — —	— / 333333	5 / 221 — — —	— / 444444	Suspicious

TABLE 1.

Results of different serological tests in a number of flocks from which only suspected sera, as tested by the routine aglutination test, had been received.

1 = 1st tube only (1: 2—5); — = no reaction at lowest dilution; * = test not completed.

Area	Species	Number Tested	Reactions				
			Agglut.	5% Sal.	Block	Coombs	Cmpl. Fix.
Louis Trichardt	Sheep	87	7	5	2	*	*
		1	—	—	—	—	*
Brits	Goats	89	13	—	9	1	*
Zeerust	Goats	4	—	—	3	—	2
		10	4	5	4	1	*
Estcourt	Sheep	3	1	1	—	—	*
		8	8	2	3	6	2
		63	10	10	*	3	*
		60	15	15	*	—	*
Pretoria	Sheep	86	—	1	3	—	37
Riversdal	Sheep	81	25	25	1	—	*
		56	20	20	*	—	*
Graaff-Reinet	Sheep	16	1	11	2	—	*
Potchefstroom	Sheep	6	—	—	—	—	3
Upington	Sheep	146	5	5	14	19	3
		137	5	5	—	—	*
		264	9	9	19	10	*

TABLE 2.

Results of different Serological tests in a number of groups of vaccinated sheep (with microscopical tests in a few animals).

Group	Number Tested	Result: number positive			
		Agglut.	5% Sal.	Block	Coombs
Ewes: '58	27	16	20	3	5
J. '59	27	15	19	1	25
F. '59	27	3	16	1	7
M. '59	39	7	22	1	6
A. '59	39	4	20	13	7
Rams: '58	2	1	1	1	1
J. '59	2	1	1	1	1
F. '59	2	*	1	*	1
M. '59	2	1	1	1	*
A. '59	2	*	1	*	*
Rams: '58	6	6	6	—	—
J. '59	17	8	10	1	9
F. '59	17	6	10	2	2
M. '59	17	10	10	*	*
A. '59	17	11	13	2	*
Rams: '58					
J. '59	34	10	28	1	33
F. '59	34	8	33	10	31
M. '59	34	20	26	5	4
A. '59	31	18	31	*	4
Rams: F. '59	36	5	—	10	—
M. '59	36	5	9	1	1
A. '59	34	7	12	11	1

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TABLE 3.

Results of different serological tests on some individual sheep in vaccinated groups.

1 = 1st tube only (1 : 2–5); — = no reaction;
* = test not completed.

	Date	Titres				
		Agglut.	5% Sal.	Block	Coombs	Cmpl. Fix.
Ram	1958					
(No.	Jan. '59	20	10	5	320	*
1917)	Feb. '59	80	80	—	320	*
	March '59	1	40	10	—	*
	April '59	20	20	—	—	*
Ewe	1958	20	10	—	320	20
(No.	Jan. '59	20	10	—	320	*
3361)	Feb. '59	20	20	1	—	*
	March '59	10	10	—	—	*
	April '59	10	10	—	640	*
Ewe	1958	320	80	—	320	1280
(No.	Jan. '59	160	160	5	320	160
3366)	Feb. '59	80	160	—	160	80
	March '59	80	80	—	—	40
	April '59	40	80	1	2560	1280
Ewe	1958	40	—	20	320	—
(No.	Jan. '59	5	10	—	320	*
3384)	Feb. '59	5	10	—	5	*
	March '59	5	5	—	—	20
	April '59	1	1	—	20	*
Ewe	1958	40	10	—	*	*
(No.	Jan. '59	5	10	—	320	*
3410)	Feb. '59	5	5	—	1	*
	March '59	5	5	—	—	*
	April '59	5	5	—	—	*
Ewe	1958	5	5	—	—	*
(No.	Jan. '59	—	—	—	320	*
3979)	Feb. '59	1	5	—	5	*
	March '59	—	—	—	—	*
	April '59	1	1	5	—	*

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TABLE 4.

Results of microscopical and serological tests on 24 infected and in-contact rams suffering from or exposed to infectious infertility.

1 = 1st tube only (1 : 2–5); + = positive; ± = doubtful result; — = no reaction of lowest dilution; * = test not completed.

Ram	Year	Semen		Brucella Reactions				
		Miscroscopically Positive	Cultures of Brucella	Agglut.	5% Sal.	Block	Coombs	Compl. Fix.
1	58	30%	—	1	1	5	—	—
	59	*	*	10	10	—	—	40
2	59	34%	+	5	5	—	—	20
3	59	*	*	1	1	—	—	20
4	58	58%	—	1	1	—	5	10
	59	*	*	1	1	1	—	10
5	59	55%	*	1	1	—	—	—
6	58	63%	+	5	5	—	20	30
	59	*	—	5	5	—	—	—
7	58	58%	+	1	1	5	10	10
	59	*	—	5	5	—	—	10
8	59	*	*	10	10	2	2	20
9	58	52%	+	1	5	5	5	—
	59	*	—	1	5	—	—	*
10	59	—	*	1	1	—	20	*
11	59	*	*	1	1	—	—	40
12	59	*	—	1	5	—	1	10
13	59	*	—	1	1	—	1	10
14	59	40%	*	1	1	—	5	—
15	59	(16%)	(+)	5	5	—	—	—
16	59	±	*	1	1	10	40	—
17	58	—	*	—	—	—	1	20
	59	*	*	—	—	5	10	20
18	59	+	*	1	—	1	10	—
19	58	±	*					
	59	*	*	—	—	20	1	10
20	58	+	*	1	—	—	—	40
	59	*	—	—	—	5	1	*
21	59	12%	*	—	1	—	—	—
22	59	75%	+	1	1	—	10	—
23	58	75%	+	—	—	—	—	10
	59	*	+	1	1	—	—	*
24	59	—	—	10	10	10	—	10

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TABLE 5.

Results of serological tests on some sheep and goats either submitted as specimens or kept in contact.

Description	Date	Reactions					Remarks
		Agglut.	5% Sal.	Block	Coombs	Compl. Fix.	
Karakul ewe	1958	1	1	—	*	*	Milk
4551	Jan.	10	10	—	320	*	Culture
Marienthal	Feb.	10	10	—	5	*	positive
(aborted)	March	10	10	—	—	*	1958
	April	10	10	—	320	*	
	May	10	10	—	—	40	
Karakul ram	1958	5	10	—	160	*	Semen
4751	Jan.	10	10	—	320	*	Culture
Marienthal	Feb.	20	10	—	320	*	positive
	March	10	20	1	320	*	1958
	April	20	20	5	160	*	
	May	20	5	1	—	—	
Merino ewe	1958	—	1	—	*	*	
3915	Jan.	—	—	—	320	*	
(Contact)	Feb.	5	10	—	1	*	
	March	—	—	—	—	*	
	April	10	10	—	20	*	
	May	10	1	5	—	10	
Jenny goat	1958	—	—	*	*	*	Foetus
6965	Jan.	—	—	—	320	*	positive
Brits	Feb.	1	1	—	—	*	Biological
	March	1	1	1	—	*	test
	April	20	20	—	—	*	1958
	May	10	10	—	—	320	
Billy goat	1958	—	—	*	*	*	
4436	Jan.	—	—	—	320	*	
(Contact)	Feb.	1	5	—	—	*	
	March	1	1	—	—	*	
	April	5	5	—	—	*	
	May	10	10	—	—	40	
Jenny goat	1958	—	—	—	*	*	
4623	Jan.	—	—	—	320	*	
(Contact)	Feb.	—	—	—	—	*	
	March	1	1	—	—	*	
	April	5	5	—	—	*	
	May	80	—	10	—	80	

Discussion

At present our diagnostic means permit little more than a flock diagnosis. A direct clinical examination is liable to reveal only chronic lesions in a flock unless one is fortunate to obtain a number of fresh foetusses with afterbirths. A direct microscopical examination of semen smears is the most reliable method of making a diagnosis. The technique of semen collection of van Rensburg and de Vos has permitted the greatest advance in this field by facilitating semen collection.

The position of *Brucella* organisms in the epithelial, lymphoid and polymorphonuclear cells, in differentially stained groups is fortunately highly diagnostic of the genital form of *Brucella* infection. Doubtful cases, with only a few suspicious organisms present, are, however, very common. Semen samples from the same ram infected for a period of some months may be positive one week and negative the next. Chronic cases frequently show *Corynebacterium, Streptococcus, Proteus,* or *Pseudomonas* also.

For control of the disease in a flock an indirect method of diagnosis, by serological tests, is the most attractive routine procedure. In sheep this gives notoriously poor results. Serologically sheep are even less reliable than human beings, pigs and goats. The genital form of brucellosis is also the least active antibody stimulating infection. Being located in the epithelium, it produces a state comparable to the so-called " Silent Entry " of brucellosis in the cow when infected per vaginam; and to the vesicular brucellosis of the bull.

In addition, variation " in vivo " is comparatively rapid in the sheep which has no " Smooth Selecting Factor " such as exists in the bovine circulation.

The present results show that the Complement Fixation test, the Coombs antiglobulin test, the Blocking antibody test and the 5% saline agglutination test are frequently superior to the routine agglutination test with ordinary antigens. Not enough is known of the circumstances wherein each of these tests will be of greatest use. It appears that recent infections show up best in the 5% Saline and Blocking tests and that in chronic infection, Complement Fixation is more reliable. The Coombs reaction seems to be strongest in the middle period, about 3 to 6 months after infection. In the present state of our knowledge no serological test can be singled out to prove freedom from infection or presence of infection in a single animal.

However, as a flock test the 5% saline agglutination technique is recommended. Additional Coombs tests and Complement Fixation tests on selected individuals in suspected flocks are justifiable.

Diagnosis of semen infection in suspected rams by microscopical examination can only be supported but not replaced by serological tests. Both methods require to be repeated several times when proof of freedom from infection is desired.

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Investigation of the value of different antigens for routine use appears to be indicated. A skin test for certification may also be tried. In special circumstances, the opsonocytophagic index might be considered.

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DISCUSSION

DR. VAN RENSBURG (Opener) congratulated the speaker on his paper but stated that it showed that serological tests were of limited value in the diagnosis of Brucellosis in sheep and goats. He advocated the more reliable method of semen examination. Even then the results were frequently negative in old standing cases but the organism could almost always be found in infections of up to twelve months' standing. In a group of animals experimentally infected 18 months ago it had been the exception not to find the organisms. None of these rams yet showed any clinical lesions. In another group of six rams infected 21 months ago, only three had developed clinical lesions to date. When examining a flock for the presence or absence of infection, all rams should be examined both clinically and by semen examination. The infection can be transmitted by contact as well as by coitus. Young rams of 5 to 6 months old, kept in contact with infected adults, frequently showed infection. Dr. van Rensburg reminded the meeting that the techniques used had been published and called on all veterinarians in sheep raising areas to equip themselves to carry out this essential service.

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THE pH. OF MEAT

L. W. VAN DEN HEEVER
Germiston

Introduction

Meat is defined in the Food, Drugs and Disinfectants Act as the sound and wholesome flesh of food animals. In fact such flesh is the striated skeletal muscles of cattle, sheep, pigs and also horses, rabbits, poultry and game, and it is closely associated in its normal situation with connective tissue, fat, bone, cartilage, tendons, blood, etc.

Because of its importance both as the functional unit of locomotion and as a most important food, striated muscle has been given considerable attention in regard to its biochemical, physiological and pathological aspects.

Striated muscle consists of 75% water and 25% solids, protein making up 4/5th of the solids. These muscle proteins are made up as follows:—

Myosin	67%
Globulin	21%
Myogen	10%
Myalbumen	1%
Myohaemoglobin, up to	1%

Myosin is therefor the major protein of the myofibril, and it plays a correspondingly important role in myocontraction. It possesses certain enzymotic properties which are intimately concerned with muscle contractility, this being the manifestation of the utilisation of the energy ultimately supplied mainly by glycogen.

Glycogen may be present in muscle tissue in quantities up to 1.5%, and is replenished from blood glucose drawn from liver reservoirs when the amount required for conversion to muscular energy is depleted. Globulin X, myogen and myalbumen are apparently the proteins which make up the sarcoplasm, whilst the myohaemoglobin plays an important part in the transfer of oxygen to the muscle. It is an ironporphyrin protein similar to blood haemoglobin, and by "loose linkage" it takes up 0_2 and forms oxy-myohaemoglobin which again readily releases 0_2 in the muscle, depending on the demand as manifested by a reduced oxygen-tension.

Millikan, as quoted by Miller(1) described oxy-myohaemoglobin as a "halfway station" between blood and the enzymes (catalase, peroxidase, oxidase) and cytochromes which play an important role in the physiology of contraction.

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The remaining one-fifth of the muscle solids consist of *nitrogenous extractives* (such as creatine, purine bases, phosphocreatine, carnosine, anserine, adenylic acid, uric acid, inosinic acid and carnitine), *non-nitrigenous substances* (such as glycogen, lactic acid, inositol, hexosephosphate, etc.), *enzymes, vitamins* (B and ascorbic acid) and *inorganic Salts* (the cations being P, Ca, Na, and Mg and the anions, phosphates, chlorides and sulphates). The inorganic phosphates are made up mainly by adenosine triphosphate (A.T.P.), adenosine diphosphate (A.D.P.) and phosphocreatine.

The Hydrogen-ion Concentration of Muscle

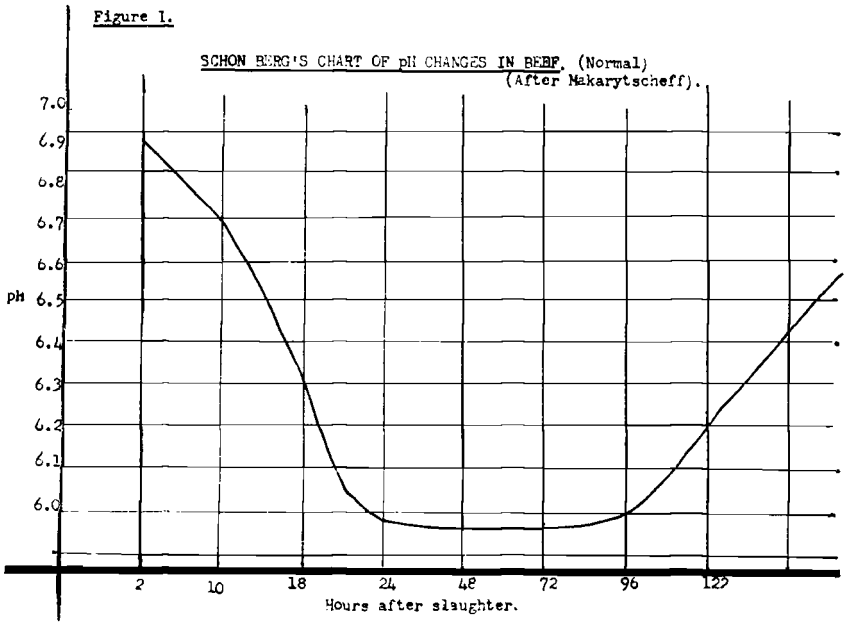
Depending on the condition of the animal and the state of activity of the muscles, the living muscle has a value of ± 7.2 (Jepsen[2]) whereas at the time of death the pH of normal muscle is given as 7.4 (Miller[1]). In living muscle the lactic acid formed from glycogen in anoxia is rarely present in amounts exceeding 0.5% and it is continually being removed by tissue fluids and buffered by blood and lymph, some of it being resynthesized to glycogen (Postma[3]). The permanent anoxia of death causes an increase in the production of lactic acid from whatever glycogen may be available and the acid may accumulate in quantities up to 1%, leading to a lowering of the pH to an " ultimate " level after 24-48 hours. The degree of such acidification depends on the glycogen available in the muscle at the time of death, and any event rarely extends below 5.4, at which pH level the process is self-limited by inactivation of the enzymes.

This development of acidity has been the subject of considerable study ([3], [7], [15] and numerous other authors) and it has an important effect on the meat of our food animals. It has always been linked with the development of rigor mortis, and most standard works offer an explanation of this phenomenon which is tied up with the developed acidity. It has, however, been demonstrated that rigor mortis can and does occur in " alkaline " meat (Miller) while Mirshky & Anson([4]) have found that whereas certain changes occur in the disulfide and sulfhydryl groups of myosin in normal natural rigor, no such changes were observed when myosin was subjected to heat or acid. The chemical changes found in myosin with natural rigor were of the same type as those encountered as a result of denaturation of myosin by dehydration. From this it would appear that the development of good rigor in a carcase is not necessarily an indication of adequate acid development whereas it has been clearly shown that rigor mortis is closely associated with the disappearance of adenosine-triphosphate from the meat, probably by enzyme action (Miller).

The development of acidity in a carcase also has other effects on the meat. " Acid " meat is light cutting, of open structure, easily releases its flavour on cooking, readily absorbs sugar, salt, brine and smoke in curing processes and forms an unfavourable medium for

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Figure 1.



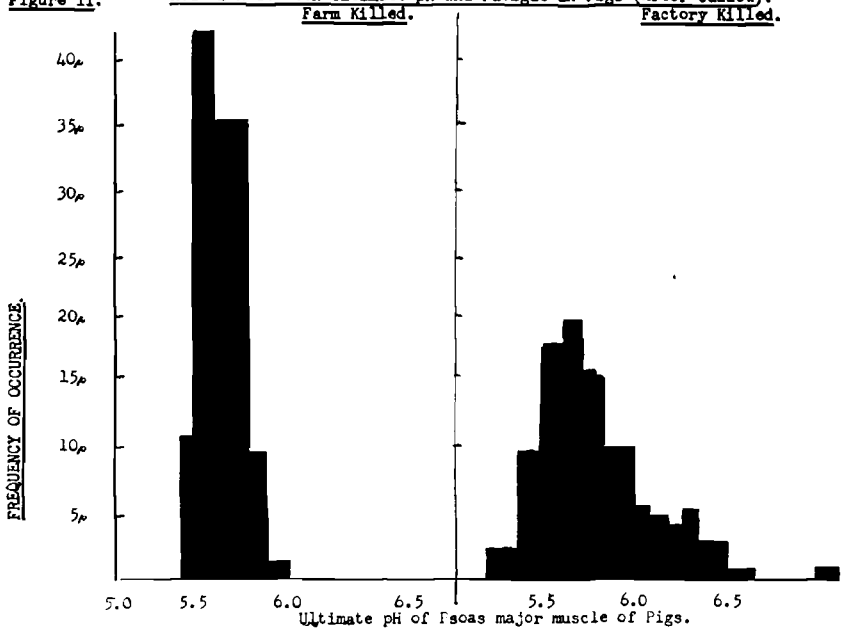
the development and growth of bacteria. Meat with inadequate acid content (" Alkaline " meat) has the reverse properties.

Schönberg(5) has charted the changes in the pH of meat against the time of storage (See figure 1) and has found that after dropping to its ultimate it gradually rises again over several days. This rise in pH is also associated with and speeded up by bacterial growth (Ingram[8]). Schönberg considers a pH value of 6.5 as the critical point, the determination being done 24-48 hours after slaughter. He states that meat from healthy well nourished and rested animals attains a pH 5.2—6.0 within 24 hours, whereas diseased, exhausted or otherwise abnormal animals produce meat which never reaches a reasonable acidity, immaterial of how long storage is undertaken.

Schoon & Ooms(7) and Lewis(8) stress that the choice of the particular muscle on which pH determinations are made is important in as much as variations up to 0.4 and more in pH values have been recorded in various muscles from the same carcases. They also showed that species variations were considerable. Postma(3) showed that pigs were especially subject to fatigue and that the absence of adequate glycogen reserves in such cases invariably led to the meat having a high pH. Schoon & Ooms(7) also link the well-known and characteristic rapid deterioration of pork, as well as bone taint, with poor acidity of carcases from fatigued pigs. Callow(9) presents a graph (Fig. 2) in which he indicates the pH values of " Farm killed " pork (where there is a minimum of fatigue due to transport, excitement and starvation) in contrast to that obtained from " factory killed " pigs, and the differences are

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Figure II. Relation between Ultimate pH and Fatigue in Pigs (After Callow).



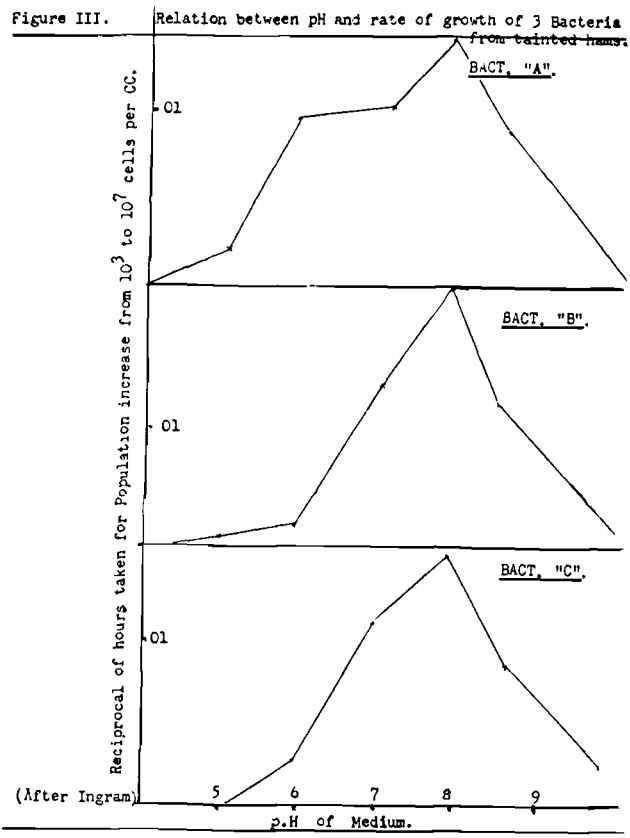
significant when viewed in the light of Ingram's graph (Fig. 3) indicating the growth of bacteria isolated from bone-tainted hams under various pH conditions.

Thornton[13] states that 5% of the hams from pigs starved and rested for one day developed bone taint, whereas none of the hams from pigs rested and fed for three days prior to slaughter showed this condition. Feeding pigs before transport is not considered as valuable as doing so after arrival at the abattoir during the resting period.

Hedrick[14] has indicated some of the known relationships between " dark-cutting " (high pH) beef and the now well-known stress syndrome exhibited by the animal body after exposure to the various abnormal conditions during marketing and the pre-slaughter period, viz. emotional excitement, exposure to adverse weather, trauma, starvation, thirst and muscular fatigue. He has also made some investigations into the period required for cattle to recover from stress if given feed and water, and has indicated the important part played by the glucocorticoids in restoring normal glycogen values to the liver and muscles. In his opinion the rate of muscle glycogen replenishment is a slower process than the rate of depletion under severe stress. He also mentions the positive use of tranquilizers to prevent the great loss of fluid during such stress periods and to prevent " dark cutting " beef at the abattoir.

Jepsen[2] considers that pH readings above 6.5 indicate meat of a poor keeping quality, and the judgment of such meat should

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be conditioned by such a reading, i.e. further bacteriological examination, conditional passing or complete condemnation. He also emphasizes that low pH meat presents conditions not conducive to bacterial growth. He states that an abnormally high pH reading is frequently associated with peritonitis, enteritis, dystocia and its complications, arthritis and cases of uncertain diagnoses suspected of sepsis. He found 21 cases out of 99 abnormally high pH readings to coincide with specific or high grade non-specific bacterial infections. It is customary to combine pH determinations with bacteriological procedures in most European countries.

The pH thereof is also known to have a considerable effect on the conditioning of meat (Greatorex[12]) and the "ripening" process which is so important in obtaining the full flavour and tenderness of meat.

Methods of pH Determination

Various methods have been used to determine the pH of meat. The Michaelis Walpole method consists of a colorimetric determination, using an indicator and an extract of the meat.

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Schönberg developed the use of a short range pH indicator called Nitrazine Yellow, at first on meat extracts and later in a 1.10,000 dilution on actual pieces of meat. This method is reasonably accurate and inexpensive, whilst at the same time being simple to apply. Electrometric methods of pH determinations have always been regarded as the most accurate but the equipment is expensive. Diemont([11]) has done considerable work in this regard, also in using the newer glass electrodes. He emphasizes the fragility of the earlier types of glass electrodes, and notes that contact with pieces of tendon ("peesplaatjes") in the meat under test can give false results. Some of the newer Beckman-type electrodes are more sturdy and seem admirably suitable for such work.

Personal Observations

pH determinations of suspect carcases have been undertaken on a limited scale at the Germiston Abattoir. The muscle used has been aportion of the M. triceps brachii, for the following reasons:—

(a) It is readily accessible in the hanging position of the side of beef.
(b) It is already cut in the process of routine examination for cysticercosis in cattle and pigs.
(c) It does not lead to mutilation of the carcase so that suspect carcases may still be passed and be found acceptable to the trade.

The electrometric determinationss were made by a portable battery operated Beckman "180" pH meter equipped with glass fibre type reference electrode filled with a saturated solution of KCl and AgCl.

The Nitrazine Yellow method involves the use of walnut size pieces of meat as free of fatty tissue, blood, etc., as possible, white porcelain dishes and a 1:10,000 solution of the indicator in distilled water. This solution is clear yellowish brown in commercial grade distilled water due to minor impurities (e.g. CO_2) and has an intrinsic pH of about 6.2. The pieces of meat are covered with the solution and pressed and turned and squeezed, left for 5 minutes and then removed to allow of judgment of the developed colour. Schönberg himself gives the following table from which pH estimations may be made:—

Brownish Violet to Violet Blue — up to	7.0 and over
Olive greenish brown, slightly cloudy	6.5
Brownish Yellow to Yellowish Brown	6.3—4
Clear Yellowish Brown	6.3—6.2
Clear Yellow	6.0 and lower.

Firstly, some attempt was made to establish a "normal" value for meat under local conditions and for this purpose meat from carcases passed as fit for consumption or condemned for cysticercosis but otherwise normal, was used, such meat being readily available. Using the Nitrazine Yellow (1:10,000) method and electrometric method, some carcases were examined and in 95%

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the pH after 24 hours' storage at hanging hall temperature was found to be below 6.3.

Subsequently, some attention was given to carcases which were likely to show inadequate acid development, e.g. carcases detained for extensive bruising, gangrene, emaciation, improper bleeding, fever, emergency slaughtered and moribund carcases. The results are given in Table 1 and it will be seen that the percentage of carcases showing inadequate acidity, taking a pH of 6.5 as the critical value, is relatively high in comparison with the "normal" carcases.

TABLE I

pH DETERMINATION ON MEAT OF CATTLE.

Kept @ Room Temperature for 24 hours after slaughter.

A. "NORMAL" CARCASES (passed as fit or condemned for C. bovis):

Carcases with meat of pH over 6.5	1	(.8%)
,, ,, ,, ,, 6.3 — 6.5	5	(4%)
,, ,, ,, ,, 6.0 — 6.3	18	(15%)
,, ,, ,, ,, below 6.0	97	(80%)
Total number of carcases examined	121	

B. CONDEMNED OR DETAINED CARCASES:

P.M. DIAGNOSIS.	Carcases	pH6.5+	pH6.3-6.5	pH6.2 or below
Emaciation	28	11	8	9
Emac. and Oedema	8	1	2	5
Emac. and Bruised	3	—	—	3
Bruised (slightly)	11	5	3	3
Bruised (Extensively)	14	6	4	4
Gangrene	3	1	2	—
Gangrene and Intramuscular Emphys.	1	—	—	1
Peritonitis and Pleuritis	7	—	2	5
Septic Pericarditis	1	—	—	1
Septic Mastitis	1	—	—	1
Septic Metritis	3	—	2	1
Tuberculosis (Gen.)	1	—	—	1
Moribund	4	3	—	1
Tuberculosis & Ext. Bruised	1	1	—	—
Oedema	3	2	—	1
Gangrenous Pneumonia	1	—	—	1
Pyaemia & Enteritis	4	—	—	1
Pyaemia & Emaciation	1	—	—	1
Peritonitis & Oment. Abscess	1	—	1	—
Ext. Bruise & Comm. Fract. of Radius	1	1	—	—
TOTALS	94	31 (32%)	24 (25.5%)	39 (42.5%)

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Discussion

It would appear that a pH determination of suspect carcases would be of some considerable value in deciding the borderline cases where organoleptic observations are insufficient to enable one, with some sound scientific basis, to pass judgment on such carcases. It should be clearly understood, however, that a pH determination alone, without ordinary meat inspection methods and possibly also without bacteriological investigations, is no satisfactory criterion for judgment regarding the fitness or otherwise of meat. It is an aid, and its use can put meat inspection on a sounder scientific basis, whilst at the same time augmenting organoleptic methods and drawing attention to minor deviations from the "normal" which might otherwise be overlooked.

The Danish Meat Inspection Rules require laboratory examination of all carcases under the following headings:—

Emergency slaughter, fever and acute disease, endocarditis verrucosa, certain forms of pneumonia, catarrhal gastro-enteritis in adults, localised peritonitis, pyometra and chronic or subacute endometritis, retained foetal membranes, dystocia and its complications, prolapsed or ruptured uterus, septic or purulent mastitis, infectious tendovaginitis and arthritis, wound infections, osteomyelitis and burns.

Our own Meat Inspection Regulations are sorely antiquated in respect of modern methods of meat inspection in as much as they make no specific provisions for detention of suspect carcases pending biochemical or bacteriological investigations.

Conclusion

The development of scientific meat inspection procedures overseas and the stress which is laid upon such methods would indicate that revision of our Meat Inspection Regulations is long overdue. In the meantime, application of such laboratory methods as are of practical value should not be neglected, and pH determinations appear to be of some value in this regard. Further work on pH determinations under South African conditions requires to be done. The antemortem feeding and resting of animals, especially pigs, appears to be essential if carcases of good keeping quality are to be produced, and deserves more attention than that at present given at South African Abattoirs.

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DISCUSSION

DR. MEARA (Opener): There is one point that Dr. van den Heever omitted under pressure of time and that is that the regulations do not allow us to hold over carcases for the period required to determine the ultimate pH, i.e. 24 hours. This is a grave defect which I sincerely hope will be remedied.

Dr. Meara then went on to speak on the roneod notes which he had distributed and which are published below.

The development of scientific methods of meat inspection in this country is likely to be accelerated by Dr. van der Heever's interesting paper on the pH of meat. His investigations on pH abnormality in the meat of casualty animals are particularly interesting. As they leave little scope for elaboration it seems opportune now to discuss those factors affecting the ultimate pH of muscle in "normal animals," in order to supplement his observations. A series of graphs and tables has been prepared in order to enable a wide field to be reviewed in the short while available.

Significance of pH:

After death the glycogen within the muscles slowly becomes broken down to form Lactic acid and the quantity of lactic acid produced in this way determines the acidity of the flesh.[1] The importance of this process derives from three main headings:

(1) *Keeping Quality*: An acidification of the flesh subsequent to slaughter is important in improving the keeping quality of the carcase. The lactic acid which is formed creates a medium inimical to bacterial growth and retards the growth of putrefactive organisms, which generally prefer an alkaline or almost neutral medium in which to develop.[2] As all the superficial tissues of a carcase are normally contaminated during slaughtering and dressing operations, with a wide variety of bacteria capable of multiplying rapidly under

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suitable conditions, the degree of acidity of the tissue is an important factor prolonging the keeping quality of the meat.

A depletion of the glycogen reserves in the live animal, such as may occur during handling and transport on the way to slaughter, disturbs the production of a normal amount of lactic acid. In order to replenish the glycogen depleted by exhaustion and fasting, and for other reasons also, it is generally recommended to rest slaughter stock adequately on arrival, and to feed them suitably.

(2) *Diseased Meat*: In the case of an animal suffering from fever or septicaemia no appreciable acidification of the meat may occur after death, and various other diseased conditions also upset the normal formation of lactic acid.[3] As shown by Dr. van der Heever, pH determination may be a useful aid in assessing the suitability of meat from diseased or casualty slaughter stock. Where facilities permit certain other biochemical tests and bacteriological methods may be performed to enhance pH investigation,[4] but these laboratory procedures entail holding a suspected carcase and viscera for a period until the investigations are completed.

The fact already mentioned by Dr. van der Heever deserves emphasis that the South African regulations do not empower authorities to hold meat pending laboratory examination. Until these regulations are revised the development of improved methods of meat inspection is likely to be impeded.

(3) *Decomposed Meat*: When the ultimate pH is achieved it is maintained for a period depending on factors like cooling procedure, the conditions of storage and the degree of bacterial contamination. With the onset of autolysis and bacterial growth the pH of the meat rises slowly and usually at about pH 6.4—6.5 signs of decomposition become evident.[3] As putrefaction develops further with associated changes of consistence and odour the pH continues to rise.

Measurement of pH:

Measurement of pH is usually only performed 24 hours post-mortem when the pH of muscle from physiologically normally animals lies between the range 5.6—6.3, or less.

(1) *The Colorimetric Test* is easily performed with a minimum of apparatus. Nitrazine yellow is usually employed as the indicator and gives an immediate result over the pH range from 6—7. Although the simple nitrazine yellow method is relatively insensitive to delicate measurement it has the advantage of presenting a clear-cut change of colour in the zone pH 6.4 to 6.5, which is the critical level in respect of meat.

The colour variations are as follows:

pH 6.0 yellow
6.2 yellow brown
6.3 yellow brown
6.4 yellow greenish
6.5 olive green, slightly turbid
6.6 wine red
6.7 red violet
6.8 blue violet.

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At pH around 5.8 or 6.0 the solution remains yellow and clear. At pH 6.4—6.5, the critical value for judging meat, the solution becomes yellow green to olive green and slightly turbid. As the pH approaches the neutral point of 7.0 the nitrazine yellow solution assumes a blue violet colour. If the indicator remains yellowish or yellowish brown the meat is generally satisfactory, but if it becomes olive-green and turbid the meat is abnormal.

A small piece of muscle weighing about 2 gm. is placed in a shallow porcelain dish and covered with a 1/10,000 solution of nitrozen yellow. The meat is pressed and squeezed to permit of tissue fluid mixing with the indicator and the colour is noted after the lapse of a minute or two.

(2) *The electrometric method* is a precise means of measuring the pH of meat. One method of performing the test is to macerate about 1 gm. of muscle in 10 ml. of 0.01 M sodium iodoacetate solution in a homogenizer and measure the pH by glass electrode.[5]

Whichever means of measurement are employed it is necessary to use a rigidly standard technique for determining pH, particularly with regard to the part of the animal and the muscle from which the sample is taken. As freezing inhibits the formation of lactic acid, the carcase should be stored at or above the temperature of chilling from the time of slaughter until testing. Muscles with as little connective tissue as possible should be selected, and the sample itself should be as free as possible from connective tissue, fat and blood.

Certain muscles respond markedly to pH effects compared with other muscles and care must be exercised in selecting a sample muscle. In the past M. Psoas has often been used as an index with useful results, but its total weight is small and its biochemical characteristics differ from other muscles forming a greater proportion of the meat from a carcase. Selection of a sample muscle is also governed by the need not to disfigure the carcase and reduce its sales value, but Dr. van der Heever has shown how the routine incision into the triceps brachii for cysticercosis examination may be used in order to derive a ready-made specimen for pH determination.

Variability of PH:

There are many factors which contribute to variability of pH in normal flesh.

(1) *Variation within the muscle*: Mean ultimate pH values may be different at different points within a muscle.

TABLE 1

Muscle.	pH Site No. 1	Site No. 2	Site No. 3
Semitendinosus	6.22	6.02	5.76
Semimembranosus	5.99	5.70	5.50
Rectus Femoris	6.19	5.91	5.80

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(2) *Variation between Muscles*: Different muscles tend to develop characteristic amounts of lactic acid and thus have different ultimate pH values.

(i) *Pork*: In pigs the ultimate pH usually lies between values of 5.3 and 6.2, but the ultimate pH is considerably more variable than in the ox or horse. Since muscle glycogen reserves tend to be relatively low and the pig is sensitive to the effects of exercise and fasting the ultimate pH values are frequently high. Unlike the horse and ox a high ultimate pH value signifies absence of muscle glycogen.

TABLE 2

"Normal" pH values for pork (Lawrie[7])

Muscle	pH 1 Hour post-mortem	pH 24 Hours' post-mortem
Psoas	6.20 ± .15	5.75 ± .18
Diaphragm	6.36 ± .12	5.99 ± .08
Long dorsi	6.71 ± .04	5.45 ± .02

(ii) *Beef*: The pattern of the change in pH normally occurring after slaughter was discussed by Moran and Bate-Smith.[8]

A large-scale investigation[9] established pH values for 50 beef muscles varying from the lowest 5.4 for M. semimembranosus to the highest 6.0 for m. sternocephalicus and long. costarum. The mean was 5.7. Howard and Lawrie (1956)[5] investigated ten bovine muscles and obtained a mean ultimate pH of 5.64 (583 samples).

An ultimate pH below 5.35—5.40 is infrequent. Considerable reserves of glycogen generally remain in beef muscle, even when the ultimate pH has been attained after glycolysis ceases.

(iii) *Mutton*: Marsh & Thompson (1958)[10] found that 83 per cent. of ultimate pH values of the long. dorsi in 51 New Zealand lambs lay between 5.4 and 5.8. However, data from Australian sheep almost invariably exceeded pH 5.7.

Lawrie (1959)[7] reported mean ultimate pH values in long. dorsi of two groups of 140 lambs and sheep in the United Kingdom as 5.57 ± .02 and 5.79 ± .02 respectively.

(iv) *Horse flesh*: Typical mean pH values for normal draught horses were reported as follows[7]:

TABLE 3

	pH 1 hour post-mortem	pH 24 hours' post-mortem
Heart	6.00 ± .03	5.81 ± .05
Psoas	6.77 ± .04	5.85 ± .06
Diaphragm	6.97 ± .05	5.91 ± .04
Long. dorsi	6.87 ± .02	5.56 ± .03

(3) *Effect of the degree of resting*: In routine abattoir practice one cannot ensure that all animals will be adequately rested in

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uniform manner. This factor makes for variability in the acidity of the flesh of "normal" animals.

(4) *Effect of condition of the animal*: Meat from lean and poor quality animals may have a high pH compared with good quality meat, as a result of low glycogen reserves in the muscular tissues of low-grade animals.[5]

TABLE 4

Effect of quality of meat on pH.

Muscle.	pH of Australian First Quality Export Grade Beef.	pH Canner Beef.
Sirloin	5.62	5.90
	5.52	5.62
	5.58	5.59
	5.47 Mean 5.55	5.72 Mean 5.71
Topside	5.52	5.73
	5.53	5.63
	5.55	5.70
	5.47 Mean 5.52	5.73 Mean 5.70
Rump	5.51	5.78
	5.54	5.64
	5.54	5.89
	5.45 Mean 5.51	5.67 Mean 5.75
Mean for All Muscles	5.53	5.72

(5) *Effect of temperaments* A chronic depletion of the muscle glycogen may occur as the result of an indeterminate excitability in temperamental animals subjected to conditions of unfamiliar handling and treatment. As a result of the state of natural excitability and associated muscle tension glycogen reserves appear to become depleted, and an abnormally high pH may ccur even in the well-fed animal well rested before slaughter.[5]

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TABLE 5

Mean pH of "normal" muscle compared with excitable muscle.

	Biceps Fem.	Semimem.	Adductor	Psoas		Gluteus Med.	Long Dorsi			Biceps Brachii
				Site 1	Site 2		Site 1	Site 2	Site 3	
Control Cattle (15)	5.51	5.49	5.61	5.57	5.55	5.52	5.49	5.49	5.55	5.67
Excitable Control	5.69	5.62	5.80	5.80	6.39	5.53	6.38	6.10	6.15	6.84

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(6) *Species Differences.*

(a) *Pig*: In the pig the glycogen reserves tend to be relatively low and become easily depleted. The psoas muscle becomes readily fatigued and its ultimate pH serves as a useful measure of exhaustion.[12], [13].

TABLE 6

Effect of treatment of pig on pH of psoas.

No. of Pigs.	Treatment.	Average pH of Psoas.
20	Killed on farm	5.60
20	Moved to factory and rested 24 hours	5.91
20	Moved to factory and rested 48 hours.	5.78
20	Moved to factory and rested 72 hours	5.50

Measurable fatigue ensues after walking even a quarter of a mile, requiring subsequent overnight resting and feeding to restore normality. A greater degree of fatigue such as that caused by passing pigs to collecting and distributing centres may require three days' resting and feeding before the glycogen reserves are restored.[14] Even the speed of the transporting lorry taking pigs from farm to factory can be related to fatigue, for on foggy days the reduced speed induced less fatigue than on fine days when travel was more speedy.[5]

TABLE 7

Travel Fatigue in Pigs.

Journey.	Average pH of Psoas.
Lorry, 40 miles. Fine Weather	5.96 5.94 6.06 6.05
Lorry, 40 miles, Fog	5.84 5.70

The different muscles of the pig also show considerably more variability in ultimate pH than in the ox and horse.

(b) *Ox*: Cattle react differently to fatigue and starvation, and their need for rest and feeding pre-slaughter is much less important than with pigs.

It was originally assumed that beef muscle would respond to ante-mortem treatment similarly to the other species studied such

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as the pig, rabbit, rat. The methods of fasting or exercise which effectively reduced the muscle glycogen reserves before slaughter in these species are most difficult to apply in the case of steers.

By normal means it is difficult to reduce the glycogen in the muscular tissues of beef animals and produce meat with a high pH. Neither fasting nor enforced exercise deplete the muscle glycogen reserves of beef steers sufficiently to raise the ultimate pH above normal values.[5]

(i) *Exercise*: Steers were hard driven by a mounted stockman for ½ an hour, and for 1½ hours, immediately before slaughter *until their gait became unsteady*, yet the muscles generally showed a normal ultimate pH after slaughter.

TABLE 8

Effect of Exercise on pH of Beef.

Period of Exercise.	Biceps.	Psoas.	Long Dorsi.
½ Hour	5.79	6.33 6.20 Mean 6.27	5.72 5.95 Mean 5.95
½ Hour	5.70	5.86 5.72 Mean 5.79	5.45 5.94 5.65 Mean 5.68
1½ Hours	5.78	5.79 5.70 Mean 5.75	5.60 5.72 5.62 Mean 5.65
1½ Hours	5.53	55.57 55.57 Mean 5.57	5.56 5.52 5.51 Mean 5.53

(ii) *Fasting*: Contrary to the expectation that a period of inadequate feeding would lower the glycogen reserves and result in a high pH of muscle steers were fasted for periods of 7, 14 and 28 days and showed a normal pH subsequent to slaughter.[16]

TABLE 9

Glycogen concentrations and ultimate pH in Psoas and Long Dorsi muscles of steers after fasting. Lawrie (1958).

	Long Dorsi.		Psoas.	
	Glycogen	Ultimate pH	Glycogen	Ultimate pH
Controls	1126 ± 98	5.50 ± 0.04	1082 ± 109	5.53 ± 0.07
Fasted 7 days	753	5.46	696	5.48
Fasted 14 days	597	5.73	717	5.55
Fasted 28 days	768	5.44	809	5.47

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The muscles of well-fed steers might have an initial glycogen content of about 1500 mgm/ gm. After 1 week's fasting the glycogen content of beef psoas and long dorsi fell by only about 300 mgm/100 gm. below normal. After fasting of 4 weeks' duration the glycogen reserves were not lowered to any greater degree, and this is attributed to the considerable proportion of glycogen in beef muscle as well as other biochemical factors peculiar to ruminant muscle. Howard and Lawrie[5] suggest that some carbohydrate-sparing mechanism operates in beef muscles during stress. Possibly ruminant muscle may derive considerable energy from the oxidation of fat and is less dependent on glycogen reserves for energy production than in non-ruminants.

(iii) *Exercise plus fastig*: A combination of stresses by exercise and fasting more severe than normally encountered depleted muscle glycogen reserves sufficiently to raise the ultimate pH above normal values. Steers were driven 150 miles on foot to railhead, and then trucked 625 miles without a rest period or feed, and then forcibly exercised for 1½ hours after arrival immediately before slaughter.[5]

TABLE 10

Effect of severe exercise and fasting on pH of Beef.

	M. Biceps	M. Psoas	M. Long. Dorsi.
150 miles overland + 625 miles by rail + 1½ hours' exercise	6.38	6.40 6.54 Mean 6.47	5.73 6.69 5.93 Mean 6.12
150 miles overland + 625 miles by rail 14 Day's Rest	6.70	5.56 5.50 Mean 5.53	5.43 5.34 5.67 Mean 5.48

(7) *Complicating Factors*:

The simpler concepts initially conceived regarding pH are gradually being shown to be too elementary and complicating factors are being uncovered.

(i) The assumption that a high ultimate pH only occurs if the glycogen reserve has been depleted is incorrect as post-mortem glycolysis may stop in various muscles at different pH levels before all the glycogen has been changed to lactic acid.[11]

TABLE 11

Horse Muscle.	Ultimate pH	Residual glycogen mgm/100 gm. muscle.
Long dorsi	5.56 ± .03	1411 ± 228
Psoas	5.35 ± .06	606 ± 143
Diaphragm	5.91 ± .04	1109 ± 116

A similar position prevails with respect to beef muscle.[5]

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(ii) Alternative means of breakdown of glycogen may also occur by way of dextrins and maltose to phosphate and glucose.[17]

(iii) *Eh.* Recently it has been stated that the presence of absence of oxygen in the flesh (oxidation-reduction potential or Eh) is at least as important as pH in so far as it affects the microstructure of meat and its resistance to bacterial spoilage.[18]

For instance in horse muscle which has an Eh of about + 250 mV after death falling rapidly to — 130 mV clostridia are unable to multiply at + 230 mV and have a prolonged log phase at high Eh values but grow rapidly at Eh —40 mV.

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DR. KLEEBERG emphasised the importance of pH in the maturing of meat.

DR. VAN DEN HEEVER (replying) said that more work was required before standards for South Africa could be laid down.

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IMMUNIZATION AGAINST CANINE DISTEMPER

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Onderstepoort

I have been approached to introduce the subject for discussion at this meeting, and I sincerely hope that we may all derive some benefit from these deliberations.

Vaccination against canine distemper quite often presents grave problems to the practitioner, who is not only faced with "bona fide" breakdowns in immunity after vaccination, but also with other disease conditions which are sometimes difficult to distinguish from distemper on clinical grounds, such as infectious hepatitis, leptospirosis and coccidiosis. There is no doubt that failure of immunization strains the relationship between the practitioner and dog-owner, who rankled with disappointment is often unwilling to accept a rational explanation. It is, therefore, most important that every practising veterinarian should know certain fundamental principles in order to be able to evaluate the results of vaccination and minimize failures. The vaccine itself is perhaps too often unjustifiably regarded as inadequate when such failures are encountered.

Before discussing the problem on a clinical level, certain introductory remarks are necessary. The present method of immunization with avianized vaccine, which was developed at Onderstepoort by Haig (1953), is now used in many countries of the world. In fact, some of the vaccines distributed by overseas firms are made from a strain of virus originally obtained from this Institute.

Avianized distemper vaccine is produced on the chorioallantoic membrane of embryonated eggs by using a seed virus, which has been attenuated by serial passage in eggs. The vaccine issued by Onderstepoort until 1957 was prepared from a strain of virus which had over 200 serial egg passages. Immunological studies carried out by Haig (1957) on a limited number of dogs, showed that the antibody response to this vaccine was unreliable. Some dogs developed high titre antibodies and others none at all. In comparison, vaccine prepared from a strain of virus which had received only 123 serial egg passages consistently produced a satisfactory antibody response.

Working independently, the Cornell workers (Burgher *et al*, 1958) showed that the original Onderstepoort strain after 200 passages in eggs did not immunize dogs consistently and produced

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lower antibody titres than a strain passaged through 50 generations only.

We are at present employing the virus strain after 123 serial egg passages as seed virus for vaccine production. However, the facilities for large-scale immunological studies do not exist and it is for this reason that we would welcome statistical data from private practitioners on the results of vaccination in practice. In spite of the promising experimental results with the present vaccine, we are now conducting immunological studies on lower passage levels in an endeavour to further improve the efficiency of the vaccine.

To serve as a basis for discussion of the problem on a clinical level, a number of questions have been submitted by the Department of Medicine.

(1) What is the best age to vaccinate puppies? Does an immune bitch confer any passive imimunity on the pups through the milk? If this is so, how long does the immunity last after weaning? Will passive immunity interfere with the development of active immunity following vaccination?

These questions can conveniently be dealt with in one comprehensive reply.

It has been established that a passive immunity against distemper is conferred on puppies through the colostrum of an immune bitch, but that the degree and duration of this immunity varies markedly from litter to litter and is dependent on the antibody titre of the mother (Gillespie *et al* (1958) and Baker *et al* (1959)). A bitch with high-grade immunity will have a high concentration of colostral antibodies which is transferred to the pups during the first day of life. The duration of the passively transferred antibodies is also related to the concentration of antibodies in the mother at the time of whelping; the higher the antibody titre of the bitch, the longer the period of passive immunity in the progeny. According to Baker *et al* (1959) this period varies from just after birth up till the age of 12 weeks. Variations within litters may also occur due to variations in the amount of colostrum taken in.

The practical problem which presents itself as a result of the passive protection of puppies is that colostral antibodies must disappear before puppies can be immunized successfully. Vaccine administered during the period of passive immunity is neutralized and ineffective. The pups become susceptible again after passive immunity has waned.

If the age at which passive antibodies disappear is not known, the following may happen: Pups born from a bitch with low grade immunity may become susceptible within 2 to 4 weeks of age and be exposed to natural infection and possibly contract distemper before they are routinely vaccinated at 10 to 12 weeks of age. On the other hand, pups born from a mother with high titre antibodies may in all good faith be vaccinated at 9 to 11 weeks of age or even earlier, before passive immunity has waned sufficiently for the

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vaccine to have any effect. These pups may be fully susceptible two weeks later, contract distemper and die.

Fortunately, the amount and duration of colostral antibodies in puppies is related to the antibody titre of the bitch prior to whelping. Baker *et al* (1959) have used this fact to determine the age at which puppies can be actively immunized and have produced an excellent nomograph for clinical application. All that is necessary is to obtain a serum sample from a bitch during pregnancy and test it for antibodies. From the nomograph is then derived the age at which the pups can be vaccinated safely. Unfortunately, the antibody titres of dogs do not remain stable and this necessitates a retest before each whelping.

(2) Will revaccination of a bitch 3 to 4 weeks before whelping enhance the passive immunity conveyed to pups? Will a vaccinated dog slowly loose its immunity, if it does not come in contact with distemper virus subsequently? If this is so, at what intervals should booster vaccination be given?

I have already pointed out that the amount of circulating antibodies to distemper does not remain constant. Gillespie *et al* (1958) have demonstrated a slight decline of antibody titre over a period of two years in dogs given virulent virus. They maintained that immunity could not be boosted by either virulent or attenuated virus. Recent investigations by Johnston (1959), however, showed a 250% increase in the average antibody titre of 32 dogs given a booster vaccination 6 months after the first injection.

Since lasting active immunity is presumed to be dependent on the persistence of virus in the tissues, in a state of equilibrium with the body's defensive mechanisms (York, 1954), it is conceivable that a booster effect will most readily be achieved in those animals with low grade immunity or after a natural decline of antibody titre.

In nature most dogs are continuously exposed to re-infection and receive a natural boosting of their immunity, unless they lead a very secluded life. It is not possible to lay down a hard and fast rule as to when or how often booster vaccinations should be given, except if the immune status of the individual animal has been determined over a period of time. Each case should rather be dealt with in accordance with the practitioners knowledge of the dog's habits and environment.

A dog living in a rural area and not exposed to natural infection to the same extent as a city dog with free access to the streets, would probably require a booster vaccination after a few years (York, 1954). It is also good practice to revaccinate a bitch about 3 to 4 weeks before whelping in order to assure that the pups receive adequate colostral antibodies, but such a procedure should not be relied upon to give full protection to all puppies until they are routinely vaccinated at the age of 10-12 weeks.

(3) How long after successful vaccination can a dog be regarded as completely immune?

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This period is probably variable and dependent on individual variations among dogs and the potency of the vaccine. Judging by the results of the use of an inactivated vaccine (Alexander, 1933) it appears that a sufficient immunity to protect against natural infection cannot be produced in less than 10 days. According to Gillespie *et al* (1958) a maximum antibody titre was only achieved 3 to 4 weeks after the injection of virulent virus. At 9 days most animals only showed a low level response. The immune response after the administration of a live attenuated virus would be of the same order.

(4) What are the possible reasons for failure of immunization, evidenced by contraction of distemper 2-5 weeks after immunization? These are briefly as follows:—

(a) Vaccination of puppies during the period of passive immunity will lead to failure of immunization. This was fully discussed before.

(b) Vaccination of dogs during the incubation period of the disease or exposure and infection of animals during or shortly after vaccination, will in many cases result in failure of immunization. The danger of exposure to natural infection during the immunization procedure cannot be too highly stressed. Every practitioner should adopt adequate hygienic measures to prevent the infection of susceptible dogs presented for vaccination at a surgery to which diseased dogs have access.

It is a well-known fact that distemper virus can remain latent in the brain of a dog for long periods. York (1954) referred to an instance when distemper virus was isolated from the brain of a dog 54 days after exposure. This explains why certain animals may develop distemper, especially the encephalitic form, long after they have been vaccinated.

(c) Decline of immunity in dogs which, by reason of their environment or lack of exposure, have not received a natural boosting of immunity, may be responsible for failure of immunization.

(d) The use of inactive vaccine or a vaccine of poor potency. Vaccine failures attributable to this cause is probably more the exception than the rule. The original vaccine distributed by Onderstepoort admittedly lacked in immunogenicity, but the strain which is used at present has given good results experimentally. However, it is well to remember that the vaccine contains a live virus, which looses its immunizing potency if adversely treated.

(4) What are your views about the etiological and immunological relationship between distemper and hardpad disease?

The available information indicates that hardpad disease or encephalitis is merely a manifestation of distemper. The only virus which has been isolated consistently from cases of encephalitis

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or hardpad has been that of distemper (Haig, 1953, York, 1954). Haig (1955) has also demonstrated a complete reciprocal cross-protection between a number of hardpad virus strains and a known distemper virus.

There are undoubtedly other causes of encephalitis in the dog but by far the most important one is distemper. Unfortunately, the trigger mechanism responsible for the development of encephalitis is not well understood.

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METRITIS and ENDOMETRITIS

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THE DISTEMPER COMPLEX

CAMPBELL DICKSON, M.R.C.V.S.,
Johannesburg

In recent years clinicians have come to recognise a group of conditions which affect the dog population, and since many of these conditions are presently considered to be different manifestations of the same basic disease, a portmanteau term which embraces the entire group of diseases has been coined and given the name of distemper complex.

Essentially, the distemper complex comprises the classical disease known as canine distemper, hard pad disease and, according to some authorities, infectious canine hepatitis all of which are known to be of viral origin. To these must be added a variety of infections of probable bacterial origin which, although distemper-like in character, are not associated with pure virus infection.

Opinions vary, however, whether there is, in effect, any difference between the viruses of classical or street distemper and those of the so-called hard pad disease, and many consider that the latter condition is merely a degenerative form of the original disease studied by Laidlaw and Dunkin. Infectious canine hepatitis is included in this group since it is known that the condition is frequently a concomitant or complicating factor in the distemper complex.

Some ten years ago work at the Wellcome Research Station indicated that a number of viruses could be implicated in the complex and, more recently this view has been confirmed at the Animal Health Trust Field Station at Newmarket. It has been suggested that no less than five different viruses or strains might be concerned in the distemper complex and practitioners in South Africa who have had experience of the polysymptomatic nature of the condition, favour this theory.

Understandably, therefore, accurate diagnoses pose many problems, and, in the absence of serological tests, practitioners frequently are unable to determine immediately whether the condition is due to a true classical virus infection or to one of the newer types of infection. In a number of instances conclusive proof of a virus having been present is not forthcoming, unless and until nervous complications are encountered at a later stage of the disease.

The clinician must, therefore, rely on his powers of observation and his experience in reaching a diagnosis; a careful evaluation of

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the symptoms, some of which are characteristic, may help to establish a differential diagnosis and bring some order in classifying (if only in his own mind) some of the diseases belonging to the distemper complex.

Distemper:

The classical form of distemper due to the virus described by Laidlaw and Dunkin, presents a fairly constant picture. The accepted incubation period varies from three to ten days and precedes a viral phase lasting from twenty-four to seventy-two hours which is accompanied by fever (103°—105°), rigors, loss of appetite and general malaise.

Once the stage of multiplication and spread of the virus throughout the body has occurred, there is a lowering of resistance which paves the way for invasion by secondary infection. A typical catarrhal rhinitis, accompanied by thick muco-purulent ocular and nasal discharges, conjunctivitis, persistent soft cough, mucous rales in the trachea and bronchi, and a patchy broncho-pneumonia may develop. Vomiting and retching following coughing may be seen along with varying degrees of enteritis.

The disease runs a course of approximately two to three weeks from the onset of the symptoms, during which time the temperature remains elevated, but tends to subside gradually. The appetite is variable, depending on the severity of the catarrhal infection. As this abates and the temperature subsides, some improvement in appetite may be noted. In uncomplicated cases without extensvie pulmonary involvement, recovery can be expected in two to three weeks. Nervous symptoms, however, are not uncommon and convulsions, chorea and posterior paraylsis may ensue any time from the second week onwards. In a number of instances these may be delayed as long as three weeks after an apparently complete recovery has taken place.

The above picture describes the type of distemper that many of the older practitioners were accustomed to meet in South Africa and in Britain prior to 1949 or thereabouts, but it must be stated categorically that the incidence of this classical form of distemper has apparently diminished sharply in the past ten years. To-day it is safe to say that, on the Witwatersrand, less than 10% of all cases of so-called distemper present these symptoms.

Hard Pad Disease:

In contrast to the foregoing, the incidence of this manifestation of the distemper complex appears to have risen, and in the urban areas of South Africa it is widespread. Whereas classical distemper rarely occurred in dogs over the age of eighteen to twenty-four months, hard pad disease or paradistemper has been encountered in dogs of all ages ranging from four weeks to eleven years. It is highly infectious and contagious, yet individual animals appear to show considerable variation in their susceptibility to the disease.

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Hard pad disease may have an incubation period of as little as seven days, but the average time is fourteen to seventeen days and may be as long as thirty-three days before typical symptoms develop. Frequently there is an initial transient attack of malaise characterised by a slight rise in temperature and varying degrees of tonsillitis and pharyngeal inflammation. This may persist for seventy-two hours followed by a remission of symptoms and an apparent recovery lasting two weeks, during which no symptoms are shown. Thereafter a relapse occurs, the temperature rises and remains elevated, while characteristic symptoms make their appearance.

Alternatively, there may be a history of vague symptoms of slight ill health which the owner, in all good faith, may be unable to pinpoint or even describe accurately. This phase may last for four weeks. Apart from an altered demeanour the animal, although not well, shows no signs of disease, is frequently afebrile, eats reasonably well and is fairly active. During this period, however, some hardening of the pads may have developed and hyperkeratinisation may be the only presenting symptom.

Again the onset of the disease may be a single isolated bout of diarrhoea lasting for twelve hours. This is frequently the case in adult dogs; no further symptoms develop for twenty to thirty days when either hardening of the pads is manifest or sudden nervous symptoms develop.

Thus symptoms of hard pad disease show considerable variation and the picture is further complicated by the fact that in a number of instances hardening of the pads does not occur. Many attacks commence with an attack of acute tonsillitis and pharyngitis, accompanied by fever (103°—105°), loss of appetite, a hard dry cough, shivering and dyspnoea. Careful examination of the patient may reveal little else, part from the throat infection and a significant injection of the blood vessels around the scleral-corneal margin and conjunctivitis. Eye and nasal discharges are scanty and frequently the barest trace of conjunctival crust may be seen. Some alteration in the animal's temperament may be noticed and it may become afraid to be out of the owner's sight. This phase often responds to the administration of antibiotics; the temperature subsides, the pharyngeal infection disappears and appetite returns. This remission of symptoms may persist for two to three weeks.

A number of cases, however, run a consistently high fever (105°—106°) from the onset of the disease and this may persist for five days, being replaced by a temperature which remains round 103° for four to five weeks. The breath is foetid, teeth are covered by a scum, and the pulse in the early stages is fast and bounding. An enteritis may be present and although appetite may not be completely lost, it is capricious.

This period of variable symptoms generally persists for three to five weeks, during which time there may be evidence of progressive keratinisation of pads and the epidermis of the nose. These

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changes may be almost imperceptible at first, but definite hardness and brittleness at the junction of the epidermis of the pad and skin may be noted as well as a shiny appearance to the pad itself.

A significant, and what many consider to be a diagnostic sign, however, is the marked wasting of the body musculature accompanied by excessive hair shedding and the production of considerable amounts of body scale or dandruff. This may occur in the absence of hardening of the pads, but frequently injected scleral vessels are seen with a slight trace of nasal and ocular discharge.

A characteristic attack of hard pad disease generally runs a course of from five to seven weeks from the commencement of symptoms until complete recovery with remission of fever, return of appetite and shedding of the pads has occurred. Where there has been an early apparent remission followed by a relapse, the entire length of the illness may extend over nine weeks.

Nervous complications are common and chorea and epileptiform convulsions frequently ensue from the third to fourth week onwards of the disease, or about one week after hardening of the pads first has been observed. In a high proportion of cases the chorea affects one or both hind legs and they would appear to be singled out more often than the fore limbs. The temporal, masseter and neck muscles may also be affected and in those cases where the nervous involvement attacks the muscles of the head and foretrunk primarily, encephalitis and convulsions can be expected to follow within twenty-four to seventy-two hours.

Posterior paresis without other nervous complications, is not a common sequel to obvious hard pad disease. A possible explanation may be the fact that chorea and convulsions which often become progressively and rapidly more severe, call for the destruction of the animal before paraplegia develops or is observed.

In approximately 25%' of cases showing chorea as the sole nervous complication, only one limb or group of muscles may become affected. Provided the animal remains otherwise well, the animal may accustom itself to the twitch; although it may never disappear completely, the chorea tends to become less pronounced and causes little inconvenience to the animal.

Finally, these animals which develop an encephalitis accompanied by an ever-increasing number of convulsions (10—50 during the course of twenty-four hours) proceed rapidly to complete breakdown of the central nervous system, and within seventy-two hours become incontinent, paraplegic and comatose.

Infectious Canine Hepatitis:

It is a moot point whether Rubarth's disease actually falls within the orbit of the distemper complex, but some clinicians are of the opinion, that conditions which they have treated as belonging to the distemper like group of diseases, have later proved to be infectious canine hepatitis. The prodromal stages, especially in the

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young animal , tend to be confusing when the attack of virus hepatitis is of the less acute variety.

Moreover, an intercurrent attack of straight distemper or hard pad disease cannot be ruled out and it is on record that the viruses of both distemper and canine virus hepatitis have been recovered simultaneously from dogs undergoing observation. However, there are some clinical diagnostic features which may help to differentiate C.V.H. from the distemperoid condition.

As distinct from a peracute attack of infectious canine hepatitis, sudden death or rapid collapse within twenty-four to forty-eight hours are unlikely to occur in either distemper or hard pad disease. In the severe form of hepatitis with a short incubation period, vomiting, intense thirst, abdominal pain, foetid breath and dirty mucous membranes suggest Rubarth's disease, rather than distemper or hard pad disease. The rapid onset, the character of the pulse, the passage of bright yellow or blood stained faeces, together with a high temperature may simulate an early form of biliary fever, but repeated blood smear examination should put the matter beyond doubt.

Infectious hepatitis runs a fairly short course of four to seven days and is rarely accompanied by a bacterial secondary invasion, while after the second or third day, well-marked symptoms of anaemia may be present. Nervous complications are probably the exception rather than the rule. A temporary bilateral or unilateral keratitis with corneal capacity may often be encountered at the start of convalescence.

Other Suspected Virus Infections:

During the past three years, some acute forms of canine infection have been seen in South Africa. These conditions may be part of the distemper complex, but they present a number of atypical symptoms which are nervous in character. A sudden attack of encephalitis accompanied by convulsions, hysteria and inco-ordination of gait may appear; other symptoms, apart from a raised temperature, are lacking, but there may be a history of a mild temporary digestive upset three to four weeks previously.

Again, an otherwise healthy dog may suddenly develop chorea which may be preceded for twelve hours by pain in the affected limb or tail. From the onset of the chorea the part may become useless, but the pain appears to persist, the animal may become frantic and may mutilate itself within twenty-four hours before convulsions and paralysis ensue.

In a proportion of cases, the nervous symptoms appear to be confined to the eye. The onset may be sudden, but frequently a change is noted in the dog's eyes and evidence of progressive blindness is seen over two to three days when paralysis of the iris (iridoplegia) occurs. No other symptoms may be present and apart from the blindness, the animal may be normal. Deafness and inco-ordination of gait have been seen more rarely while a few

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cases have occurred where the animal, without showing typical symptoms of chorea or convulsions, appeared to become stupid and disorientated.

A few cases of an apparent meningeal infection have been seen; these differed from the previous ones since the characteristic symptom appeared to be opisthotonus accompanied by severe occipital pain, rigidity of the neck and a marked febrile reaction.

Most practitioners have had the unhappy experience of nervous symptoms developing in dogs some four weeks after immunisation against distemper with egg adapted vaccine. It is possible that in a proportion of these cases the animal may already have been incubating a neurotropic virus infection, but it is equally true that many of them are unlikely to have come into contact with virus prior to inoculation.

Again, some adult animals which later develop nervous symptoms suggesting a neurotropic virus infection, are known to have been immunised apparently successfully with distemper vaccine and have withstood a challenge by exposure to natural infection.

These problem cases, however, are not confined to South Africa and similar evidence is available from Britain and America. To account for these so-called breakdowns, it has been suggested that the initial immunity may not be of a high order or of long duration and in time tends to wear off. Alternatively, as far as hard pad disease is concerned, it is suggested that the original virus has degenerated and in so doing has altered or lost its antigenic or immunising properties.

In dealing with the atypical forms in the distemper complex, it is possible that, in some areas with high dog populations, new strains of virus may have evolved and may be almost endemic. Such new strains are not likely to be covered by existing vaccines.

No information, however, is available on the possibility of mutations of existing viruses having occurred. The theory has not found favour among virologists, but bearing in mind that mutation of the influenza virus of humans was reported in an outbreak of that disease some two years ago, it is pertinent to enquire whether a similar mutation may not occur with distemper virus, either in the chick embryo or in the vaccinated animal with a consequent alteration in the immunising properties and an increase or decrease in virulence in neurotropic affinities.

Finally, practitioners must ask themselves whether the increased use of widespectrum antibiotics in the treatment of canine disease may not have contributed to some of the present-day problems in the distemper complex. These antibiotics are known to obliterate almost all bacteria pathogenic and otherwise, and it is just possible that by interfering with the balance of nature, some bacteria which may have had an inhibting influence on the spread and multiplication of viruses have also been destroyed.

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THE TREATMENT OF DISTEMPER

W. VON BACKSTRÖM

For the purpose of this paper Distemper and Hard-Pad is treated as variants of the same disease.

Because this is such a vast subject, I shall have to confine this brief summary to the patient of average age and state of nutrition.

A few brief thoughts on immunisation as applied in practice fall within the scope of this paper.

Immunisation

The minimum age at which we immunize is eight weeks. This is also the personally preferred and recommended time to clients.

If we assume that the average dam has been exposed to distemper, then we must accept the established fact that immune-bodies are transmitted by the dam in her colostrum and milk to the pup in sufficient quantity to give them a good immunity up to four weeks of age and a relatively good immunity up to 6 weeks or longer, depending on when they are weaned. If weaned at 6 weeks the immune bodies will have completely disappeared from the pup at 7—8 weeks. These can, therefore, no longer block the modified virus as used in the vaccine, and one can expect a solid immunity to develop.

This practice has been followed by me ever since the Onderstepoort vaccine became available, with excellent results. It is estimated that we have had about 6 to 10 "breakdowns" (in 1,000), and these mostly within 2 weeks of immunisation. It is presumed that such cases were in the incubation stage of naturally contracted distemper when immunised.

At one time it was recommended that the egg-adapted virus vaccine be used for treatment in the very early stages, a recommendation based on the theory that immunity would develop as rapidly as the "Street" virus infection. This has been proved to be a fallacy. The modified virus must be given at least 48 to 72 hours before exposure to natural infection to be effective.

I always warn clients to watch for any sign of a reaction for 72 hours after inoculation, and to be particularly careful not to expose the dog to distemper infection or even to cold or wet during this period — the latter to avoid confusion.

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If the owner is uncertain as to whether his dog has been immunised or not, we recommend strongly doing it in a dog under 2 years of age and suggest it be done in even older dogs, and hope the owner will agree.

Treatment

Treatment will be dealt with under the following headings:

(1) General Nursing.
(2) Special Drugs, Medicines, etc.

General Nursing

Good nursing in my opinion is more vital in successful distemper treatment than drugs, vitamins, etc. This contention is supported by the following facts:

(a) Once the virus of distemper has penetrated the cell-membrane, there is no effective agent.
(b) The whole aim of treatment is to support the invaded cell by nutritional methods. Antibiotics, etc., are used solely to combat the *secondary* invaders.
(c) The catabolic breakdown of tissue is increased by 20 to 30% due to temperatures varying between 103.5° to 105°. Temperature control is, therefore, important.
(d) Due to dehydration and anorexia, the patient is still further weakened; which makes it essential to administer large quantities of nutrients and liquids per os. We all know that forceful administration of proteins is essential in the distemper-patient; such nutrients are best given per os. I recommend egg-flips mainly with glucose, or in the very poor patient, protein hydrolysate, or amino-acids.
(e) Relapses in distemper seem to be caused by exposure to cold or wet. The patient kept at an even temperature, e.g. in a closed room, does much better than the one that is allowed to wander about the house and into the yard. I always advise that clients only allow their dogs out after 10 a.m. and *not* after 4 p.m. and then only if the weather is fine. From the above it will be seen that the best place to nurse a dog is in a Veterinary Hospital, where ideal conditions can be created. This is a common practice in America, but is rarely if ever done in South Africa mainly because of:
    (1) financial considerations, i.e. running a completely separate distemper isolation hospital with separate staff;
    (2) because many of us believe that many different strains of distemper exist, i.e. mild strains, neurotropic strains, etc. It would be grossly unfair to the patient and owner to expose every dog to dangerous strains.

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(1) *Special Drugs*

Hyperimmune Serum is in my opinion of most value in prophylaxis, e.g. in pups suckling and too young to be immunised, or in susceptible contact cases where I use it for 14 days and then inoculate with the vaccine after another 9—10 days, if no symptoms of distemper have developed by then.

Before the price became so exorbitant, I used it a great deal in very early cases, i.e. within the first five days of the commencement of symptoms; as soon as signs of secondary infection appeared, I no longer used it. In the early cases I do believe that it was of definite benefit (the immune bodies probably protecting those cells not already invaded). In the latter stages of the disease it was useless. The dosage rate was 2-3cc. per Kilo given intravenously and repeated at 4—5 day intervals for 12—15 days.

(2) *Antibiotics and Sulphonamides*

Depending on the secondary invader, antibiotics and sulphonamides are of equal benefit. They are always prescribed for 5-7 days, even in the first uncomplicated viraemia, and a warning sounded that if a persistent cough, vomiting or diarrhoea persists or starts after cessation of treatment, I be consulted again immediately to determine which organ-system is affected and to prescribe the most effective sulpha drug or antibiotic.

In connection with antibiotics, I prefer Chloromycetin Palmitate, because of ease of administration. Because of the predisposition of Terramycin to cause diarrhoea, I always avoid this preparation in the case of pups or in cases complicated by enteritis. Let me add, however, that when Terramycin Forte is used, or the Terramycin egg-formula, this danger disappears. The Terramycin egg-formula, as a matter of fact, gives excellent results in pups! This because it is so heavily fortified with vitamins.

Because of the tendency of Aureomycin to create nausea, I never prescribe this, especially in the inappetent distemper case, except where the owner already has a supply, in which case I prescribe Amphogel simultaneously.

Latterly Terramycin Animal Formula Soluble Powder has been used very extensively, but always with Vitamin B compound.

The opinion has been expressed that sulphonamide therapy in distemper, especially if given in maximum dosage or over a prolonged period, predisposes to nervous complications. If this surmise had been correct, one would have expected fewer nervous complications with the advent of the antibiotics. In my experience, this has not been the case. Sulphonamides are equally effective as the antibiotics in the larger and older dog. It is also much more economical.

(3) *Vitamins*

The essential vitamins are A, B and C. Although I prescribe large quantities of milk, I am afraid that I have had no experience

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in prescribing massive doses of vitamin A (50,000 I.U. daily), as recommended by South American Veterinarians. I find it difficult enough to get the owner to use vitamin B compound plus sulpha drugs or antibiotics two or three times a day!

I always prescribe massive vitamin B therapy and in my experience it is of definite benefit, not only in stimulating the appetite, but in helping to prevent nervous complications. (The vitamin B compound in tablet form, 4 to 8 daily for 4 to 6 weeks.)

In practice daily injections of vitamin B compound is impracticable and too expensive, although infinitely to be preferred, since the absorption is much better than giving it per os. A new preparation allows one to inject vitamins B and C simultaneously in a single injection.

(4) *Fluid Therapy*

In severely debilitated animals 5% dextrose-saline intravenously at a dosage rate of 10—15cc. per lb. daily is invaluable. Dextrose-saline must never be used subcutaneously or intraperitoneally. Where i/v drip administration is impracticable, give lactate — Ringer's solution subcutaneously or intraperitoneally.

(5) *Cortical Steroids*

I have had too little personal experience in the use of cortical steroids in distemper to pass on any personal observations. It is obvious, however, that in certain cases where the owners can afford it, it would be of great benefit to help in combating stress. It is given at a dosage-rate of 1 mgm. per lb. twice daily! However, a course of 3 to 4 days during the worst phase of the disease is apparently of immense benefit.

(6) *Anticonvulsants*

Since a variable percentage of patients develop either chorea or fits, one searches endlessly for drugs to control, or cure, these nervous complications.

Where the chorea is severe and affects whole muscle groups or large parts of the body, euthanasia is invariably recommended. Where the patient seems to be adapting itself physically and psychologically to localised muscle spasms, I have found tranquilizers very useful during this adaptation stage.

No tranquilizer works any better than any other, and, in my experience, *none* of them, even in very massive doses, are of any benefit whatsoever in severe chorea.

In cases of meningitis or encephalitis fits, where the attacks are not too severe or frequent, I have found Mysoline (I.C.I.) of great benefit. The real secret of successful treatment with this drug is to use it at 6-hourly intervals giving 25—50 mgm. per Kilo over 24 hours. After about 2 weeks the dose can be reduced to 2/3rd or ½ the former. To effect cure treatment preferably should be continued for 2 months: in some instances one has to continue indefinitely.

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(7) *Other Drugs Tried and Found to be of Benefit*

Through the years one continuously finds optimistic reports in veterinary journals about new drugs for treating distemper, e.g. Pacatal for chorea, penicillin intracranially, etc. Most prove to be as effective as our native method of cutting off the ear-tips. I would, however, like to mention a few where I gained the impression that the patients were benefited — but who knows with distemper?

(a) Hexamine Camphorate (Amphotropin — Bayer): 2 cc. every 2nd to 3rd day i/v, increasing up to a maximum of 6 cc. When available this drug seemed to increase the effectiveness of the sulphas or antibiotics quite markedly in some cases.

(b) Potassium Chlorate: I—V grains t.d.s. per os. In cases with severe skin eruptions and otorrhoea it definitely seems to expedite healing.

(c) ACB_{12} (A.C.T.H. 30 units plus vitamin B_{12}, 1,000 mgm. per cc.), given daily for 5—7 days, was only used in two cases. In one case for anorexia and stress and in other for fits; it was of immense benefit — but at what a price!

DISCUSSION ON SYMPOSIUM ON DISTEMPER

DR. VAN DER WALT, in opening the discussion, complimented the speakers on the way that they had focussed attention on what was known of the disease and on the great gaps in our knowledge, especially as regards early differential diagnosis and treatment. Personally, he felt that periodic booster vaccinations were to be recommended for dogs which were seldom exposed to infection. As an example he quoted a recent outbreak of distemper among the older dogs at the Police Dog Depot. All dogs were immunised at an early age and not a single young dog contracted the disease.

Dr. van der Walt said that his experience with the Onderstepoort vaccine was similar to that of Dr. von Backström. He thought that five or six cases of apparent failure in a thousand was as good as one could expect from any biological product.

Dr. van der Walt continued. I wish to elaborate on the changing clinical picture of distemper. As a student I saw nothing but the classical street distemper with a streaming muco-purulent discharge from the nose and caked eyes. Nervous symptoms were rare and the animal usually died of pneumonia, enteritis or toxaemia. To-day we seldom see such cases. Treatment with sulphapyridine and Omnadin was usually successful. In the rare nervous cases, there were a few fits and almost invariably clonic contraction of the masseter muscles. This is rarely seen nowadays.

Somwhere between 1945 and 1947 the clinical picture changed dramatically. Hardpad appeared. I use the word "hardpad" in distinction to the previously described "street distemper." I think we can assume that the two are virtually the same disease. The

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copious secretions were gone. The disease started with diarrhoea, red eyes, slight nasal and ocular discharge and hardening of the pads in about 25% of cases. It usually ended in encephalitis about three weeks later. Apparently, this change took place all over the world at about the same time. Was this a new virus spread after the war or did the virus suddenly change? Since then the picture has become even more complicated with infectious hepatitis and other viruses entering into it.

I feel that Haig's work at Onderstepoort must be regarded as the biggest advance since that of Laidlow and Dunkin. Our only solution at the moment is a blanket immunisation of the dog population. I wonder if the Association should not issue a pamphlet on immunisation for distribution by veterinarians to their clients.

I want to ask Dr. Weiss what would happen if one gave a bitch a booster dose of vaccine about four weeks before whelping and then gave the pups virulent vaccine during their first day of life.

DR. MALHERBE: There are two questions I wish to put to Dr. Weiss.

We accept that the immunity of adult dogs is boosted from time to time by natural exposure. We also accept that we cannot immunise pups while acquired antibodies are present. How is it that the antibodies in the immune adult do not neutralise the booster infection? Secondly, I would like to ask about the so-called interference phenomenon, that is, giving a large dose of vaccine to a dog which already has distemper. In my experience it has never worked but claims for it are still made.

In regard to the differential diagnosis between hepatitis (Rubarth's Disease) and Distemper I have an idea the transaminase liver function test may prove of value. We have only done a few cases where the diagnosis has been in doubt. In cases that have proved to be Rubarth's, the transaminase figures have been very high indicating liver necrosis. Small areas of liver necrosis occur in Rubarth's but not in distemper. This work is in the early stages but it looks promising.

DR. LE ROUX: After a caesarean, the pups are often not able to suckle for anything up to six hours, yet they appear to acquire a passive immunity. It appears that antibodies can be absorbed after the accepted limit of three hours.

With regard to diagnosis I believe the examination of conjunctival scraping for inclusion bodies has been tried. Could anyone comment on this?

In treatment I always use an anti-distemper serum which is a mixed serum against distemper and the common complicating organisms. Using this serum and sulphatriads my mortality rate has been 14% over the last 132 cases. This does not include dogs which were destroyed at the first visit on account of nervous symptoms.

I have seen dogs in the same house going down with hardpad

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and distemper at the same time. In my opinion the virus is the same, the variable factor being the susceptibility of the patient.

DR. MORFORD described several puzzling cases he had encountered recently. Dogs had been presented with a history of vomiting yet they retained their appetite. X-ray examination for foreign body had been negative. Careful enquiries had revealed that the animals had shown diarrhoea for a few days about a week previously. About a week to a fortnight after presentation they died in typical distemper fits. Other dogs presented had obviously been unwell but had shown no definite symptoms. The temperature had not been more than 102. Many of these had subsequently died in fits. He had used the Onderstepoort vaccine ever since it had become available with good results but recently the results had not been so good. He wondered whether yet another variant had arisen.

DR. ALEXANDER said that the appearance of hardpad might be due to some other virus which interfered with the development of an adequate immunity to the distemper virus or it might be due to another unidentified pathogenic virus. It would appear that hardpad was a clinical syndrome which occurred in animals partially immune to distemper. He stressed the importance of making a confirmed diagnosis and promised the full assistance of the virology section in this regard.

DR. MASON emphasised the difficulties of making a confirmed diagnosis in practice and pointed out that interference with immunisation by passively acquired antibodies was well known in many other diseases. He quoted diphtheria in humans as an example.

DR. WEISS, in replying to Dr. Malherbe, explained that a high titre immunity could not be boosted by reinfection but a low titre immunity could. Another factor was that the subcutaneous injection of distemper vaccine into a pup carrying antibodies brought the virus directly into the circulation where it was neutralised. In a natural infection the virus was introduced onto a mucous membrane. It is well known that focal infections can develop in spite of circulating antibodies and act as antigenic stimuli.

Dr. Weiss said that he did not think that a large dose of vaccine could exert any interference action in a dog already showing symptoms as most of the susceptible cells would already have been affected by the virulent virus and no further blocking could take place. The interference would be with the action of the vaccine virus. The interference phenomenon might act if the vaccine strain were introduced just prior to the virulent infection.

In reply to Dr. van der Walt, Dr. Weiss said that a booster dose of vaccine to the bitch could only increase the titre of antibodies in the milk. The antibodies in the pup's circulation would immediately neutralise the virulent virus.

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Jl. S.A.V.M.A. 30 (3), 1959

A FEW REMARKS ON STATE VETERINARY ORGANISATIONS IN A NUMBER OF EUROPEAN COUNTRIES

M. C. LAMBRECHTS

During a recent official tour in Europe in connection with the control and eradication of Bovine Tuberculosis, veterinary organizations in a number of countries were also discussed.

It is intended to deal briefly with veterinary services in Italy, England, the Republic of Ireland, Norway, Sweden, Denmark, the Netherlands, Germany, Austria, Switzerland and Spain.

To discuss in any detail the veterinary organizations of the countries mentioned will take up more time than could be allowed for a talk of this nature. Further information can be supplied on request.

General

Veterinary Services have become an essential part of modern society. The need for this service is recognized by all governments. The manner in which the service is to be made available to the nation differs from country to country and is dictated to a large extent by the needs of the particular country.

The rapid increase in human populations underlined the need for greater production of foodstuffs of animal origin. Often this need could not be supplied at home and resulted in the mass movement of animals and animal products between various countries. This in turn increased the danger of the spread of animal diseases to regions where they do not occur. Steps had therefore to be taken not only to control and eradicate animal diseases in the country but to prevent the introduction of diseases from outside. The development of better and more valuable animals also created a need for more veterinary services. Farmers often organized themselves into Associations or co-operatives and employed their own veterinarians.

Veterinarians are increasingly employed not only as inspectors of foods of animal origin but sometimes also of other foods intended for human consumption. The progressive development of the whole plan has extended the scope of the veterinarian to various branches of commerce and industry. All these developments are again based on veterinary education and research.

The Development of State Veterinary Services

State veterinary services were often established as a result of the necessity to control certain epidemic diseases, the organizations

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created to control these diseases being retained as the nucleus of a general service. In other instances the increasing need to control diseases of animals in the country, for reasons of economy and food supply, as well as the protection of the country's livestock against the introduction of diseases, necessitated the creation of a state veterinary service.

In countries where no serious epidemic diseases occurred the development of the state services were slow and farmers often established their own veterinary services to supply the needs of a highly developed animal industry. These services were sometimes integrated into the state service at a later stage or remained as such and complementary to the state service.

The establishment of private veterinary services were usually the result of a definite need and the reluctance of the State to supply these services. They seldom cover the entire country and ultimately result in expensive overlapping and duplication. They have the advantage of being adapted to particular regions but must lack uniformity of purpose.

In addition to the services mentioned there had been the progressive development of private veterinary practice. There are, however, few European countries where private practitioners are not in some way or other attached to the state machine. This is generally done in a part-time capacity or the execution of certain duties on a fee basis.

Veterinary education is of fundamental importance to ensure a steady supply of a sufficient number of veterinarians with the required training. It is still a state function in countries like Norway, Sweden and Denmark, etc. Elsewhere the training of veterinarians is the function of one or more universities and veterinary schools. The state remains interested in such training at all times.

The extent of veterinary training is impressive in a number of countries where the stock populations are relatively smaller than that in the Union. There are, for instance, no less than ten veterinary faculties in Italy and the total number of veterinarians in the countries visited range from more than 500 to nearly 8,000.

Veterinary control of animal diseases cannot be efficiently executed without veterinary research and it is a general feature of all state veterinary services for the director to have at his disposal one or more state veterinary research institutes. These institutes are responsible for general research, the production of vaccines and other biological products and diagnosis. Where these products are produced at state institutes uniformity in quality can generally be assured. In many countries the production of vaccines, etc., is also undertaken by private firms and in practically all those countries standards are prescribed by law and a state institute is responsible to control all products before they are allowed to be sold — examples are the State Institute at Alfort in France, and the Paul-Ehrlich Institute in Frankfurt, Germany.

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In more recent times the tendency has been to decentralize services and veterinary investigation centres or diagnostic laboratories have been established in many countries to serve particular regions or areas where the services are required. These laboratories also undertake a certain amount of research of a regional nature and co-operate with the central laboratories in research projects covering the entire country or large parts of it.

They further play a major role in disease campaigns, e.g. tuberculosis and brucellosis. There are for instance, 18 such centres in Great Britain, 10 in Norway, 10 in Sweden, 23 in Germany, 3 in Austria, 1 in nearly every Canton in Switzerland and 12 in Spain. In the Netherlands there is a laboratory attached to each of the 11 provicial veterinary services. In Italy there are 10 experimental prophylactic laboratories with a further 39 subcentres.

Most veterinary faculties and schools are also concerned with veterinary research and are often state-aided for the purposes, e.g. in Germany.

There is also a tendency to establish specialized institutes to deal with particular problems, e.g. Foot and Mouth disease, Salmonellosis, Brucellosis, Food Canning (animal products), etc.

Progressively more attention has been paid to zoonoses and even sections for the particular control of these diseases have been established.

Finally, it was found that the control of animal diseases could only be effectively controlled through international co-operation. As a result there were established the well-known organizations through which this co-operation was made possible.

The Place of Veterinary Services in the State Machine

Veterinary services, like all other services, had to develop in competition with the other services. This had a material influence on what is regarded to fall within the veterinary feld in the various countries. The veterinarian plays an important part not only in animal health but also in the production of animal products of high quality and free from disease. Animal breeding and feeding is intimately tied up with his functions. Hs duties are therefore closely connected with animal husbandry in general and also with public health. The latter relationship becomes more important in view of the large number of zoonoses.

It is therefore not surprising that there appears to have been some difficulty in placing veterinary services in the state machine. They were made a part of either Agriculture or Health. Veterinary services in England, the Republic of Ireland, Norway, Denmark, Austria and Spain form part of the departments of Agriculture. In Sweden a Veterinary Board was established in 1947 which is responsible to the Crown through Agriculture. In the Netherlands the Director of Veterinary Services is also Chief Inspector of Public Health and is responsible to both the Departments of Agricul-

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ture and Health. In Italy the general direction of veterinary services falls under a Commissioner of Hygiene und Public Health and is an equal partner to Medical and Pharmaceutical Services. There exists, however, the necessary legislation to ensure co-operation between veterinary and agricultural services.

The Federal Veterinary Service in Germany falls under the Department of Agriculture. The veterinary services of the states are, however, all part of the Departments of Health except those in the states of Schleswig-Holstein, Niedersachsen und Nordrhein-land-Pfalz.

These developments are interesting and present evidence of the fact that governments are realizing that the entire field of animal health and animal products, even in relation to public health, fall within the scope of the veterinarian. The background and training of the veterinarian form the best foundation for specialization to render the specialized services of the production, inspection and further preparation of foodstuffs of animal origin.

In Italy and the Netherlands the control of products of animal origin is entirely under veterinary control. Even abattoir construction and municipal meat and milk inspection are ultimately controlled. The same position applies in a number of the Federal States of Western Germany. Meat and milk inspection is under veterinary control in all the rest of the countries mentioned except England. Italy even provides for specialized training in the canning of animal products.

Whilst it is correct and in the interests of the particular services that the veterinarian should function in his own field, it remains most important that liaison should not only be maintained but ensured with the Departments of Agriculture and Health.

A feature of disease control in countries like Italy and Austria is that the civil control officers, Prefect and Mayor (Italy) and "State Chief", "District Chief" and Mayor (Austria), are primarily responsible for veterinary measures against disease with the assistance of the veterinarians concerned.

In Denmark the private practitioner and police chief take the first steps to control outbreaks of disease.

The Scope and Organization of Veterinary Services

The State Veterinary Services are generally charged with the following duties:—

1. Control and eradication of diseases of animals declared under law.
2. Research, vaccine and other biological production and diagnosis.
3. Advice in connection with stock disease legislation.
4. Control and registration of veterinary remedies.
5. International co-operation in the control of animal diseases.
6. Stock disease reports.
7. Control of artificial insemination as a means of disease

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control. The quality of the seed is also a veterinary responsibility.

8. Inspection of meat for export and import purposes. In some countries also inspection for internal consumption forms part of the duties of the state service.
   Milk inspection is also sometimes included.
9. Control of the import and export of animals and border control.
10. Control of zoonoses.
11. Implementation of the laws for prevention of cruelty to animals.
12. Representation on control bodies of farmers' organizations and co-operatives, etc.
13. Execution of such other duties as may become necessary from time to time.

In some countries the veterinary service is also responsible for the hygiene and control of stock markets, agricultural shows and other gatherings of animals; public drinking places, stables, manure disposal, collecting and handling of offal, abattoir construction; the movement of animals and the means of transport; bees and honey products; the control of the veterinary profession. Also municipal meat and milk inspection and private abattoirs are controlled in certain instances.

Finally, the state veterinary service is insurance against the decimation of the country's livestock and animal industry by the invasion of disease.

The organization of the veterinary services naturally varies but it is interesting to note that a certain pattern of control arrangement is adhered to practically throughout. The Director, or Chief, is assisted by a deputy or deputies followed by a number of senior officers each of whom is responsible for a particular part of control (one or more diseases, imports and exports, etc.). Thereafter follows provincial, regional and/or divisional control with the State Veterinarian right at the front of action, sometimes assisted by lay units.

The inclusion of jurists in the veterinary organization, e.g. Norway, Sweden, Germany, is interesting and must serve a very useful purpose.

A further feature in many countries is the use made of private practitioners. In Italy they are employed as part-time border control officers, officers in control of ports and airports, in certain instances, and part-time meat inspectors. In Switzerland very much the same pattern applies. In England large numbers of practitioners are on the special and general panel and perform state duties at prescribed fees. Practitioners perform part-time state duties in all the other countries concerned and in Sweden this organization is so arranged that very few practitioners are engaged in practice only. Sometimes practitioners are employed full-time on a temporary basis, e.g. in England, Spain, Germany.

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These officers are mainly engaged on tuberculin testing and the control of brucellosis.

Research institutes generally enjoy a fair amount of autonomy but always fall under the direction of the Chief of the Service.

State veterinary research is often supplemented by special research organizations like the Agricultural Research Council in England, specialist institutes, regional laboratories, universities and private firms.

Admission of veterinarians to the state service in e.g. Germany and Austria is subject to some 8 years' experience as a practitioner followed by about a year's intensive study in veterinary law, meat and milk inspection and finally a State Examination. State veterinarians are allowed to practice privately on their own account in Norway and Sweden.

Technical assistants for employment in laboratories are specially trained and receive diplomas on completion of their studies, e.g. Germany. The same applies to lay meat inspectors. The latter class of officials are not extensively used on the continent where meat inspection is essentially carried out by veterinarians.

The large numbers of veterinarians in state employment in the various countries, either full- or part-time, are proof of the requirements of the modern State Veterinary Service.

Conclusion and Discussion

Observations in Europe indicate that the responsibility of the state in regard to veterinary control does not decrease with modern development. The opposite is rather the case. Improved animals and farming methods, increased demands for foodstuffs of animal origin and the development in food preparation dictate not only bigger but more specialized state veterinary services. The animal industry has also become relatively more precious and must be protected. The large numbers of veterinarians required to combat and eradicate diseases like bovine tuberculosis are beyond the scope of any state service. This difficulty had been overcome by making extensive use of the services of practising veterinarians who have often been integrated in the state machine. In this way it is generally possible to establish a sufficient veterinary force to meet all eventualities.

Veterinary research is undertaken on an extensive scale. Apart from topical research, modern developments have also dictated fundamental research. It is further a recognised fact that central research institutes can, in most instances, no longer cope with all the needs of research and diagnosis. Especially the latter service had to be decentralized in the interests of disease control generally, regional research in co-operation with the central institutes and the needs of particular regions and types of farming. Particular circumstances cannot always be reproduced at the central research institute. These laboratories also lend themselves as administrative centres of a regional nature.

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Specialist institutes have been established, sometimes at very considerable cost, as a further safeguard of the countries' animal industries.

Veterinary education can be the function of the state or universities, if the latter are financially able to undertake this service. The state should, however, remain interested in the training of veterinarians through its State Service and by way of financial support where necessary. The curriculum should be realistic and in step with modern demands. Provision for post-graduate and specialized training have become essential facets of this service and must provide the trained manpower required for the varied aspects of modern veterinary administration and control.

Efficient veterinary administration can be achieved in more than one way. The Swedish system of a Veterinary Board whose Director in Chief is also chairman of the controlling body of the State Research Institute and *ex officio* member of the controlling body of the Royal Veterinary College, appears to have several organizational advantages. The four members of the Veterinary Board are also Heads of Departments and administer specific aspects of control. The controlling bodies of both the Royal Veterinary College and the Research Institute are appointed for definite terms of office.

The patterns of administrative organization found elsewhere are obviously based on experience and the needs of the country. The general arrangement of a Director, assisted by Deputies or other senior officials as Heads of Departments or Sections, responsible for particular aspects of control, must have the merit of establishing specialized control. Specialization of this nature has the further advantage of placing at the disposal of the director a well balanced advisory body of responsible officials.

A scientific advisory committee of the nature of that existing in Sweden appears to be a further modern innovation which is essential to enable the director to perform his duties efficiently. Representation on this committee can also serve to establish essential liaison with other services.

The director (chief) of veterinary services should be represented either directly, or through his staff, on all bodies, organizations, etc., which are concerned with animals and their products.

Regional veterinary administration is usually arranged in Provincial, Regional, Divisional and District veterinarians' districts. The extent of these services depend on the particular needs.

The function of the veterinarian in connection with the production, handling, inspection, etc., of foodstuffs of animal origin, has been widely recognised. This public health service is variously rendered, either directly by the State Veterinary Administration and in conjunction with local authorities, farmers' associations and co-operatives or indirectly by the departments of health with State veterinary control of imports and exports. Fractional veterinary control is detrimental because it prevents proper planning on a

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national scale and sometimes results in expensive overlapping of control, which hampers efficiency.

The ideal and most efficient control and administration could be achieved by placing the entire aspect of hygiene and public health, of a veterinary nature, in the hands of the state veterinary administration. The control of animal transport, markets, shows, expositions, slaughterings, care of meat and other animal products, disposal of manure, etc., form essential aspects of the control of animal diseases and zoonoses. The further treatment and manufacture of animal products also rightly belong in the veterinary field.

A unified service will enable of a balanced approach not only of the specialized training required to perform all these functions, but also a planned service with enormous advantages to the state economy.

A service of this nature should obviously be an autonomous unit. Its functions should be clearly defined by law and provision made for the necessary liaison and co-operation with allied departments.

Acknowledgements

The author wishes to thank sincerely all the colleagues who so kindly supplied information on this subject.

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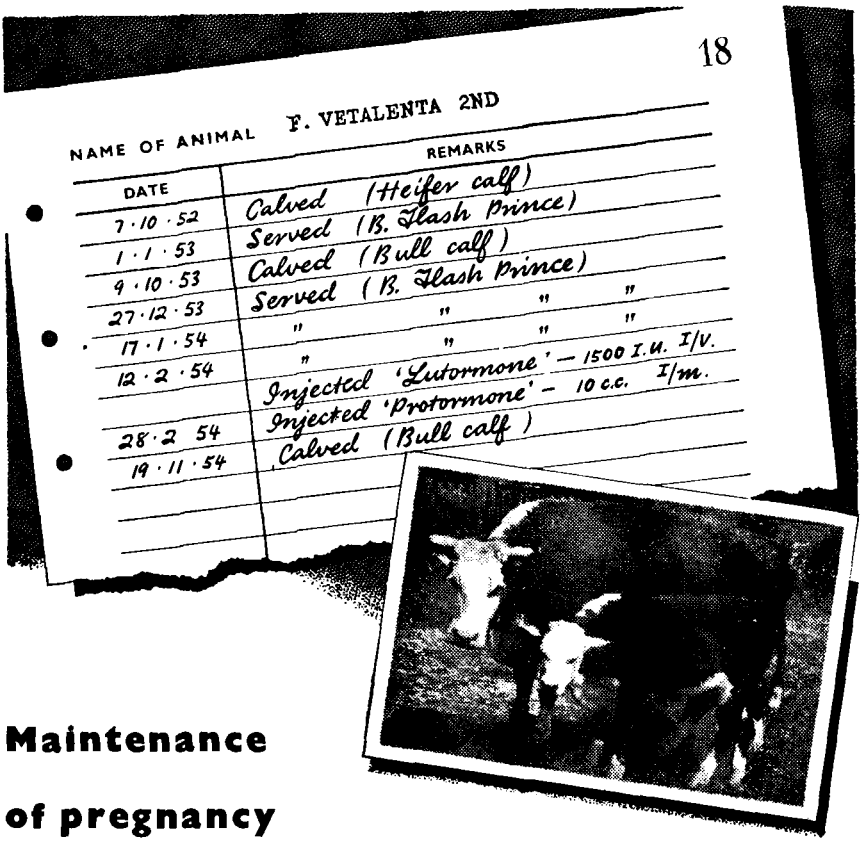
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THE USE OF LOW LEVEL PHENOTHIAZINE IN A SALT — BONE MEAL LICK FOR THE CONTROL OF INTERNAL PARASITES IN SHEEP

R. J. THOMAS
Ermelo

Introduction

A considerable number of reports have appeared in the literature on low level phenothiazine dosing since it was reported in the early 1940's that continued daily administration of phenothiazine in small amounts caused a marked suppression of parasite egg output, and inhibition of larval development in the faeces of sheep and cattle. (Shorb and Haberman, 1950; Haberman and Shorb, 1952). To-day it is generally agreed that a minimum daily intake of 0.5 gm. per sheep is required; at that dosage egg output is suppressed and larval development inhibited, but the adult worm burden not eliminated from the host. (Edwards, 1947; U.S.D.A., 1950; Harwood, 1953).

Review of Literature

A large number of trials have been carried out, both under laboratory conditions and in the field, to determine the value of this system in the control of parasitism in sheep. Most of this work has been done in North America, where it is the practise to allow the sheep free access to a lick containing 9 parts salt to 1 part phenothiazine. Some reports are also available from Britain.

The American work was largely concerned with the control of wire worm *Haemonchus contortus,* and the results from both short-term experiments (Colglazier and Turner, 1955; Turner and Colglazier, 1956; Seghetti and Marsh, 1945), and long-term field trials (Rietz and Wilson, 1949; Price et al. 1953; U.S.D.A., 1954) showed that where *H. contortus* predominated, satisfactory control of parasitism in both ewes and lambs resulted from the use of a 9:1 salt-phenothiazine mixture. Swales (1952) reported good results against both *H. contortus* and the nodular worm *Oesophagostonum columbianum.* In several cases (Colglazier and Turner, 1955; Swales, 1952), successful control was achieved under conditions in which non-medicated lambs suffered severely from clinical haemonchosis. Petersen et al. (1944), using a 14:1 salt-phenothiazine mixture, found that while satisfactory control was obtained in a flock maintained in isolation, clinical parasitism occurred when these sheep were moved to a heavily contaminated pasture, thus being exposed suddenly to a high rate of infection.

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Shelton et al. (1957) reported clinical parasitism occurring in both ewes and lambs on irrigated plots, receiving 9:1 salt-phenothiazine mixture combined with fortnightly dosing with phenothiazine or a copper-nicotine preparation. However, these sheep were exposed to an extremely high rate of infection, pasture larval counts reaching 51,000 larvae per Kilogram of grass, and it is probable that no dosing system would be effective under such circumstances. The authors themselves point out that their results do not agree with those obtained elsewhere under irrigation conditions, where satisfactory control was obtained, and ascribe the failure to the abnormal conditions of the experiment.

It was generally found that given free access to the mixture, both ewes and lambs consumed approximately 5 gm. per day, thus receiving $\frac{1}{2}$ gm. per day of phenothiazine in a 9:1 mixture. Shelton et al. (1957) stated that lambs did not take much of the lick until after weaning, but Turner and Colglazier (1956) found that lambs of two months old would take 5 gm. per day.

In Britain, Harbour et al. (1946) reported a significant egg suppression effect with a 14:1 salt-phenothiazine mixture, but obtained better results from monthly therapeutic dosing. In this case the average consumption of lick only provided 0.25 gm. of phenothiazine per day, which is well below the accepted optimum requirement. Page (1949) reported an 80% suppression of egg output in lambs with a heavy infection of *H. contortus*.

In Australia, Gordon (1958) stated that this system has not been used on a large scale due to unsatisfactory results from field trials. No detailed reports are available, but the poor results were apparently due to an inadequate consumption of the lick by the sheep.

There is thus some variation in the degree of success obtained in different areas using this system. In general it appears that where *H. contortus* is the predominant species and where sheep receive $\frac{1}{2}$ gm. of phenothiazine per day, satisfactory control of parasitism is obtained. In general, no trouble was experienced in getting sheep of all ages to take the required quantity of lick, and no reports of toxicity or adverse physiological effects have been published.

However, in recent years, there have been reports that phenothiazine is losing its effectiveness in some areas due to the suspected development of resistant strains of parasites following extensive and prolonged use of the drug (Bailey and Herlich [1953]; Baker et al. [1954]). Leland et al. (1957) made a detailed investigation of one of these strains of *H. contortus* and found it to be 4-8 times as resistant as a control strain. Observations on farms showed that in the flock from which this strain was isolated, salt-phenothiazine mixture in normal dosage did not effectively inhibit egg production or larval development, and that therapeutic doses of phenothiazine were also ineffective. A daily intake of 2 gm. of phenothiazine was required to control this strain compared with

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0.5 gm. for a normal strain. On this farm both therapeutic and low-level dosage of phenothiazine had been used extensively for over 10 years, and the authors considered this to be a cause of the development of the resistant strain. Low-level phenothiazine dosing was not believed to be the only cause of the development of the drug-resistant parasites and therefore there was no justification for the condemnation of the method for that reason alone. The recommendations emphasised the importance of alternating phenothiazine with other drugs, and supported the continued use of salt-phenothiazine mixtures in addition to drenching with therapeutic doses.

No information is available on the suitability of the low level dosing system under South African farming conditions, but several factors suggest that it would be of value. Firstly, conditions in some regions of South Africa are similar to those parts of America where the system has been successful, in that there is a warm climate with high summer temperatures and summer rainfall, and the predominant parasite is *H. contortus*. Secondly, the provision of a salt-bone meal lick is a common practice, and the incorporation of phenothiazine is a simple matter. In the summer rainfall areas of the Union a tremendous build-up of infection with *H. contortus* occurs in summer, and control measures against this infection are generally based on a regular 3-weekly dosing system. This gives some measure of control but even so a considerable loss of condition usually occurs, particularly during the late summer, and clinical manifestations are quite common.

The 3-weekly dosing system suffers from several main disadvantages, the first of which is that the drugs in common use are effective only against adult parasites and not against immature stages. Thus, at one 3-weekly dosing, only one third of the total parasite burden will be removed, the remaining two-thirds reaching maturity and causing considerable damage before the next dose is given. Secondly, the rate of infection in summer is so high that during the 3 weeks between successive dosings a large burden of young stages can accumulate and these are probably just as pathogenic as the adults. This applies particularly to *H. contortus* because of the very high egg output of the female worm. In addition, there is a strong prejudice against dosing ewes during the last 2-3 months of pregnancy, which in an autumn lambing area covers the late summer period when maximum infection is occurring, combined with a steady decline in nutrition. There is thus a very severe risk of clinical disease, or at least a marked loss in condition, at this time.

For these reasons it was decided to carry out trials in the Eastern Transvaal highveld at Ermelo to investigate the efficacy of continual low-level phenothiazine dosing through the medium of a salt-bone meal lick as a method of parasite control, either alone or in combination with therapeutic dosing.

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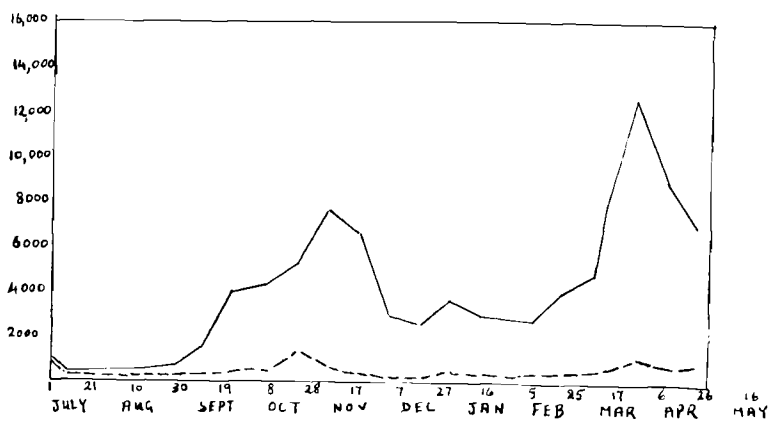
Method

Two comparable flocks of 25 ewes and 25 lambs were selected at random from a single breeding flock and placed in adjoining 10 morgen camps, in which the conditions are believed to be identical. When the experiment began in July, 1958, the lambs were about 3 months old. The experiment terminated in May, 1959. Each group of sheep remained throughout in the same camp and were subjected to the same treatment in regard to feeding and management except that one group received a salt-bone meal lick containing 10% phenothiazine while the other group received the salt-bone meal lick with no addition. The plain lick contained 2 parts bone meal to one part salt, the medicated lick 6 parts bone meal, 3 parts salt and 1 part phenothiazine. A daily amount of lick was set out allowing 5 gms. per sheep, equivalent to ½ gm. phenothiazine per day. Faeces samples were taken fortnightly from both flocks for egg counts and larval differentiation, and the sheep were weighed monthly. Blood samples were taken from 10 lambs in each group in April, 1959. In May, 1959, 10 lambs from each group were slaughtered for accurate determination of the worm burden. It was intended that the control flock should receive no anthelmintic treatment, but in December and February clinical symptoms of parasitism were apparent. To obviate the possibility of severe mortality with consequent loss of comparative data it was necessary to adminster two therapeutic doses of phenothiazine to this group.

(1) *Faeces Egg Counts*:

The results are shown graphically in figures 1 and 2.

FIGURE 1.
Egg Counts: Ewes.



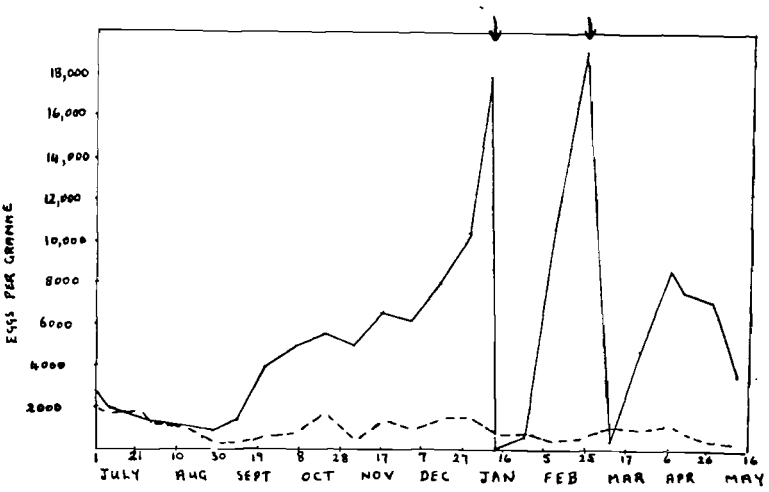
Low level phenothiazine - - - - - Controls ———

Worm egg counts in both ewes and lambs in the treated flock remained at low levels throughout. Mean counts ranged from

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FIGURE 2.

EGG COUNTS: LAMBS.



Low level phenothiazine - - - - - -

Controls ———

Dosed 20 gm phenothiazine at arrows

100 to 1,300 eggs per gram in the ewes and 250 to 1,900 eggs per gram in the lambs and showed only minor fluctuations. In 5 lambs counts of over 1,000 eggs per gram persisted throughout the experimental period, while the highest individual count recorded was 6,500 eggs per gram on December 30th.

By contrast mean egg counts in the controls showed a very marked seasonal rise in summer, reaching peaks of 13,000 eggs per gram in the ewes, and 18,000 eggs per gram in the lambs. The mean egg count in the lambs was only checked at this level by anthelmintic treatment. Individual counts in the lambs often reached 25,000 eggs per gram.

(2) *Larval Differentiation*:

Counts of greater than 100 larvae were obtained regularly from over 90% of faeces cultures from the controls, and showed a mixed infection with *H. contortus*, *Trichostrongylus colubriformis* (the bankrupt worm) and *O. columbianum*. *H. contortus* predominated in the lambs, accounting for over 90% of the total viable egg output.

In the treated group, counts of 100 larvae were usually obtained from less than 20% of the faeces cultures. Accurate conclusions could not be drawn from the small number of larvae recovered, but they indicated that the proportion of *Trichostrongylus* larvae was considerably higher than in the controls, while *Oesophagostonum* larvae were almost completely absent.

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FIGURE 3.

BODY WEIGHT: EWES.

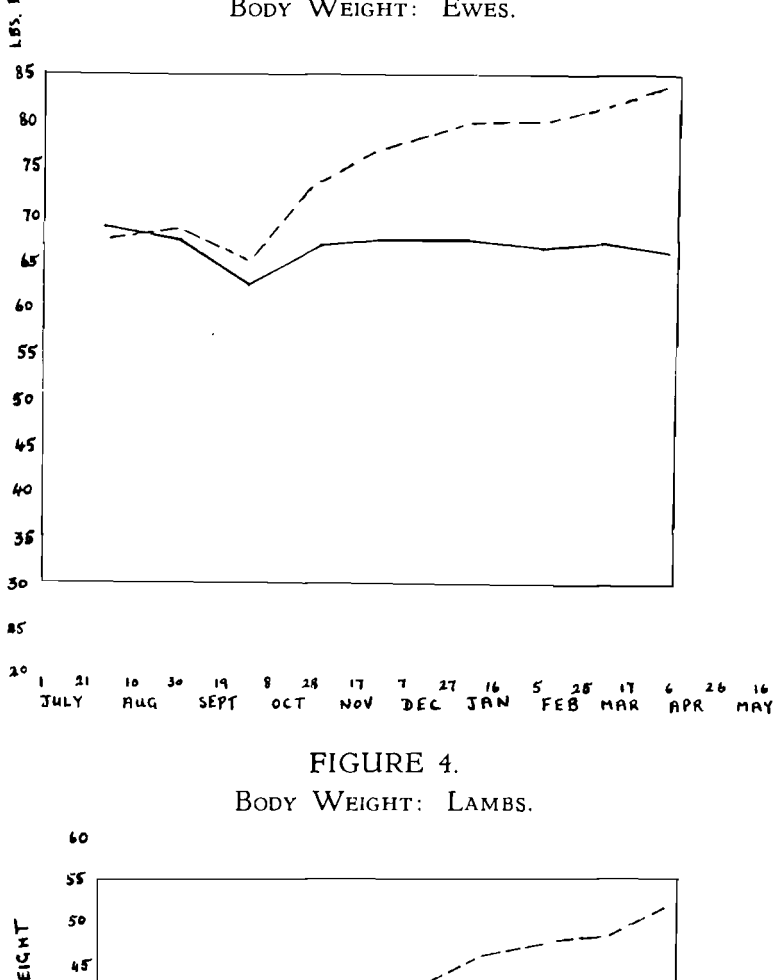
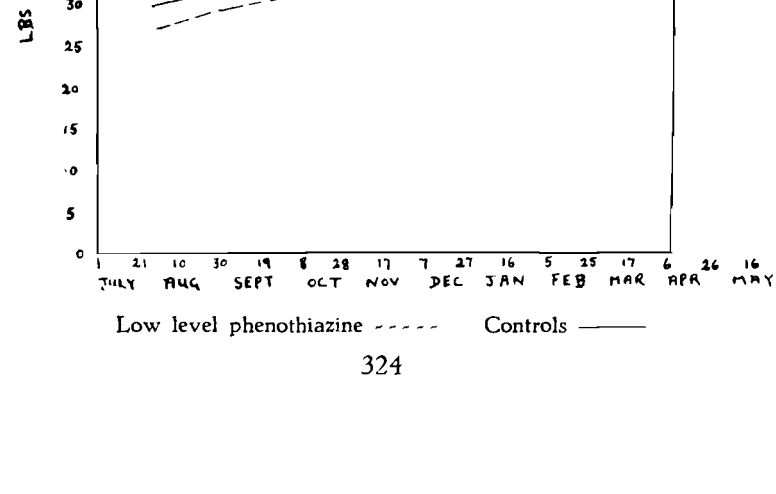


FIGURE 4.

BODY WEIGHT: LAMBS.



Low level phenothiazine - - - - - Controls ———

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(3) *Live Weight Gains*:

The results are shown graphically in figures 3 and 4.

(a) *Ewes*: In the treated group the mean body weight declined slightly in the period July to September from 67 to 65 lbs. then increased steadily to reach 84 lbs. in April, a live weight gain of 18 lbs.

The controls showed a decline from 68.5 to 62 lbs. from July to September, rose to 66.5 lbs. in October and then remained stationary at that figure throughout the summer, thus showing an overall loss of 2 lbs.

(b) *Lambs*: In the treated group, the mean body weight showed a steady increase throughout from 27 lbs. in July to 44 lbs. in December and 52 lbs. in April, a gain of 25 lbs. representing a live weight increase of 92%.

In the control lambs, the mean weight showed an increase from 29.5 lbs. in July to 39.5 lbs. in December, but then declined to 37.5 lbs. in April, an overall gain of 8lbs., representing a live weight increase of only 27%.

(4) *Clinical Disease*:

In the controls, clinical signs of verminosis were apparent during January to April as evidenced by anaemia, soft or fluid faeces, loss of weight and unkempt wool; 3 ewes and 7 lambs died from verminosis during this period, and much higher lamb losses were only prevented by anthelmintic dosing.

The treated group showed no clinical signs of parasitism and no losses occurred. Furthermore, at no time did this group show any check in condition or weight gain.

(5) (a) *Post-mortem Results*:

10 lambs from each group were slaughtered in May, and total worm counts carried out. The mean numbers of worms recovered are shown in Table 1, together with the mean egg counts and haemoglobin levels.

TABLE 1.

MEAN WORM COUNT, EGG COUNT AND BLOOD HAEMOGLOBIN LEVELS.

	Treated Lambs	Controls
H. contortus	304	1,019
T. colubriformis	952	925
O. columbianum	10	163
Mean egg count	370 e.p.g.	10,960 e.p.g.
Mean haemoglobin levels	8.5 gm.%	5.85 m.%

6 out of 10 controls were infected with small numbers of lung worms (D. filaria), and 4 out of 10 treated lambs.

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The worm burden in the untreated lambs at slaughter was considerably lower than that found in lambs from this group which died from verminosis during the summer. This is due in part to the effect of the second dose of phenothiazine given in February, and in part to the development of natural resistance in the surviving animals. Sheep dying from haemonchosis during the summer contained up to 8,000 *H. contortus*.

(b) *Examination of Parasites*:

Length measurements were made on 50 males and 50 females of *H. contortus* from the treated lambs, and on the same number from the controls. The females were also examined for the presence of eggs. Similar observations were made on 25 males and females of *T. colubriformis* from each group. Only small numbers of *O. columbianum* were recovered, and measurements were made on only 2 males and 5 females from the treated group and 5 males and 5 females from the controls. The results are shown in Tables 2 and 3.

TABLE 2.

	Mean Length	
	Males	Females
H. contortus		
Treated lambs	16.1 mm	17.0 mm
Controls	15.7 mm	19.8 mm
T. colubriformis		
Treated lambs	6.1 mm	7.6 mm
Controls	6.2 mm	6.7 mm
O. columbianum		
Treated lambs	12.5 mm	18.25 mm
Controls	14.4 mm	20.4 mm

TABLE 3.

	Egg Development
H. contortus	
Treated lambs	37 females contained no eggs, 12 contained very few eggs, 1 contained a large number of eggs.
Controls	All 50 females contained large numbers of eggs.
T. colubriformis	
Treated lambs	Average of 15 eggs per female worm.
Controls	Average of 13 eggs per female worm.
O. columbianum	
Treated lambs	5 females examined contained no eggs.
Controls	5 females examined all contained large numbers of eggs.

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(c) *Wool Production*:

The 10 lambs slaughtered from each group were shorn before slaughter, and the fleeces weighed and examined. The mean results for the two groups are given in Table 4.

TABLE 4.

	Fleece weight	% grease	% suint	% clean yield
Treated lambs	3 lb. 2 oz.	12.6	7.5	68.4
Controls	2 lb. 4 oz.	13.8	7.3	67.4

Fairly extensive areas of staining with phenothiazine were found in the fleeces from the treated group, particularly on the neck and shoulders. A variety of methods were used to attempt to remove the stains, but none were successful.

Discussion

The results obtained in this study agree closely with the general conclusions of other workers, and indicate that a high level of parasite control was achieved by this system. A general reduction in egg output of up to 90% compared with undosed controls is in line with the 80-90% reduction reported by Page (1949), Price et al. (1953) and others.

The results of larval culture, showing 80% inhibition of larval development, are also very similar to those obtained by American workers. As the effect of this system of treatment is largely one of egg suppression, egg counts alone indicate a high degree of control of pasture contamination, but are not an accurate guide to the degree of control of adult worm burden achieved and to the economic value of the method. However, the difference in weight gain between the treated group and the controls, together with the complete absence of clinical signs of parasitism in the treated animals indicates that a high, and economically important, level of control of worm burden was obtained. The losses suffered by the controls, despite two doses of phenothiazine, indicated that under these conditions both groups were exposed to a very seriouss risk of clinical disease, and this emphasises the efficient degree of control resulting from the low level dosing system.

From the economic aspect the results obtained fully justify the costs incurred. The cost of the phenothiazine used is estimated at approximately two shillings per head per year. This expenditure resulted in the elimination of all mortality from verminosis, an average weight gain 15 to 18 lbs. per head higher than in the controls, and a wool clip 140% of that from the controls. The usual farm dosing programme, involving regular 3-weekly dosing using mainly cheaper remedies, supplemented by phenothiazine twice a year, costs approximately 9d. to 1s. per sheep per year,

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and involves a considerable amount of time and labour. Furthermore, under the conditions of severe challenge which occurred in this experiment, it is unlikely that this 3-weekly dosing system would have given adequate parasite control in mid-summer, and considerable loss of condition and possibly some deaths would have resulted. The 3-weekly dosing system is widely practised in Eastern Transvaal, and farmers generally agree that during the mid- to late-summer period it does not result in adequate control of parasites; some loss of condition always occurs. Thus there would appear to be strong economic justification for recommending the introduction of the low-level phenothiazine dosing system as an alternative method of parasite control.

Other studies on the seasonal incidence of worm infection in sheep suggest that an effective system could be planned for the high-veld region using low-level phenothiazine only over the infection period from November to April combined with a single dose of some other remedy such as a copper-nicotine preparation in September or October depending on weather conditions to minimise the *H. contortus* burden, and a final full dose of phenothiazine or tetrachlorethylene in May to reduce the *Trichostrongylus* burden to a minimum. Further dosing through the winter would be unnecessary. The total cost of this programme would be approximately 1 shilling per sheep per year, very little more than the cost of the normal dosing programme, and showing a marked reduction in time and labour. This shortened programme has still to be tried out in practice. A suitable experiment has been planned for the 1959-1960 season.

A comparison of the treated and control flocks shows clearly the influence of worm infestation on growth and production, quite apart from the actual loss from deaths due to parasitism. This is, of course, not a comparison between infested and worm-free sheep, but between a group with a naturally acquired infection and one in which the infection was controlled efficiently.

Over the period July to April the treated ewes and lambs showed live weight increases of 26% and 92% respectively. In the untreated group over the same period the surviving ewes showed a loss in weight of 2.5 lbs., i.e. a live weight decrease of 4%, while the lambs showed a live weight increase of only 27%. Thus the heavy worm burden in the surviving lambs caused a 65% depression of live weight gain as compared with the treated group in which parasite infection was adequately controlled. The untreated lambs also showed a 30% depression of wool production, with virtually no differences in grease, suint or clean yield. It must be concluded that this represents an actual depression of production of wool keratin. These results are in agreement with those of Spedding (1955), who found a 25% reduction in live weight gain in sheep with a fairly light infection, and Carter et al. (1946), who found a 40% reduction in wool production in sheep with moderately heavy infections.

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The graphs of live weight gain (figures 3 and 4), show that the treated group made steady weight gains throughout the spring-autumn period, whereas the controls showed a gain in weight only during September-October. During the period of worm infestation from November to April the ewes just maintained their weight, while the lambs actually showed a loss in weight during January to March. Thus, under conditions of heavy parasitic infection there was a complete failure to benefit from the high nutritional level of the grazing available during the summer months. Therefore the economic cost of parasitism is of major importance, and far outweighs the cost of an efficient control system.

The post-mortem results (Table I), confirm the conclusions of other workers that the low level phenothiazine system does not eliminate the worm burden but operates largely through a very marked suppression of egg production and inhibition of larval development. As a result pasture contamination with infective larvae remains extremely low even during the period of most favourable climatic conditions. Thus under this system the sheep are never exposed to high rates of infection, and therefore heavy worm burdens do not develop. The low rate of infection which occurs probably maintains a low worm burden similar to that in the slaughtered treated lambs, which does not have any marked effect on the health of the animals and can be adequately controlled by their own resistance mechanisms.

This sterilisation of the pasture is of great importance, for it means that when the intake of phenothiazine is uneven, and some animals do not take an adequate amount, the overall pasture contamination remains too low to produce a dangerous infection even in these animals. As pasture sterilisation is the major effect of this system it is essential that the animals should not be suddenly exposed to a heavily contaminated pasture or control is likely to break down, due to the failure of the sheep to develop an adequate immunity. It is therefore essential either to make the medicated lick available to the entire flock or to ensure that treated sheep do not graze over ground occupied by untreated sheep during that summer. Preferably all the sheep should receive the same treatment so that maximum value is obtained from the pasture sterilisation. In America (Turner and Colglazier, 1956) and Canada (Swales, 1952) it has been shown that due to the sterilisation of pastures the parasitic infection declined in successive years. While this is never likely to lead to complete eradication of infection, it would reduce the overall worm burden to a low level, and raise the productivity and economic efficiency of the flock accordingly. Further, it is obvious that no sheep should be introduced into the flock or onto the grazing prior to a full therapeutic dose of phenothiazine.

While the major effect of the treatment is the suppression of egg production and larval development, it is possible that some effect is also exerted on the worm burden already present in the

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animals. This is suggested by the fact that the treated sheep showed a higher weight gain than the controls during August-September, at which time no fresh infection was occurring due to unfavourable climatic conditions, and the only influence the dosing system could have had was on the existing worm burden of the sheep. Both groups were maintained under identical conditions at this time.

In full therapeutic doses it is generally agreed that phenothiazine is highly effective against *H. contortus* and *O. columbianum*, and rather less effective against *T. colubriformis*. Observations on the worms recovered at post-mortem from the two groups confirm that a similar gradation in effectiveness occurs when the low level system is used. The results of these observations are shown in Tables 2 and 3, and the three species are considered separately below.

T. colubriformis:

Length measurements were made on male and female worms from both groups, and no significant differences were observed. Similar numbers of eggs were observed in females from both treated and untreated lambs, and in both groups the eggs appeared normal. Thus the dosing system does not appear to have any influence on the development of the parasites or on egg production. It is probable that the bulk of the egg output in the treated lambs was due to this species. However, despite the presence of quite large numbers of *Trichostrongylus* eggs in the faeces, very few larvae were recovered from faeces cultures in the treated group, and it must be assumed that either the eggs or the developing larval stages in the faeces were killed by the phenothiazine present. Thus against this species the action of phenothiazine appears to be inhibition of egg or larval development. The presence of an average of 952 worms in the treated lambs suggests that the drug had no effect on the establishment of this species in the sheep.

H. contortus:

The mean length measurements show no marked differences between the worms recovered from treated and untreated lambs, and indicate that the low-level system does not interfere with the normal growth of the parasite. However, all 50 females examined from the untreated group contained large numbers of eggs, whereas in those from the treated group only one contained a large number, 12 contained small numbers of abnormal eggs and 37 or 74% contained no eggs at all. Thus the administration of low-level phenothiazine appears to exert a marked effect on egg production in the female worms. The presence of a mean burden of 304 worms suggests that in view of the low rate of infection occurring it is likely that the dosing system did not have any effect on the development and establishment of *H. contortus* in these sheep.

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O. columbianum:

Very few adults of this species were recovered from the treated lambs, but these showed no marked size differences from those found in the controls. Of 5 females examined from the treated lambs none contained eggs, whereas females of similar size from the controls contained large numbers of eggs. Again the major effect appeared to be a suppression of egg production. However, an interesting feature observed at post-mortem was that, in the treated group, only 1 lamb showed the presence of nodules in the wall of the large intestine, whereas in the controls 9 out of 10 lambs showed extensive nodule formation, often involving both the small and large intestines. The absence of nodules from 90% of the treated lambs may be due solely to a very low rate of infection with this species, but it is possible that the complete absence of nodules, and the very low adult burdens recorded may indicate some additional activity of the drug preventing the establishment of ingested larvae. It has been suggested that the presence of large numbers of nodules may be a contributory factor in the incidence of intussusception. If this is correct, then the low-level dosing system should be of considerable importance in controlling this disease.

Thus there is a gradation of effectiveness against these three species, being highly effective against *O. columbianum* and *H. contortus* and considerably less effective against *T. colubriformis*. This may have a considerable bearing on the value of the low level dosing system in different areas, and may to some extent explain the lack of success from the use of this method reported by some authors. For example, in Scotland and the North of England, the major parasites are species of *Trichostrongylus, Cooperia, Ostertagia* and *Nematodirus*. *H. contortus* occurs only in very small numbers, while the species of *Oesophagostomum, O. venulosum,* does not cause nodule formation and is therefore not a serious problem. Of the major parasites, phenothiazine is only highly effective against *Ostertagia*, and much less effective against the rest. This would explain the rather poor results obtained with the low-level system by Harbour et al. (1946) in this area.

The lower efficiency against *Trichostrongylus* species is a slight disadvantage. However, this trial was carried out using commercial phenothiazine of coarse particle size, and it is possible that a higher efficiency would be obtained from the use of microfine phenothiazine, which in therapeutic doses is more active against *Trichostrongylus*. Furthermore, there is considerable evidence that a heavy infestation with *H. contortus* greatly reduces an animal's resistance to infection with *Trichostrongylus* species, and control of *H. contortus* enhances the ability to withstand *Trichostrongylus* infection. Thus the efficient control of the former species should enable the sheep to withstand the reduced infection with *T. colubriformis*.

During the course of the work a major complication appeared

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in that marked staining of the wool occurred, especially in the lambs, over the neck and shoulders. This staining, which would detract from the value of the fleece, is attributed to the fine particles of phenothiazine being blown into the fleece and should not be difficult to obviate. In this trial the salt-bone meal-phenothiazine lick was fed dry in shallow open troughs on the ground. In the 1959-1960 trial it is proposed to moisten the lick with a mixture of 10 lbs. of molasses and 10 lbs of water to each 100 lbs. of lick. This appears to be effective in binding the phenothiazine and it is hoped that very little staining will occur. Alternatively, the lick troughs could be housed under cover of a small shelter open on one side only. A further possibility, which is being investigated, is the use of a lick in pellet form.

Summary

1. The literature in regard to low-level phenothiazine administration in a lick has been summarised. In general it appears that where *H. contortus* is the dominant parasite, and sufficient lick is consumed, a high level of control is obtained. Where the system has not given satisfactory results, this has usually been due to inadequate consumption of the lick, in areas where a lick is not normally used.

2. The results of a trial carried out in the Eastern Transvaal highveld are reported. In this trial a high level of control of parasitism was obtained over an 11 month period in ewes and lambs. This was shown by low egg counts, high weight gains, and an absence of clinical disease in the treated flock, as compared with an untreated flock in which severe clinical verminosis occurred. The results indicate that the major effect is one of suppression of egg production and inhibition of larval development, and that a gradation in effectiveness against *T. colubriformis*, *H. contortus* and *O. columbianum* occurs, similar to that found with therapeutic dosing.

3. The only major disadvantage encountered was a marked staining of the wool on the head and shoulders, and suggestions are made for overcoming this difficulty.

Acknowledgements

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THE XVIth INTERNATIONAL VETERINARY CONGRESS MADRID, 1959

Dr. Alexander, who is our representative on the Permanent Committee of the World's Veterinary Congress, gave a very interesting and entertaining description of the proceedings.

He explained that these congresses were scheduled to take place every fourth year. The previous one was held in Stockholm in 1953 and the following one should have taken place in the Argentine in 1957. Owing to trouble in that country at the time, this was abandoned and another venue could not be found till 1959.

Practically every country in the world was represented and two new countries were admitted this year, namely Mexico and Peru.

The funds of the congress are derived from a levy of 1s. per member from each national organisation. In this case the Spanish government also contributed pound for pound. Over 2,000 members registered, the registration fee being £10.

The papers were divided into two categories: "Main Papers" and "Sectional Papers." The former were delivered in a large hall where simultaneous translation was provided. Each paper was allowed 30 minutes, 20 minutes for presentation and 10 minutes for discussion.

There were fifteen sections. Papers were presented by these sections simultaneously in fifteen different rooms. In all 540 such papers were delivered. Dr. Alexander very amusingly described the difficulties of trying to hear a given paper under such circumstances, especially when schedules became disorganised.

Brief Report of the meetings of the Permanent Committee for the International Veterinary Congresses, held in Madrid on 22nd, 26th and 27th May, 1959.

The following Members of the Bureau of the Permanent Committee were present: Professor W. I. B. Beveridge, President; Professor Dr. K. Wagener, Vice-President; Professor Dr. L. de Blieck, Deputy-Secretary; and Professor Dr. Jac. Jansen, General Secretary.

In total 22 countries were represented, viz.: Argentine, Australia, Austria, Belgium, Bulgaria, Canada, Czechoslovakia, Denmark, Finland, France, Germany, Hungary, Ireland, Italy, Japan, Netherlands, New Zealand, Norway, Peru, Portugal, Spain, Sweden, Switzerland, Union of South Africa, United Kingdom, U.S.A., U.S.S.A., Yugoslavia.

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The meeting was presided over by Professor W. I. B. Beveridge.

2. The Minutes of the Meeting of the Permanent Committee, held on May 22nd, 1958, were accepted.
2. The Report on the activities of the Permanent Committee during the year 1958, the Balance Sheet of the Secretariat of the Permanent Committee on the year 1958, and the Budget of the Secretariat of the Permanent Committee for the year 1959, were accepted.
3. The Report of the Congress Fund and the Financial Statement on the year 1957, were accepted.

   The minutes and reports sub 1, 2 and 3 were sent to the members of the Permanent Committee with a copy for the National Committees.
4. A proposal to alter the name of the International Veterinary Congresses to World Veterinary Congresses, to call the organisation built up in connection with these Congresses, the World Veterinary Association and the Permanent Committee the Permanent Committee of the World Veterinary Association was accepted.
5. The Constitution of the new Assocation was accepted unanimously.

   It was further agreed to draw up Rules based on the Constitution. A recommendation of the Congress Fund Committee to lay down in the Rules that persons from countries which are not contributing to the Congress Fund, must be obliged to pay a Congress fee of at least 50% more than people from contributing countries, was accepted.
6. Affiliation of International Associations of Veterinary Specialists and Intervet.

   As sections of congresses and for the formation of specialist associations the following headings, recommended by the planning committee, which was set up in the meeting of May 22nd, 1958, to study the affiliation of International Associations of Veterinary Specialists, were accepted:

   Anatomy
   Physiology, Biochemistry, Pharmacology (including Animal Behaviour)
   Zootechnics (including Nutrition, Animal Breeding and Production)
   Pathology (including Chemical Pathology)
   Infectious diseases (including Microbiology and Immunology)
   Parasitology (including Helminthology, Protozoology and Entomology)
   Hygiene of Animal Products (Veterinary Food Hygiene)
   Clinical Medicine
   Tropical Veterinary Medicine
   Surgery (including Anaesthesia and Radiology)

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Veterinary State Medicine and Veterinary Public Health
Small Animal Specialists
Avian Specialists
Veterinary Education
Professional interests.

As a result of a meeting, which the Bureau of the Permanent Committee and Dr. Wooldridge (member of the Planning Committee) had with the representatives of Specialist groups, a proposal was submitted to the Permanent Committee to establish a Scientific Programme Advisory Committee, consisting of the members of the Bureau of the Permanent Committee and one representative from each affiliated Specialist Association.

7. Honorary Members: A proposal to nominate Professor Dr. R. Manninger, Budapest, and Professor J. Verge, Alfort, Honorary Members of the World Veterinary Association, was accepted unanimously.
8. New Memberships: Formal applications for membership were received from Iran and Mexico. Both new members were welcomed.
9. Film Project: The Catalogue of Veterinary Films, prepared by the Secretariat of the Permanent Committee under the direction of Professor Dr. L. de Blieck, was shown. It would be distributed to the Members after the Congress. A motion of thanks to Professor de Blieck for the splendid work done, was carried unanimously. It was announced that further copies were available at the price of D.fl.5. — or its equivalent at the Secretariat of the Permanent Committee.

   The Film Committee of the Permanent Committee, which was at the same time the Standing Committee for the Veterinary Film of the International Scientific Film Association (I.S.F.A.), was re-elected.

   The next (XIIIth) I.S.F.A. Congress will be held in London and Oxford from 23rd September — 2nd October, 1959.
10. Seat and date of the next (XVIIth) International Veterinary Congress: A vote by ballot was taken, the result of which was that the next Congress will be held in Germany (at Hannover) in 1963.
11. A motion to offer Official Observer Status to O.I.E., W.H.O. and F.A.O. was carried unanimously.
12. Resolutions of the Congress. The Resolutions proposed by the Sub-sections of the Congress — in so far as they had been handed in in time — had been examined by the Resolution Committee, elected by the Permanent Committee, which consisted of: Sr. E. R. Alcázar, Dr. W. A. Hagan, Prof. R. Ferrando, Prof. Dr. R. Schmidt and Prof. Dr. Jac. Jansen. Of these Resolutions nine were accepted by the Permanent Committe. These Resolutions have already been sent to the members for publication.

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13. The list of representatives of members was accepted and con-

After the Closing Session of the Congress on May 27th, 1959, the Permanent Committee of the World Veterinary Association met for the first time to elect its Bureau.

The result of the election was:

Professor W. I. B. Veveridge: President.
Professor Dr. K. Wagener: Vice-President.
Dr. W. A. Hagan: Vice-President.
Professor Dr. L. de Blieck: Deputy-Secretary.

Professor Dr. Jac. Jansen, Secretary-Treasurer, whose office is permanent, remains in function.

"FUNGAL DISEASES OF ANIMALS"

by

J. C. Ainsworth and P. K. C. Austwick

Obtainable from any major bookseller or from:
Commonwealth Agricultural Bureaux,
Central Sales Branch, Farnham Royal,
Nr. Slough, Bucks, England.

Price: 20 shillings.

Although fungi have always been responsible for disease and death among domestic animals the study of these organisms has been somewhat neglected. At present, however, it is a rapidly growing subject, and since the existing literature on fungal diseases is widely spread in different journals, all mycologists welcome this concise text book. It is the first text book in English on this subject and is divided into 17 chapters each of which deals systematically with a group of diseases caused by related fungi. The very comprehensive bibliography makes this book a valuable addition to the liberary of research workers in the field of mycology. Practising veterinarians will find a solution to many obscure conditions in this book. The illustrations contained in the text are superb.

B.C.J.

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MINUTES OF THE 54th ANNUAL GENERAL MEETING, HELD AT ONDERSTEPOORT ON THE 27th AUGUST, 1959, AT 9 A.M.

PRESENT: The President (in the Chair) and 106 members of the Association

1. CONFIRMATION OF THE MINUTES OF THE 53rd ANNUAL GENERAL MEETING.

Dr. M. J. Meeser, seconded by Dr. J. R. Scheuber, moved that the minutes be confirmed. Agreed.

2. MATTERS ARISING FROM THESE MINUTES:

(a) Control of Faculty by the Department of Agriculture — Resolution (iv) of the 1957 Annual General Meeting. Resolved in 1958: On a motion of Professor J. H. R. Bisschop, seconded by Professor M. W. Henning, the meeting decided to instruct Council to reconsider this matter.

The President intimated that the resolution had been considered at the Council meeting immediately preceding Congress. Council had decided to appoint a committee, consisting of the President, three other members and a Faculty representative, to draw up a memorandum for presentation to the Minister of Agricultural Technical Services as a preliminary to requesting an interview to discuss this and other matters of significance to the profession. It was hoped this would eventuate before the next Annual General Meeting.

The meeting approved of the steps taken to date.

(b) Resolution regarding the castration, spaying or performance of any major operation on animals which have reached sexual maturity.

On Council's instruction this resolution had been published in the Journal and a notice was also sent to every member of the Association and to the Veterinary Board. — Approved.

(c) Resolution by Dr. L. W. v. d. Heever regarding the possible harmful effects of antibiotic contaminated milk.

The President reported that considerable progress had been made in this matter and that a deputation from the Association, together with a representative of the Division of Veterinary Services had interviewed the Secretary for Public Health. Details of this meeting would be published in the minutes of Council and it was believed that better control of the whole problem would be forthcoming in the not very distant future. — Agreed.

(d) Resolution by Dr. R. Clark in regard to the establishment of regional diagnostic centres.

No action had been required but the Hon. Secretary had discovered that a copy of this resolution had not been forwarded to the Director of Veterinary Services.

(e) Resolution by Professor K. v. d. Walt requesting the appointment of a special employment committee of Council.

Council had decided that its General Purposes Committee was performing the functions envisaged by this resolution. The President had recently visited Pietermaritzburg to interview the Natal Veterinary Co-op. regarding the conditions of employment and members were assured that everything possible was being done to explore venues of employment and safeguard conditions of service for veterinarians. — Agreed.

(f) Resolution by Dr. A. B. le Grange calling for investigation of the desirability of corrective operations for inherited defects in potential stud book calves.

Council had instructed Drs. S. W. J. van Rensburg and la Grange to compile an informative article for the guidance of the profession.

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Dr. van Rensburg informed the meeting of some of the difficulties being encountered in the preparation of this article for the Journal, but gave the assurance that the matter was receiving attention and that a geneticist would also be consulted when their material was complete.

(g) Resolution by Dr. A. J. Louw for the alteration of the Constitution to enable a full-time Secretary-Treasurer, who would also be Public Relations Officer, to be appointed.

This involved an increase of subscription and amendment of the Constitution, and would be dealt with by the Special General Meeting which would follow the Annual General Meeting.

(h) On the suggestion of Dr. A. M. Diesel the meeting agreed that a short report of the Trade Exhibition be published in the Journal and also be made available to the exhibitors.

3. MEMBERSHIP:

(a) Deaths. The death of five colleagues, not all of whom were members of the Association, were reported:

Drs. Cordy (Natal), I. Mowat (Johannesburg), G. Watt (Florida), Ben Runciman (Johannesburg), G. Carey (England).

The meeting stood as a token of respect. Condolences had been addressed to the bereaved families.

(b) Resignations: Dr. Roe, Tanganyika.

The Association had also lost track of one member whose correspondence had all been returned marked "address unknown." Dr. Lambrechts stated that he was able to supply the address of this member.

(c) New Members: Council had approved applications for membership received during the year from the following, and recommended their acceptance by the meeting: Drs. D. G. Clow, W. H. Chase, V. Caeiro, S. W. Bayer, C. T. McDonald, H. Hellig, W. J. Ehret, J. M. O'Grady, H. G. J. Coetzee, M. A. J. Azzie, R. Bothma, B. J. H. Barnard, C. J. Roos, J. P. J. Smit, I. Wolleschak, W. L. Jenkins, R. Clatworthy, H. N. van der Made, W. A. S. Brummer, R. R. van der Veen, D. S. Etherington, W. E. Galpin, H. Boedes, R. J. Bezuidenhout, K. L. Malkin, G. Bisschop, T. A. Theron.

Membership of the above was approved by the meeting on the motion of Dr. Kirkpatrick.

(d) Election of Honorary Associatio Member.

Council wished to recommend to the meeting the acceptance of Mr. P. M. Bekker as an Honorary Associate Member. On the motion of Dr. B. C. Jansen, seconded by Dr. L. C. Blomefield, the meeting unanimously approved of this recommendation.

The President congratulated Mr. Bekker and expressed his pleasure that the Association had seen fit to indicate its esteem in this manner.

(e) Life Members:

The Hon. Secretary announced that the following had become Life Members during the year: Drs. L. C. Blomefield, R. Clark, M. C. Robinson, J. A. Thorburn, B. M. Horwitz, W. O. Neitz, F. B. Wright, J. Zwarenstein and M. Zschokke.

These gentlemen were congratulated by the President.

4. CONSIDERATION OF REPORTS:

(a) Balance Sheet and Auditors' Report.

These had been circularised to all members prior to the meeting and had also been approved by the last Council meeting. The Hon. Treasurer pointed out that there was an excess of income over expenditure of £333.18.0 and that printing costs of the Journal had been reduced by £50.0.0 during the year. The amount of £303.9.9 was outstanding in subscriptions but it was expected that, with the exception of money owed by one member, the remainder of this amount would be collected in due course.

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At the end of the financial year the sum of £1,670.17.4 was owing to the Book Fund, and credit was now limited to £20 per person and no further credit was granted if an account had not been paid within 90 days of presentation. No serious bad debts were anticipated, but the Finance Committee had recommended to Council that the Book Fund be closed. The Prize Fund Reserve Account now stood at £1,882.5.6 and Council was considering how best to utilise this money to assist education, publication or the making of films of veterinary interest by members.

After Drs. Malherbe and Irwin had queried the desirability of abolishing the Book Fund, the President explained that Council had taken the decision to give up this Fund after careful investigation. The facts indicated that the Association was not justified in carrying the tremendous financial commitments and all the work involved for the very small benefits which accrued. The students did not get their books any cheaper than they would anywhere else.

On the motion of Dr. R. du Toit the Balance Sheet and Auditors' Report was accepted by the meeting. The President thanked the Hon. Treasurer for his loyal service to the Association during the year.

(b) General Purposes Committee.

Dr. R. du Toit, Convenor of this Committee gave the meeting a brief outline of the numerous matters dealt with during the year, most of which had been reported in the Council's minutes.

This committee also undertook the formidable task of organising the annual congress.

(c) Editorial Committee.

Dr. Clark reported that the supply of material had greatly improved during the year and costs had been reduced by judicious abbreviation of some articles. Council had recently approved of additional expenditure of £100 on the Journal during the forthcoming year. He appealed to members to maintain the flow of contributions to the Journal.

(d) Library Committee.

Dr. Clark reported that the library had been taken over by the University of Pretoria, and great progress had been made. The University had spent over £500 on text books for the library, which was now housed in the common room of the old hostel. It was expected that another £500 worth of text books would be acquired during the ensuing year.

The Division of Veterinary Services had also been very helpful. He was willing to arrange an exhibition of the latest text books as suggested by Dr. J. Dorè for the next Congress, provided these were insured by the Association. Dr. Clark also thanked the Association for the assistance rendered in obtaining journals for the library. These would be bound each year. An appeal was made to members to donate good books to the library, which was not only a technical library.

(e) Disciplinary Committee.

The Governor, Dr. P. S. Snyman, briefly indicated the activities of this committee during the year and explained that the functions of the committee were not yet clearly defined, especially its relationship to the Veterinary Board. This matter was still being clarified and legal advice had been sought.

5. NOTIFICATION OF COUNCIL AND ELECTION OF REPRESENTATIVE ON THE VETERINARY BOARD.

After the detailed results of voting had been notified the President announced the election of the following gentlemen to the offices shown:

Vice-President: Dr. R. du Toit.

New Members of Council: Dr. J. S. van Heerden, R. Clark, L. W. v. d. Heever and C. F. B. Hofmeyr.

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The constitution of Council for the ensuing year was:

President: Dr. H. P. Steyn.
Vice-President: Dr. R. du Toit.
Hon. Secretary: Dr. R. K. Loveday.
Hon. Treasurer: Dr. G. D. Sutton.
Members 1958/1960:
Dr. P. S. Snyman.
Dr. M. de Lange.
Dr. A. F. Tarr.
Dr. M. C. Lambrechts.
Members 1959/1961:
Dr. J. S. van Heerden.
Dr. R. Clark.
Dr. L. W. van den Heever.
Dr. C. F. B. Hofmeyr.
Hon. Life Vice-Presidents:
Dr. P. J. du Toit.
Col. J. A. Irvine-Smith.
Dr. A. C. Kirkpatrick.
Col. C. J. van Heerden.
Dr. R. A. Alexander.
Dr. E. M. Robinson.
Dr. H. H. Curson.
Dr. P. R. Viljoen.
Dr. G. v. d. M. de Kock.
Dr. S. W. J. van Rensburg.
Dr. J. Quinlan.
Dr. A. M. Diesel.

The President explained that under the new Constitution the Hon. Secretary, the Hon. Treasurer and the Editor of the Journal are co-opted to Council and no longer elected. Council had co-opted Dr. Loveday as Hon. Secretary with Dr. C. J. Muller as Assistant Secretary, Dr. Sutton was Hon. Treasurer and the new Editor would be Dr. H. P. A. de Boom with Dr. Clark as Co-Editor.

6. GENERAL:

(a) The President expressed the hearty thanks of the Association to the Medical Exhibitors' Association for their generous cheque of £100 towards the Benevolent Fund. This was an annual institution and was much appreciated. He also thanked the exhibitors for the fine service they rendered in keeping the profession abreast of new developments.

Mr. Goddin replied on behalf of the exhibitors and expressed thanks for the courtesy and hospitality received.

(b) The President conveyed the Association's thanks to Mesdames Klerck and Glover for their efficient services and presented them with tokens of appreciation.

(c) Professor K. van der Walt drew the attention of the meeting to an article which had appeared the previous day in "Die Vaderland" and read the article to the meeting. Professor van Rensburg stated that this article concerned the Presidential address and it was an unheard of thing to permit discussion of this address. The President thereupon ruled accordingly.

(d) Dr. DuCasse asked that more time for discussion be made available at future Congresses, as the present Congress had been rather rushed. The President gave the assurance that this would be done.

(e) Dr. G. C. van Drimmelen asked that the Congress programme be regarded as a publication and be printed on good paper for inclusion in the Journal. He also requested that one afternoon of Congress be set aside in future, for sectional meetings. The President explained that there was a resolution on these lines to be proposed under the next item.

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7. RESOLUTIONS:

(a) Proposed by Dr. B. M. Horwitz and seconded by Dr. R. Clark:
"That the annual conference of the S.A. Veterinary Medical Association be extended to four days and not three days as at present."
— Unanimously agreed.

(b) Proposed by Dr. B. M. Horwitz and seconded by Dr. P. L. Uys:
"That an afternoon during Congress be set aside for Group Meetings."
— Carried unanimously.

8. (a) The President thereupon informed the meeting regarding the proposed formation of a new organisation, to be called "The International Women's Auxiliary to the Veterinary Profession," and explained the objects of this body. Council would later circularise members with more detailed information, so that wives of veterinary surgeons could be asked whether they wished to form a branch of this organisation in South Africa.

(b) The next announcement was that the year 1962 had been declared Animal Health Year, and was also the 100th anniversary of the International Veterinary Congress. Dr. Alexander had raised the issue that it might be advisable to organise a specially big Congress that year to mark Animal Health Year.

In terms of a previous resolution, the Association was committed to holding a big Congress in 1960, which coincided with the Union's 50th Anniversary Celebrations. It was for members to decide whether they wanted specially big Congresses in 1960 and 1962 or only in 1962. The meeting unanimously decided that specially big congresses would be held both in 1960 and 1962, and that it would be left to Council to decide how long each Congress would be.

(c) In conclusion, the President paid warm tribute to the services rendered by Dr. S. van Heerden, the retiring Hon. Secretary, who had held office during the past four years of extremely great activity in the Association and had performed a herculean task. The Council had seen fit to donate an honorarium to him as a token of appreciation for what he had done.

MINUTES OF SPECIAL GENERAL MEETING HELD AT ONDERSTEPOORT ON THE 27th AUGUST, 1959, AT 11.30 A.M.

PRESENT: The President (in the Chair) and 106 members of the Association. This number constituted a quorum of more than one-fourth of the total membership of the Association.

The notice dated the 4th August, 1959, convening a Special General Meeting for the purpose of amending Articles 7 and 19 of the Constitution, had been sent to all members.

The President invited Dr. S. W. J. van Rensburg, Chairman of the Finance Committee, to propose the amendments to Articles 7 and 19.

Dr. van Rensburg explained that the decision to appoint a full-time Secretary-Treasurer, who would also be Public Relations Officer, had been taken by a resolution passed at last year's Annual General Meeting. Council had therefore

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instructed the Finance Committee to investigate the changes of Constituion necessary to implement this decision. It had been found impossible to finance a permanent secretary with the present income and therefore certain alterations were proposed. Such an official would require his own office, clerical assistance and travelling expenses in addition to a salary. Although £35 per month, presently paid to a secretarial firm would be saved, it was also necessary to raise the annual subscription to £5.0.0 and the cost of Life Membership to £75, or 40 annual subscriptions. A strong appeal was also made to Life Members and Hon. Life Vice-Presidents to make what contributions they could afford towards this new venture until they reached the age of 65 years. The proposal was formally made in terms of the above suggested amendments and seconded by Dr. J. S. van Heerden.

Dr. Edwards felt that it would be a mistake to lengthen the period for Life Membership from 30 to 40 years.

On a point of information Dr. van Rensburg elaborated the numerous functions and duties which such a full-time official would be called upon to perform.

After further discussion Dr. I. van Schalkwyk, seconded by Dr. I. McFarlane, proposed that the annual subscription be raised to £10 per annum so that more funds would be available to attract the right type of person for the position. This proposal was lost, as it did not gain the requisite three-quarters of the votes of those present required to amend the Constitution.

Dr. D. Shone asked that consideration be given to reducing the subscriptions of those members of the Association domiciled outside the Union.

Dr. van Schalkwyk thereupon proposed that the whole matter should be referred back to Council for re-consideration, but the President pointed out that this would have the effect of delaying the matter for two years.

A proposal by Dr. T. W.Naude, seconded by Dr. M. C. Lambrechts, that the annual subscription be increased to £7.10.0 as from 1st April, 1960, was put to the vote and carried by 83 votes in favour of, 21 votes against and 3 abstentions.

After further discussion, a proposal by Dr. Alexander, seconded by Dr. Ryksen, that Life Members should pay an annual subscription for 40 years or until attaining the age of 65 years, was voted upon and carried with only 3 dissentient votes.

On the proposal of Dr. Alexander, seconded by Dr. Snyman, it was also agreed by the same majority as for the previous resolution, that the lump sum payment for Life Membership would be a figure laid down actuarially.

Dr. van Rensburg then read the new Articles 7 and 19 which had been agreed upon by the meeting as follows:

"Article 7: Life Members shall comprise every person who at the date of the adoption of these Articles, is on the Register as a Life Member of the Association, and every person admitted as an Ordinary Member thereafter who has paid a single subscription of £ ..... (to be decided by the actuary) to the Association, or who has paid 40 annual subscriptions as an Ordinary Member or has attained the age of 65 years. Life Members shall be entitled to the same privileges of membership as Ordinary Members."

"Article 19: "An Ordinary Member shall pay, on election, the sum of £7.10.0 (Seven pounds ten shillings) which shall be the first Annual Subscription. After expiration of the first year or portion thereof, an annual subscription of £7.10.0 (Seven pounds ten shillings) shall be payable by all Ordinary Members. The annual subscription is due and payable annually as of the 1st April in each year and shall be paid not later than the 1st May of that year."

Dr. Diesel informed the meeting that the request for a reduction in annual subscription for members resident outside the Union could be considered by Council, which was empowered by the Constitution (Article 19) at its discretion to suspend the payment of the annual subscription or any portion of it, of any individual member as it may determine. This power solved the whole problem.

This concluded the Special General Meeting.

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OBITUARY

MAJOR J. J. G. KEPPEL

The death of Major John Joseph Guiney Keppel, O.B.E., M.R.C.V.S., occurred in Cape Town on October 1st, 1959. He was 75 years of age.

Born in County Cork in 1884 he qualified at the Royal Veterinary College, Dublin, in 1911. He was a keen sportsman and played rugby for Ireland from 1905 to 1912. He was also an oarsman and polo player.

He came to south Africa in 1912. During World War I he served with the Royal Army Veterinary Corps and with the 12th Lancers. He was awarded the O.B.E. and was four times mentioned in despatches.

He was stationed in many parts of the Union, including the Transkei, Potchefstroom, Middelburg (Tvl.), Piet Retief, Dundee and as Sub-Director of Veterinary Services at Bloemfontein and Cape Town. During his service he dealt with many epizootic diseases such as Rabies, Foot and Mouth Disease, Swine Fever, Sheep Scab, Dourine and Horse Sickness.

He retired in 1944 and a year later purchased the old historic farm of Spier Lyndoch, near Stellenbosch. Here, with his son, he farmed wine, tobacco and Jersey cattle.

He leaves a wife, a son and a daughter. To them we extend our heartfelt sympathy.

A.M.D.

THE ROYAL SHOW

The Royal Show in 1960, organised by the Royal Agricultural Society of England, 35 Belgrave Square, London, S.W.1, will take place at Cambridge (about fifty miles from London) from the 5th — 8th July.

Its Council cordially invites overseas visitors engaged in farming, agricultural administration, research and education, and if they can let us know their addresses IN ENGLAND, guest tickets giving free admission to the Show and the Overseas Pavillion will be forwarded towards the end of June next.

As guest tickets are intended for agriculturalists resident overseas (and their wives) it would be helpful if any reply to this invitation could give the names and addresses in England of those on whose behalf application is made, with some indication as to their connection with agriculture. Also, it would be a help if the Secretary could be informed of the days on which visitors hope to attend, if this is known.

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TRADE EXHIBITS

The undermentioned firms staged exhibits at the 54th Annual Congress of the Association and amongst others showed the following Veterinary pharmaceuticals, instruments and appliances.

FIRM	Representative	EXHIBIT
MAYBAKER (S.A.) (PTY.) LTD. P.O. Box 1130, Port Elizabeth.	Mr. Goddin G. Holmshaw	"AGAVIN" — Intra uterine injection for *uterine infections* "ROVAMYCIN" — Intra mammary injection — *bovine mastitis* "STRINACIN" — Triple sulphonamide (Sulphapyridine, Sulphamerazine and Sulphadiazine with Streptomycin — *Scours in calves and pigs.*
BURROUGHS WELLCOME & CO. P.O. Box 10293, Johannesburg.	L. M. Day	"FIOVAX" — Vaccine — *Feline infectious enteritis* "TEMADEX" — *Skin dressing* — Sarcophic and Otodectic Mange in dogs — Otodectic mange in cats.
E. J. REED (PTY.) LTD. (Hanger and Vessa) 79 De Villiers Street, Johannesburg.	F. W. Dickson	"TUBEGAUZ" — Bandages and applicators (metal rings) of different sizes — *for neat and easy bandaging* of tails, legs, paws, heads, etc., of small animals (and fingers of operators).
DATONS SOUTH AFRICAN NATIONAL STOCK REMEDIES (PTY.) LTD. P.O. Box 194, Nigel, Tvl.	B. Gamsu	"SPIROTRYPAN forte" — Treatment of anaplasmosis "BERENIL" — Treatment of babesiosis in cattle and dogs "HOSTACYCLINE" — Antibiotic
PFIZER LABORATORIES (S.A.) (PTY.) LTD. P.O. Box 7324, Johannesburg.	B. Dixon	"TERRAMYCIN A.D." — *Scour tablets* for treatment of scours in domestic and other animals. "TERRAMYCIN" — *Injectable solution* — Treatment of Heartwater, Gallsickness, etc. "EMBRYOSTAT" — Intra-vaginal and intra-uterine injection — an aid to increased conception.
PARKE DAVIS LABORATORIES (PTY.) LTD. P.O. Box 9971, Johannesburg.	K. Quarmby	"CHLOROMYCETIN SYCCINATE" Antimicrobial agent for intravenous Intramuscular and subcutaneous injection. "CHLOROMYCETIN TINCTURE" — contagious footrot and other suppurative conditions. "CHLOROMYCETIN OPHTHALMIC OINTMENT"
SCHERING CORPORATION (SCHERAG (PTY.) LTD.) 127/129 Anderson St., Johannesburg.	D. Langbridge	"TRILAFON" — Tranquilizing drug "AZIUM" — potent corticosteroid
AGRICURA LABORATORIA BPK. Posbus 55, Silverton.	E. Consul	'n Reeks instrumente wat vir die praktiserende veearts noodsaaklik is, en 'n aantal middels soos byvoorbeeld: "INTORVIT" — 'n oplossing van vitamien A in olie vir binnespierse inspyting "DELTASOL" — 'n Delta BHC watermengbare olie vir die dip van skape "AGRICURA OOGPOEIER" — vir aansteeklike en ander oogontstekingss
RECKETT COLEMAN (AFRICA) Priestman House, Rosebank, Cape Town.	J. G. Strachan	"DETTOL" — Instrument sterlization "DETTOL CREAM" — Antiseptic "212 DISINFECTANT"
GLAXO LABORATORIES (S.A.) (PTY.) LTD. Manchester Road, Wadeville, Tvl.	E. O. Warnes	"GRISOVIN" — Treatment of ringworm, dogs and cats "TRIPLOPEN" — Three penicillins giving high blood level and long sustained effect "BIOTEXIN CERATE" — Bactericidal — bovine mastitis

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FIRM	Representative	EXHIBIT
SOUTH AFRICAN CYANAMID (PTY.) LTD. P.O. Box 7552, Johannesburg.	N. L. Butler	"AUREOMYCIN" — Soluble powder — Heartwater and anaplasmosis "VARIZYME" — for liquification of fibrin and vicious exudates — abscesses, cysts and ulcerations "TARGOT" — Mastitis ointment
ALLEN HANBURY'S (AFRICA) LTD. P.O. Box 1710, Johannesburg.	C. T. R. Schnetler	"DEQUADIN" — Wound-powder "SURGICAL SUPPLIES"
IMPERIAL CHEMICAL INDUSTRIES (S.A.) LTD. P.O. Box 11270, Johannesburg.	P. L. Lewis	"DICTYCIDE" — Elimination of lungworms "HABITANE" — Antibacterial agent
GOLDFIELDS VETERINARY MEDICAL SUPPLIES 144 Hay Street, Turffontein, Johannesburg.	E. Feinberg	"NEO-SULPHENTRIN" — Treatment of Infectious Enteritis in all species "RYOTIN" — Treatment of Canker (Otitis) "NEOVAGON" — Treatment of vaginal infections
A. S. RUFFEL (PTY.) LTD. P.O. Box 7824, Johannesburg.	B. Kaplan	"NITROPEN" — Treatment Mastitis Comprehensive range of Veterinary instruments and pharmaceuticals
MILBORROW & CO. (PTY.) LTD. P.O. Box 216, Pietermaritzburg.	G. Milborrow	VETERINARY INSTRUMENTS and APPLIANCES
CHAS. F. THACKRAY (S.A.) (PTY.) LTD. P.O. Box 2726, 235 Bree Street, Johannesburg.	B. Conway	SURGICAL INSTRUMENTS, SUTURE MATERIAL and STERILIZERS
TAUBER & CORSSEN (PTY.) LTD. P.O. Box 1366, Johannesburg.	F. M. Thornton	"LEITZ" Microscopes, Cameras, Projectors "GALLENKAMP" Glass fronted over and muffle furnace
B. OWEN JONES in association with MAC DONALD ADAMS & CO. 27 Fraser Street, Johannesburg.	P. Tyler	"EMIL" Greenline and Goldline glassware "B.D.H." Chemicals: Memmert ovens "M.S.A." Centrifuges and wide range of laboratory equipment
OPTICAL INSTRUMENTS (PTY.) LTD. P.O. Box 1561, Johannesburg.	T. Steeg	ZEISS TELEVISION Microscope and other microscopes Wide range of Medical and Veterinary technical instruments — Zeiss Cameras, field-glasses
PROTEA HOLDINGS LTD. P.O. Box 7793, Johannesburg.	A. M. Enzelhofer A. P. Steyn	Miniature camera for photomicrography Chemical and physical laboratory equipment
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RETURN OF OUTBREAKS OF SCHEDULED DISEASES IN THE UNION OF SOUTH AFRICA DURING THE PERIOD 1/6/1959 TO 31/8/1959.

DISEASE	CAPE	NATAL	O.F.S.	TRANSVAAL
Anthrax	1 Mafeking	1 Msinga	1 Bethlehem 1 Bloemfontein 1 Boshof 1 Lindley 1 Reitz 1 Senekal 1 Ventersburg	1 Klerksdorp 1 Lichtenburg
Bacillary White Diarrhoea		1 Estcourt	1 Blomefontein	1 Brits 1 Pietersburg 1 Zoutpansberg
Epivaginitis				1 Middelburg
Foot and Mouth Disease				1 Barberton 1 Pilgrimsrest
Fowl Cholera	1 Vredenburg			
Fowl Typhoid	1 Alexandria	1 Pinetown	1 Kroonstad	1 Pretoria 1 Rustenburg
Lumpy Skin Disease	1 Bellville 6 Flagstaff 2 Lusikisiki 1 St. Marks	1 Nongoma	1 Bethlehem 2 Ficksburg 1 Jacobsdal 1 Wesselsbron	1 Bronkhorstspruit 1 Heidelberg 3 Pietersburg 1 Potchefstroom 1 Potgietersrus 1 Rustenburg 1 Ventersdorp 1 Wolmaransstad 2 Zoutpansberg
Mange (Goat)	3 Elliotdale 3 Glen Grey 2 Idutwa 3 Kentani 9 King William's Town 3 Middeldrift 4 Mqanduli 1 Nqamkwe 1 Peddie 2 St. Marks 1 Tsomo	3 Highflats 1 Hlabisa 1 Inguavuma 1 Ndwedwe		
Mange (Equine)	4 King William's Town 1 Qumbu 4 St. Marks 2 Umtata 1 Willowvale			
Mange (Bovine)	1 Middeldrift 1 Wynberg			1 Lydenburg
Mange (Porcine)	2 Middelburg	1 Ixopo		
Rabies	3 Vryburg (civet cat, meercat, dog) 1 Warrenton (bovine)		2 Boshof (dog, bovine) 2 Bultfontein (bovine, cat) 1 Hoopstad (duiker) 1 Kroonstad (dog) 1 Senekal (meercat) 1 Theunissen (dog)	1 Bloemhof (bovine) 7 Letaba (6 dogs, 1 bovine) 1 Schweizer Reneke (meercat) 1 Wolmaransstad (meercat) 3 Zoutpansberg (3 dogs)
Sheep Scab	2 Mafeking			
Trypanosomiasis (Dourine)	1 Kuruman			
Tuberculosis (Avian)	1 Vryburg 1 Bredasdorp		1 Bloemfontein	
Tuberculosis (Bovine)	1 Cape Town 1 East London 1 Malmesbury 1 Taungs 1 Vredenburg 1 Wynberg	1 Kranskop 1 Lions River 1 Ndwedwe 1 Inanda 2 Pietermaritzburg 2 Pinetown	1 Lindley 1 Parys	2 Marico 1 Brits 4 Klerksdorp 1 Pietersburg 1 Potchefstroom 4 Potgietersrus 1 Warmbad 1 Zoutpansberg

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EDITOR'S NOTE

This issue contains the rest of Congress papers submitted for publication as well as the discussions and reply thereto.

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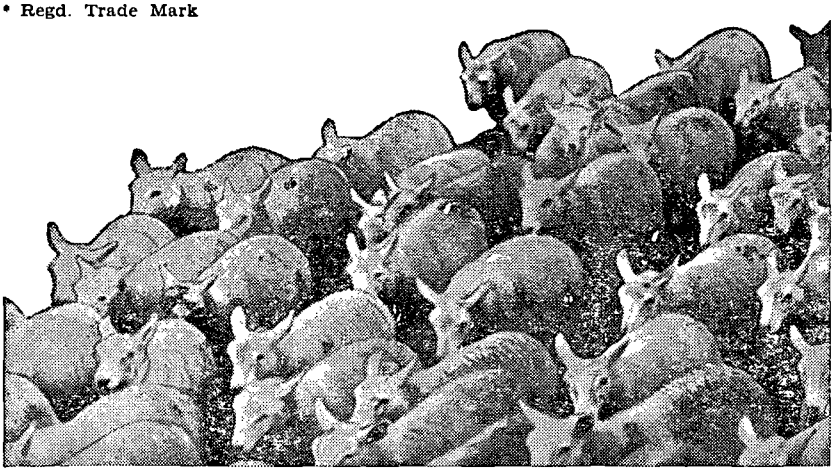
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1. Aust. Vet. Jl. 1954, 30:209.

2. Vet. Rec. 1955, 67:74.

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DIPSTOF EN DIPPING

'n Oorsig oor die Aard en Gebruik van Dipstof vir die Bestryding van Bosluise op Beeste, vir die tydperk 1889 *tot* 1959 *in Suid-Afrika*

P. M. BEKKER

('n Voordrag gegee voor die 54ste Jaarkongres van die S.A.V.M.V. te Onderstepoort, 25—27 Augustus, 1959)

Alhoewel die gebruik van dipstof eerstens bedoel is om uitwendige parasiete op diere te bestry is dit hoofsaaklik van waarde om, wat beeste betref, siektes te beheer.

Vanaf die vroegste Bybelse tye, Levitikus XXII, 22 (1490 V.C.), word bepaal dat 'n dier wat skurfte het nie as offer gebring mag word nie. Alhoewel die skurfte of brandsiekte en seer sekerlik ook bosluise van die vroegste tye van die bestaan van die dier bekend was, is skynbaar geen georganiseerde bestrydingsmetodes aangewend nie. Individue het waarskynlik persoonlik probeer om van peste ontslae te raak. So is in die tyd van Van Riebeeck, aan die begin van die tweede helfte van die 17de eeu, bepaal dat wagters spesiale aandag moes skenk aan brandsiekte en dat skape wat besmet is dadelik uit die kudde verwyder en geslag moes word.

Volgens Veld (1952) (Cooper & Nephews-publikasie) is skape omstreeks 1810 vir die eerste keer in aangemaakte insektegif gedip. Hierdie deur die boer self bereide dipstof, was dikwels gevaarlik. Dit, skynbaar, het aanleiding gegee dat William Cooper in 1843 'n arseenswawelskaapdip ontwerp het, wat vandag nog in 'n ietwat gewysigde vorm gebruik word en as Coopers se skaapdippoeier bekend staan.

Wat die presiese samestelling van die dipstowwe in die begin was, was nie altyd duidelik nie en waarskynlik om hierdie rede het, volgens die Jaarverslag van die Sekretaris van Landbou (1890-91), die Kaapse Parlement besluit dat die samestelling van dipstof (destyds natuurlik nog net vir skape) bekend gemaak moes word voordat dit erken en op die goewermentslys geplaas kon word. Hiervolgens is die volgende samestellings bekend gemaak.

1. „Cooper's Dip": 37% As_2O_3 teenwoordig as arseen, arseensulfiede en swawel.
2. „Little's Dipping Powder": 23% As_2O_3, ook teenwoordig as arseen, sulfiede en swawel.
3. „McDougall's Dip": Bevat slegs 1.03% As_2O_3.

In 'n pamflet (1891-92) van die Departement Landbou, van die V.S.A. word formules aangegee om insekte op vrugtebome te bestry.

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1. Keroseen-emulsie: 2 gl. keroseen (paraffien); ½ lb. seep; 1 gl. water. Afdunning: 1 in 10 tot 25.
2. Harpuiswasmengsels:

	I	II	III
Harpuis	18	30	40 lb.
NaOH	5	9	5 lb.
Visolie	2½	4½	— pt.
Water na	100	100	50 gl.

3. Arseenpreparate: Witarseen (As_2O_3), arseniete, Parysgroen, ens. 1 lb. op 100 na 250 gl. water. Vir As_2O_3, 1 lb. op 50 gl. water.

Ek verwys na bogemelde vir bome-bedoelde-spuitmiddels en ne skaapdipstof omdat dit die basis van latere beesdippe gevorm het. Op die stadium was bogemelde, veral die witarseen (As_2O_3), bekend en ook gemruik om sprinkane uit te roei. Die arseen is met bytsoda gekook om dit in oplossing te bring. Verder wil dit voorkom asof dit ook oorspronklik gebruik is om tuisgemaakte skaapdipstof te berei. Selfs is net As_2O_3 in water gekook en was meeste As_2O_3 nie eers in oplossing nie. (Natal Agric. J. Band 11, blds. 404.)

Die geskiedenis van dipping in Suid-Afrika oor die afgelope 70 jaar kan gerieflikheidshalwe in drie periodes van 20, 30 en 20 jaar verdeel word. Die wordingsjare voor 1910 het afgesluit met die werk van Watkins-Pitchford. Die tweede periode, tot ongeveer 1940, kan beskou word as 'n tydperk waarin slegs voortgegaan is met 'n reeds geformuleerde beleid om bosluise sistematies te bekamp, terwyl die laaste twintig jaar as die bestandeblouboslu:stydvak, waarin die nuwere insektemiddels te voorskyn getree het, beskou kan word.

1. Die Eerste Tydperk: 1889-1910

Die geskiedenis van die beginjare kan gevind word in drie gebiede, nl., Ciskei (Grahamstad), Natal en Transvaal en is respektiewelik gekoppel aan drie siektes, nl., Hartwater, Rooiwater (Piroplasmose) en Ooskuskoors (Teileriose) en aan die bestryding respektiewelik van drie soorte bosluise, nl., bont- (*Amblyomma hebraeum*), blou- (*Boophilus decoloratus*) en die bruinbosluis (*Rhipicephalus appendiculatus*), asook aan drie persone (twee boere en 'n veearts), nl., Arthur Douglas (Heatherton Towers, Grahamstad), Joseph Baynes (Nel's Rust, Richmond, Natal), en H. Watkins-Pitchford (Veearts, Pietermaritzburg).

Veral moet die twee boere beskou word as die baanbrekers op gebied van bekamping van bosluise terwyl Watkins-Pitchford vir die dipproewe in dié verband gesorg het.

A. *Bosluisbetryding in die Ciskei (Grahamstadgebied)*

William Rogers (1889-90), soos andere ook voor hom, verwys na die feit dat die gebiede in die Ciskei, wat vroeër uitstekende streke vir skape was, nou waardeloos geword het weëns, wat hy noem, galsiekte en hartwater. Aan hom was dit bekend dat diere

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in 1860 aan dieselfde siekte in Peddie dood is. In sy dagboek, onder datum 24-7-1865, het hy die volgende geskryf: „Two sheep died of water round the heart." Volgens hom het die siekte stadig binnenslands versprei. Baie het die siekte aan oorbeweiding, giftige plante en so meer toegeskryf.

In 'n lesing voor die Cradockse Boerevereniging (18-7-1891) verwys mnr. Henry Ogilvie ook na swaar verliese wat hy gely het as gevolg van hartwater onder sy skape op sy plaas Graskop (nou Grasslands) Grahamstad in 1863-64. Ook verwys hy na skaapdipbakke wat ongeveer dié tyd in gebruik gekom het, nadat 'n mnr. P. W. Court 'n besoek aan Australië gebring het en volgens hulle model hier 'n dipbak gebou het. Voor die tyd, volgens Ogilvie, is in 1857 met 'n uitbreek van brandsiekte gebruik gemaak van „a box, 6'x3', to wash sheep" wat 3 dae geneem het om 1,000 skape te behandel.

Met betrekking tot vrektes van skape, lê mnr. John Webb op 23-12-1877 getuienis voor die „Cattle Diseases Commission" in Grahamstad af en spreek die opinie uit dat die siektes voorgekom het vandat die bontbosluis 8 of 9 jaar vantevore tot daar versprei het. Hy het die bosluis 14 of 15 jaar tevore (1863) vir die eerste keer gesien. Hy het ook meegedeel dat mnr. William Bowker hom vertel het dat hy 40 jaar tevore ($\pm$1837) 'n bontbosluis van 'n koei afgetrek het wat van Soeloeland af gekom het.

Nadat skaapdipbakke in gebruik geneem is en meer boere die bontbosluis as 'n vyand beskou het, is dit nie buitengewoon om van J. B. Hellier (1890-91) te verneem dat kalwers op die plaas Chumie, Alice, in dieselfde dipbak as skape gedip is nie. Dit is dan ook volgens die geskiedenis die eerste geval van dip van beeste om bosluise te betry. Dit is uitstekend gevind om vrektes te voorkom, gevolglik het veearts Hellier dan ook voorsien dat hierdie plan van die boer nog in die toekoms toegepas sou word om bsluise aan die kus te bestry, waar hulle 'n groot probleem geskep het. Waar die boer skynbaar in 'n arseendipstof gedip het, het Hellier kalkswawel aanbeveel om kalwers te was waar daar nie 'n skaapdipbak was nie. Ook ander verwysings na bosluise op skape wat vrek, maar dat vrektes ophou as die skape gedip word, het verskyn.

In Band 4, „Agricultural Journal of the Cape of Good Hope", blds. 202 (1891-92) het die eerste berigte in S.A. verskyn oor die klassieke werk van Smith en Kilborne (1893). Hulle het in 1889 proewe uitgevoer waardeur hulle beide die bloedparasiet vasgestel het sowel as die feit dat die bloubosluis (*Boophilus annulatus*) die draer is van die siekte „Texas fever" (Rooiwater, Piroplasmose), in Amerika.

Wat bestrydingsmetodes betref, gee D. Hutcheon, „Colonial Veterinary Surgeon" in 1897 'n aanduiding dat dr. John Connaway van Missouri 'n dipbak laat bou het op die prinsiep van die skaapdipbak, maar net groter, en hierin is verskillende stowwe getoets, onder meer arseen en paraffien-emulsies. Laasgenoemde stof is

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volgens ander verwysings in die begin taamlik algemeen gebruik.

Na aanleiding van die toediening van paraffien deur middel van spuitpompe op plante en die gebruik in dipbakke om bosluise te bestry deur die Amerikaners, het boere ook in die Grahamstadgebied diere met paraffien begin bespuit om die bontbosluise te probeer bestry. Hulle was van mening dat die bespuiting goedkoper as dipping sou wees. In die verband het die boer Arthur Douglas (1898) die voortou geneem en 'n spuitgang ontwerp, asook 'n spuitpomp, waarmee 'n mengsel (soos verlang) van paraffien en water, gesuig uit twee houers, op die dier gespuit kon word. Op een stadium was hierdie metode selfs tot in Natal in algemene gebruik en is die Douglas Bespuitingsmetedo met die dipbak vergelyk. Die wetenskaplike C. B. Lounsbury verwys in 1902 na die verskeidenheid van stowwe wat op 'n bloot toevallige en onsamehangende manier deur boere gebruik is om bosluise te probeer bestry. Om 'n idee te kry van alle moontlike en onmoontlike stowwe, wat persone geprobeer het, gee 'n opsomming van 'n lesing deur Lewellyn Roberts, Cottlesbrook Creamery, Adelaide (1899) 'n goeie deursnit van wat alles gebruik is. Lounsbury het skynbaar heelwat proewe saam met Roberts uitgevoer.

Die man wat uit hierdie chaotiese toestand orde probeer skep het, was Arthur Douglas, Heatherton Towers, Grahamstad. Hy staan dan ook bekend as die eerste persoon in Suid-Afrika wat bosluise op 'n sistematiese manier probeer bestry het. Hy het naamlik in 1896 besef dat hy iets moes doen om bosluise effektief te bestry of om beesboerdery te staak. Hy het al die verskillende metodes probeer, selfs 'n dipbak gebou, maar op sy spuitgang en paraffien besluit. Volgens Lounsbury het Douglas nie geweet dat paraffien reeds in Amerika vir die bestryding van bosluise gebruik is nie. Douglas het dan ook in 1898 'n demonstrasie gegee van sy bespuitingsmetode. Die Douglas-spuitgang is later opgevolg deur die van L. J. Roberts, Cottlesbrook, Adelaide. Douglas se mengsel wat op die dier gespuit was, het 25% paraffien, dié van Roberts 20% en dié van Roberts en Lounsbury 15% bevat. Ongeveer 10 gl. paraffien is per 100 beeste gebruik en die diere is elke 12 na 14 dae behandel. Die metode was tot so 'n mate suksesvol dat Douglas en andere kon voortgaan met beesboerdery.

Skynbaar het Lounsbury (1900) en Roberts na aanleiding van sukses met Cooper se Skaapdippoeier, waarna voorheen verwys is, met oplosbare arseen en seep proewe uitgevoer, dit ook na aanleiding van die sogenaamde sukses met soortgelyke dipstof (Archer se Dip — 1899) in Queensland. Hulle het gebruik gemaak van witarseen (As_2O_3), nl., 8 onse en 2 onse seepsode (NaOH), m.a.w. die samestelling van wat later arseniet-van-soda geword het, en 'n 8-ons-steen seep. Die arseen en soda is gekook, by die seepoplossing gevoeg en opgemaak na 4 gl. (1.25% As_2O_3), 6 gl. (0.83%) en 8 gl. (0.625%). Die dipstof is aan die agterkant en onderkant aan diere toegedien. Die eerste twee konsentrasies het kwaai branding veroorsaak maar laasgenoemde het nie

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nadelige gevolge gehad nie en tog al die bosluise gedood. Ongeveer dieselfde tyd het Roberts ook proewe met Archer se dip ($1\frac{1}{2}$ lb. As_2O_3, 3 lb. soda, 3 lb. seep, 100 gellings water) op dubbelstrekte (0.30% As_2O_3) uitgevoer en gevind dat dit slegs die helfte van die bosluise dood. Skynbaar was hulle te haastig vir resultate. Op die stadium het alle aanduidings bestaan dat arseen, 'n goedkoper dipstof s paraffien, 'n oplossing van die bontbosluise en daarmee van die hartwaterprobleem, sou bied.

Alhoewel die boere algemeen van mening was dat hartwater deur die bontbosluise oorgedra is, het Lounsbury (1900/B) eers in 1899 in proewe aanduiding in dié rigting gekry en dit in 1900 bewys.

Verdere werk is gedoen deur Lounsbury (1905) in Rosebank (K.P.) gedurende Desember 1904, wat beskou kan word as 'n voortsetting van werk wat hy saam met Roberts van Fort Beaufort gedurende 1899-1900 gedoen het, toe hulle gevind het dat arseniet-van-soda („Scrub exterminator") met of sonder seep doeltreffend is om bosluise te bestry. Hy was skepties teenoor die waarde van seep en stockholmteer in arseen-dipstof, soos bv. in Demuth se dip, ens., teenoor arseniet-van-soda. In proewe het hy dan ook bewys dat die effek van die dipstof geheel-en-al van die arseengehalte afhanklik was.

Hy het ook die effek van arseen op die uitbroeivermoë van eiers nagegaan, sowel as sy afwerende waarde. Hy het gevind dat bloubosluislarwes wat twee dae na dipping op arseengedipte (0.13-0.15% As_2O_3) diere geplaas is, almal binne 'n paar dae vrek, so ook die meeste wat na 4 dae op beeste gesit is, maar skynbaar was die mortaliteit van dié wat na 7 dae op diere geplaas is, nie hoog nie.

Ook het hy nagegaan wat die toksiese effek van bespuiting van diere was: 3.3% As_2O_3 het die dood van twee diere binne 48 uur veroorsaak, terwyl 0.27%, vyftien keer met weeklikse tussenposes toegedien, geen effek gehad het nie. Die effek op skape, wat nogal maklik vrek, is ook nagegaan.

Die feit dat tot dusver net na hartwater en die bontbosluis in die gebied verwys is, meen nie dat Rooiwater nie ook 'n probleem was nie. Dit was wel die geval vanaf 1883, toe die siekte so ver as Oos-Londen en King William's Town versprei het, nadat dit al in 1870 aan die Tugela waargeneem is, en met die transportroetes versprei het [volgens Hutcheon (1900) en andere]. Hartwater en die bestryding van die bontbosluise was egter 'n groter probeelm in die Grahamstadgebied.

B. *Die Begin van Dipping in Natal en Transvaal*

Waar hartwater die probleem in die Ciskei was, was rooiwater weer in Natal 'n eersterangse moeilikheid, veral omdat baie stoet-beeste en veral hulle ingevoer is om kuddes te verbeter. In die verband kan veral Joseph Baynes as die pionier geskou word.

Die begin van dipping in Natal is tot 'n groot mate gekoppel

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met verwikkelinge in die verband in Australië. So word verwys deur P. R. Gordon (1898) na die groot uitgawes met die bou van dipbakke en aankoop van olie in 1896 — skynbaar was net, of veral, olie toe gebruik.

In aanmerkings in dr. Tidswell (1901/2) se rapport oor „Tick fever" of rooiwater aan die goewerment van New South Wales, verwys hy na tussenposes van 8 weke tussen dippings en na samestelling van dipstowwe veral met insluiting van arseen. Sover as wat aan hom bekend was, is nog geen sistematiese proewe in verband met dipping in Australië gedoen nie. P. R. Gordon (1901/2) verwys egter in dieselfde band na diptoetse met beeste op 13 Augustus (skynbaar 1900) waar 22 beeste in Quibell se dipstof (1 op 60) gedip is en waar alle bosluise 10 dae later dood was. Dit was 'n arseendipstof, maar geen sterkte word aangegee nie.

In 'n onderhoud het mnr. Booker (1901/2), 'n Queenslandse boer, wat saam met 50 beeste, wat daar aangekoop is, na Natal gekom het, blyk dat feitlik almal in Australië Christian se dipstof gebruik. Dit het bestaan uit: 6 lb. arseen (As_2O_3), 24 lb. wassoda, 24 lb. seep, 5 gl. stockholmteer en 400 gl. water, die hele volume gekook vir 6 uur (gee 0.15% As_2O_3). In Band 5, Natal Agricultural Journal (1902), blds. 249 word na Christian se dip verwys as bevattende 8 lb. arseen en op blds. 171 dat Queensland nou 'n standaard dip aanvaar het, nl., 8 lb. arseen, $4\frac{1}{2}$ lb. bytsoda, 8 lb. harde vet en $2\frac{1}{2}$ gl. stockholmteer in 400 gl. water (=0.20% As_2O_3).

Omdat mnr. Joseph Baynes alreeds baie verliese gely het deur ingevoerde stoetvee aan rooiwater te verloor, het hy in Januarie 1902 'n dipbak op sy plaas Nel's Rust volgens planne voorsien deur bogemelde mnr. Booker (1902) gebou. Dit was die eerste dipbak in Suid-Afrika en het dipping in arseen ingelei. Die dipbak was die „inglytipe". Die boom van die bak was ongeveer $14\frac{1}{2}$ vt. en op die oppervlakte ongeveer 38 vt. lank, met n wydte van $2\frac{1}{2}$ vt. onder en $3\frac{1}{4}$ vt. bo, met 'n totale diepte van 8 vt., waarvan die regaf gedeelte aan die einde van die skuinsingang $5\frac{1}{2}$ vt. was, met 'n kapasiteit van ongeveer 3,500 gl. Dit was van 'n dak voorsien. Die dipstof gebruik was die Christiantipe met 6 lb. arseen (=0.15% As_2O_3). Hier, net soos in Australië, is die dipstof in hoeveelhede van 400 gallon gekook en is dit veronderstel dat die dipstof op 'n temperatuur van 100°F gebruik behoort te word. Hulle het ook eers paraffien uitgetoets maar dit onbevredigend gevind. Later het S. B. Woollatt (1903) „Principal Veterinary Surgeon", aanbeveel dat 8 lb. in plaas van 6 lb. As_2O_3 in die formulasie gebruik word om dus 0.20% As_2O_3 te gee.

Kopieë van hierdie plan met besonderhede is deur Geo. D. Alexander (1902) (Baynes se bestuurder), opgestel vir die Kaapprovinsie en vir Rhodesië. Uit ander geskrifte blyk egter dat die Landboudepartement van die Transvaal op een stadium die

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bou van dipbakke as te duur, en die kook van die dipstof in 400-gallon-hoeveelhede as onprakties beskou en die afgekeur het. Later, in 1903 [„Transvaal Agricultural Journal", 2 (5), blds. 256] word verwys na die aanbou van dipbakke deur die Departement en aankoop van spuite („Sprinklers for sprinkling cattle").

Die dipping op Nel's Rust het sulke goeie uitwerking gehad dat heelwat vooraanstaande boere ook dipbakke begin bou het. Ook het dippe verskyn, soos Qu.bell s'n, wat nie gekook moes word nie [Natal Agric. J. (1904), 7, blds. 316]. Demuth se dip was ook 'n soortgelyke een. Die gevolg was dat hoofveearts Woollatt besluit het dat dipbakke sonder kookgeriewe opgerig sou word, wat dan ook al op daardie stadium deur heelwat boere uitgetoets was.

Alhoewel heelwat boere dipbakke na aanleiding van die sukses op Nel's Rust aangeskaf het, was daar nog nie 'n georganiseerde poging om bosluise te bestry nie. Toe egter breek „Rhodesiese Rooiwater" (later geblyk 'n nuwe siekte, Ooskuskoors, te wees) in Mei 1902 by Komatipoort uit, soos beskrywe deur dr. Arnold Theiler (1902/3). Hierna is baie werk gedoen in Transvaal op die siekte sowel as op rooiwater, maar meer bepaald op die biologies-veeartsenykundige sy. Min werk is oor die dipsy van sake gedoen, behalwe Theiler (1904) se Nelspruit proewe, 22-10-1903, waar paraffien, arseen en Izal-arseen gebruik is (Izal 'n koolteer-dip) om diere elke 8 dae te bespuit. Die arseen was afkomstig van Demuth se dip (11% As_2O_3) en die afdunning 1 in 25, skynbaar 'n sterkte van 0.44% As_2O_3. (Hierdie laasgenoemde feite is uit latere gegewens afgelei). Sy bevindings was dat dipping elke 8 dae in genoemde sterkte diere nie teen Ooskuskoors op 'n straf besmette plaas kon vrywaar nie.

Na aanleiding van die ander gebiede se werk en voorbeeld is ook 'n begin gemaak met die bestryding — deur middel van arseen-dipstof — van die bruisbosluis, wat later, volgens Lounsbury (1903), geblyk het die draer van Ooskuskoors te wees.

As gevolg van hierdie Ooskuskoorsbedreiging van die beeskuddes, veral in Natal, waar die siekte op 9 Julie 1903 in Ingwavuma uitgebreek het, is daar meer doelbewuste pogings aangewend om bosluise sistematies te bestry. Dit het dan ook ten gevolg gehad dat heelwat dipproewe uitgevoer is.

Behalwe kleiner proewe deur S. T. Amos (1904 en 1905) om bosluise te bestry, dosering van knoffelwater, dipping van trekosse in arseen en nagaan van effek op hulle, het veral H. Watkins-Pitchford uitgebreide dipproewe gedoen.

Volgens 'n berig in die „East London Despatch" (Maart 1905) het 'n firma, wat op een tydstip feitlik die alleen voorsieners was van dipstof in Suid-Afrika, die plaas Gonubie Park van Generaal-Majoor Sir E. Y. Brabant aangekoop. Die firma, nl. Cooper en Nephews, sou in daardie gebied deur proewe op 'n praktiese wyse aan die boere demonstreer en bewys dat arseen-dipstof bosluise

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doeltreffend kan bestry, asook gestandardiseerde dipstof aan boere verskaf.

Allereers gee H. Watkins-Pitchford 'n beskrywing van die „Natal Spraying Device" wat na aanleiding van die „Douglas Race" ontwerp is. Die groot verskil hier is dat 'n pypstelsel, met spuitkoppe en 'n kragaangedrewe pompstelsel aangebring is. In hierdie spuitgang het hy die diere, drie op 'n slag, gehou en bespuit totdat hulle deeglik nat was. As dipstof het hy aanbeveel: arseniet-van-soda (12 lb.), sagte seep (5 lb.), paraffien (1 blik waarskynlik 4 gl.), gliserien (5 lb.) in 400 gelling water. Dit sou 'n 0.24% As_2O_3 dipstof gee vir 'n arseniet wat 80% As_2O_3 bevat.

Met bogenoemde spuitgang en later met behulp van 'n dipbak het Watkins-Pitchford (1909) volgens sy eerste rapport die doeltreffendheid en bruikbaarheid van sowat 'n dosyn verskillende eiendomsdippe uitgetoets, veral met die oog op die gereëlde gebruik, elke vyf dae, vir die bestryding van die bruinbosluis, die vernaamste oordraer van Ooskuskoors. Die meeste van die dippe wat hy uitgetoets het, waaronder Tixol, wat vandag nog voortbestaan, het arseen bevat, 'n paar was koolteerdippe en een „geelektroliseerde seewater". Ongelukkig word die sterktes nie aangegee nie, aangesien dit waarskynlik ook nie aan hom bekend was nie. Waar hy dus na sekeres verwys wat nie effektief was nie, is dit nie duidelik of die arseenwaarde wel hoog genoeg was nie.

Veral het hy baie werk gedoen in verband met die notwikkeling van sy „laboratoriumdip", dit wil sê arseen-paraffien-emulsie. In die bespuitingstoetse het hy aanvanklik 1 lb. arseniet-van-soda per 20 gl. water waarby mengsels van seep, paraffien en gliserien gevoeg is, gebruik, dit wil sê, 0.40% As_2O_3. Dit het hy later verminder na 0.3% en met seep, gliserien en paraffien vir elf bespuitings met sukses gebruik. Toe het hy 'n 3,000-gellingdipbak gebruik met arseniet-van-soda, seep en paraffien:

(a) 1 lb. op 33 gallon water (0.24% As_2O_3)
(b) 1 lb. op 40 gallon water (0.20% As_2O_3)
(c) 1 lb. op 46 gallon water (0.17% As_2O_3)

Met eersgenoemde twee het hy nog brand van die diere gekry, maar nie met laasgenoemde nie, terwyl dit nog effektief was op bosluise. Sy finale aanbeveling was dan:

8½ lb. arseniet-van-soda
5½ lb. sagte seep
2 gl. paraffien
400 gl. water

Dit gee 'n konsentrasie van 0.17% As_2O_3.

In Watkins-Pitchford (1910) se tweede rapport verwys hy na werk wat hy gedoen het en wat bewys het —

(a) dat die hare van gedipte diere (elke dag vir 10 dae behandel) op 'n vk. vt. oppervlakte 0.201 en die vel 0.196 gm As_2O_3 bevat het;

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(b) dat die effek van arseendipstof op 'n dier vir 2 na 3 dae nog 'n goeie dodende effek het op bosluise, maar na verloop van 'n week na 50% val;
(c) dat die effek groter is op bosluise na dipping, in die geval van diere wat gereëld gedip word, teenoor die wat nie voorheen gereëld behandel is nie;
(d) dat arseen, alhoewel 'n wateroplossing, deur die vel geabsorbeer en deur die niere in die urien uitgeskei word.

Alhoewel dipstof nie voorheen getoets is, in elke geval nie in die veld nie, verwys hy na 'n toetser deur sy broer dr. Watkins-Pitchford ontwerp, alhoewel geen besonderhede van die toetser verstrek word nie.

Volgens Watkins-Pitchford (1911) se derde rapport, van werk wat skynbaar al in die vorige jaar gedoen is, het hy veral 'n uitweg probeer vind om in geval van Ooskuskoors, gedagtig aan die kort volsuigperiode van die bruinbosluis, met korter tussenposes te dip. Hierop het hy uitgebreide werk gedoen en bewys dat deur elke drie dae in 'n sterkte van 0.08% As_2O_3 te dip, hy die bruisbosluis op siek Ooskuskoorsdiere in so 'n mate kon beheer, dat hulle nie die siekte na gesonder diere in dieselfde kampie kon oordra nie. Besmette veld kon hy skadeloos stel deur beweiding met donkies en skape. Hy het verder werk gedoen op:

(a) Die tyd wat bruin larwes en nimfe geneem het om vol te suig.
(b) Effek van dipstof op volgesuigde bosluiswyfies.
(c) Die afwerende effek van die 0.08% As_2O_3 dip op bosluise.
(d) Die relatiewe aktiwiteit van bosluise gedurende dag en nag.
(e) Die nawerking van 0.08% As_2O_3 op bosluise wat opklim na dipping.
(f) Die effek van werklike dipping op melproduksie (grootste afname ± 3% een dag na dipping).
(g) Die onderwerp van die Isometer-dip-toetser. (Hy het van die dipstof by 'n spesifieke hoeveelheid toetsvloeistof gevoeg en so bepaal of dit te swak of te sterk was).

Die bogenoemde uiters omvattende en interessante werk van Watkins-Pitchford kan met reg beskou word as die afronding van die werk gedurende die eerste tydperk.

II. Die Tweede Tydperk: 1910 — 1940

Wetgewing

Hierdie tydperk kan in werklikheid tot 'n groot mate beskou word as 'n tydsbestek waarin reeds opgedane kennis toegepas is. Arseen was die aanvaarde dipstof. Regulasies is in werking gebring, sterktes voorgeskryf, metodes van bepaling is aandag aan geskenk, veranderinge wat die dipstof ondergaan is bestudeer. Proewe is ook uitgevoer om vas te stel hoe effektief die dipstof is, watter effek dit op die dier het, ens., kortom, wat dipstof self betref, is baie min nuuts tot die kennis bygevoeg.

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verwys is. Ook het Bedford en Jorden (1934) in proewe bewys, dat daar geen waarheid steek in die populêre idee van boere, dat alwyndosering bosluisbesmetting van diere kan verhoed nie.

Dipbakke

In 'n rapport oor dipstowwe en dipping in Natal verwys dr. A. Theiler en G. E. Gray (1912) onder meer na tipes dipbakke en word verwys na die "Unie-dipbak" wat beskrywe is deur R. W. Dixon (1911); die ingang verskil van die Nel's Rust-bak aangesien dit 'n regaf-ingang het. Verder is die idee soos beliggaam in die Nel's Rust-dipbak gevolg. Dit is dieselfde bak wat verskyn in Farmer's Bulletin no. 23 (1912) waarin R. W. Dixon behandel „East Coast Fever: Its Prevention and Eradication". Die dipbak het by die ingang twee vasskoptrappies, is 6 vt. diep, 2 vt. wyd (onder) en 4½ vt. bo, met 'n boom van 24 voet en 'n totale lengte van 43½ vt., kapasiteit 3,900 gls. Die bak is dus 'n bietjie groter as die Nel's Rust-bak van 3,500 gl. Dit het die basis gevorm vir al Onderstepoort se latere dipbakpanne.

In die begin van die twintiger jare is planne uitgegee onder datum 21.6.21 waar die vasskoptrappies deur slegs 'n plat rowwe oppervlak met groewe vervang is. Verder was die dipbak presies dieselfde soos deur Dixon beskrywe. Sowat tien jaar later verskyn planne onder datum Junie 1930 met verdere veranderings. Die ingang kry 'n voor, sowat 6 na 9 dm. diep, 1¾ vt. wyd, ongeveer 6 dm. van die regaf-val („Bolster"-tipe). Die mate is ook ietwat verander deurdat die bak heelwat dieper is en die kapasiteit gevolglik na 4,500 gelling vermeerder is. Die mate van die bak was die volgende: wydte, onder 1 vt. 9 dm.; bo 4 vt. 6 dm.; diepte 7 vt. 6 dm.; lengte van boom 24 vt.; totale lengte op oppervlak 43½ vt.

Hierdie dipbak het voortbestaan tot die koms van die nuwe sintetiese insektemiddels en is toe verklein na konsultasie met beamptes van die afdeling veeartsenydiens na die volgende mate: Wydte onder 1 vt. 3 dm., bo 4 vt. 6 dm., diepte 7 vt., lengte van boom 14 vt., totale lengte op oppervlak 35 vt., met 'n kapasiteit van 3,000 gl. Besonderhede van hierdie bak is beskrywe deur L. C. Blomefield (1955). Die vastrapplek van die ingang is weer verander na trappies (5 van 9 dm. wyd en 2½ dm. hoog) soos dit dan ook die beleid van die Afdeling was sedert die begin van die veertigerjare.

Toets van Arseendipstof

Onder die werk van Watkins-Pitchford is reeds verwys na die Isometer-arseendiptoetser, dat die dipstof by 'n bepaalde hoeveelheid gestandardiseerde jodiumoplossing gevoeg is en dat die hoeveelheid arseen (As_2O_3) net in die vorm van arseniet so vasgestel kon word. Die en alle latere toetsers is natuurlik almal baseer op die bekende chemiese feit dat jodium As_2O_3 na As_2O_5

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oksideer in 'n neutrale medium en dat As_2O_5 in 'n suurmedium met 'n reduseermiddel, soos natriumtiosulfaat, na As_2O_3 gereduseer kan word.

Green (1915) verwys na 'n toetser van Cooper en Nephews wat in Amerika verkrybaar was, maar nog nie in Suid-Afrika nie, sowel as na 'n toetser van die „United States Bureau of Animal Industry". Die enigste toetser wat toe in Suid-Afrika verkrybaar was, was die Isometer (waarna reeds verwys is) wat deur Henwood, Souter en Kie. van Durban voorsien is. Hy doen egter aan die hand hoe 'n goedkoper toetser saamgestel kon word, in die hoop dat 'n handelsfirma sou onderneem om so 'n toetser te vervaardig en te bemark.

Die voorgestelde toetser en toetsmetode kom op die volgende neer. Slegs vir arseniet word getoets en wel as volg. Die vuil dipstof, wat moontlik paraffien en seep kan bevat, word in 'n afgemerkte bottel met 'n mengsel natriumbikarbonaat en magnesiumsulfaat behandel. Van die skoon gedeelte of (indien nodig) gefiltreerde gedeelte word in een van vier afgemerkte buise gevoeg tot op die onderste merk. Gestandardiseerde jodium in een van die drie sterktes (vir 3-, 5- of 14-daagse sterktes) word uit 'n drupbottel bygevoeg, geskud en met stysel-behandelde papier vir 'n blou endpunt getoets. Die buise word so afgemerk om lesings te gee van die hoeveelheid arseniet-van-soda (80% As_2O_3) of water wat by 100 gellings dipstof gevoeg moet word om die korrekte sterkte, afhangende van die diptussenpose, te gee. Ook kon een buis gebruik word met die vier aparte skale op papier aangebring, sodat dit agter die buis geplaas kon word om die lesings te neem.

Green eindig bogemelde besonderhede deur te verwys na Chapin (1914) se metode van bepaling, wat hy by die ter perse gaan van sy werk ontvang het. Chapin gebruik 'n wyebek bottel waarin die titrasie gedoen word, na die byvoeging van 'n bikarbonaat-styseltablet. Hy het 'n afgemerkte maatsilinder gebruik om die dipstof af te meet en ook vir jodiumbyvoeging.

Van Zyl (1923) het 'n verskeidenheid van metodes uitgetoets, ook die Isometer, en tot die gevolgtrekking gekom dat dit wenslik is om die dipstof skoon te maak, voordat titrasies uitgevoer word. Hiervoor gebruik hy dierkool en verwarming na byvoeging van swawelsuur. (Skrywer (P.M.B.) se ondervinding is egter dat die gebruik van koolstof, omdat arseen skynbaar ge-oklideer word, geneig is om te lae waardes te gee.) Vir die bepaling van totale arseen het hy gevind dat tiosulfaat volledige reduksie gee van As_2O_5 na As_2O_3 in swawelsuurmedium. Ook het die reduksie deur middel van kaliumjodied en swawelsuur ewe goeie resultate gelewer, maar van die praktiese oogpunt sou die tiosulfaatreduksie vir 'n veldtoetser verkiesliker wees. Die veldmetode (Isometer), tot op die stadium in gebruik, was dus beskou as ongewens, veral nog omdat dit slegs arseniet kon toets.

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Verskeie werkers in die buiteland sowel as in Suid-Afrika het gerapporteer oor die oksidasie van arseniet (As_2O_3) dipstof na arsenaat (As_2O_5) in die dipbak. Lewis (1914) verwys na Brunnich (1909) in Australië, Cooper & Laws (1911/13), Fuller (1911) en veral na waarnemings deur Williams (1913). Laasgenoemde het bevind dat vars arseniet-dipmonsters baie min verander indien in die laboratorium gehou. In monsters van vuil gebruikte dip vind oksidasie na arsenaat vinnig plaas. Monsters gedurende wintermaande geneem, toon nie die vinnige oksidasie nie. Skynbaar het die „arsenaatvormende faktor" uitgesterf gedurende die koue winter. Volgens proewe was hy seker dat bakterieë hiervoor verantwoordelik was, soos dan ook later deur Green (1918) bewys is dat in dipbakke reduserende en oksiderende bakterieë verantwoordelik is vir die verandering van arsenaat na arseniet en omgekeerd.

Bykomend het Williams (1922) getoon dat arseendippe waarin koolteer is, meer geneig is tot oksidasie. In 15 dipbakke met 'n arseenkoolteer dip, was 41.6% van die arseen geoksideer. In 6 arsenietdippe was die oksidasie slegs 2.3%. Van Zyl (1923) verwys na die neiging tot oksidasie in teenwoordigheid van fenole. Hoe langer die tussenpose tussen dipping hoe groter die neiging tot die vorming van arsenaat. Die oksidasie van arseniet na 'n arsenaat, wat ook al die oorsaak, het 'n probleem geskep i.v.m. die toets van dipstof met die Isometer.

Na aanleiding van die werk van Green (1915) en Van Zyl (1923) is 'n toetser op Onderstepoort ontwerp. In die Jaarverslag van die Afdeling Veeartsenydiens (1923) verskyn 'n aankondiging dat 'n nuwe laboratoriumdipproewer ontwerp is en hopelik teen 5/- of 7/6 aan die publiek voorsien sal kan word. Dit word nie gesê deur wie dit ontwerp is nie. Geen beskrywing daarvan kan gevind word nie. Dit word egter algemeen aangeneem dat Green dit ontwerp het. Dit skyn egter of J. P. van Zyl ook heelwat met die ontwikkeling daarvan te doen gehad het na aanleiding van werk wat hy in 1923 in die verband gedoen het.

In die Jaarrapport van die Sekretaris van Landbou (1926) word, onder die Onderstepoortverslag, die volgende aangegee. „Laboratoriumtoetsapparaat van Dipstof. 'n Aantal toetsapparate vir dipstof is verlede jaar aan veldbeamptes van die Departement uitgegee om te probeer, en na 'n kleine wysiging daarin aangebring is, is daar 'n bestelling gemaak om met die uitreiking daarvan aan die publiek 'n begin te maak."

Die eerste gepubliseerde besonderhede het onder datum 5.2.27 verskyn toe 3,000 kopieë van die gebruiksaanwysings (839—5.2.27 —3000) gedruk is, en 'n begin gemaak is met die uitreik van die toetsers. Hierdie bewoording het as sodanig woordeliks voortbestaan totdat Bekker (1944/55) klein veranderings aan die gebruiksaanwysings aangebring het sowel as die hele pamflet in 'n

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ander vorm omskep het en die toetser "Jodometer-Diptoetser" genoem het.

In wese kom die toets daarop neer dat 100 ml. dipstof skoongemaak en aangesuur word met twee rooigekleurde suur tablette, bevattende kaliumpirosulfaat (1.50 gm. $K_2S_2O_7$, 0.45 gm. talk, 0.025 gm. $NaHCO_3$, en stysel per 2.5-gm.-tablet). Die „suur" bestaan uit die suurkaliumsulfaat ($KHSO_4$) wat gevorm word as kaliumpirosulfaat hidroliseer. Die suurgehalte van 25 ml. van die skoongemaakte dipstof word geneutraliseer deur 1 na 1½ alkaliniserende witgekleurde bikarbonaattablette te gebruik (1.15 gm. $NaCHO_3$, 0.12 gm. melksuiker, stysel en talk per 1.5-gm.-tablet) en die arseniet daarin met 0.0505 N jodium tot 'n blou endpunt te titreer. Elke 1 ml. jodium hier gebruik stel dus 0.01% As_2O_3 in die dipstof voor.

Om ook vir arsenaat te toets word 'n ½ na 1 reduserende blougekleurde natriumtiosulfaattablet (0.25 gm. $Na_2S_2O_3$ en talk per 0.272-gm.-tablet) by 25 ml. van die aangesuurde skoon dipstof gevoeg. Na 'n tydreaksie van 6 minute word die oormaat tiosulfaat (wat meer as 5 ml. jodium moet opneem) na die onaktiewe tetrationaat met jodium geoksideer, terwyl die medium nog suur is. Na neutralisering van die suur word bogenoemde titrasie vir arseniet herhaal en die totale arseengehalte vasgestel.

In die jongste pamflet (Desember 1955) verskyn 'n dipbakskets en wenke oor die korrekte gebruik van arseendipstof. Daar word nog vermeld dat die arseengedeelte slegs 2/3de die doeltreffendheid van arseniet besit en berekenings vir die korreksie hiervoor word aangebring. Skrywer (P.M.B.) heg geen waarde hieraan nie. Met die volgende publikasie van die pamflet sal die „2/3de berekening" dan ook weggelaat word. Die rede vir hierdie houding is te vind in sekere praktiese implikasies sowel as die twyfel of die arsenaat werklik net 2/3de die doeltreffendheid van arseniet besit, as die sterkte daarvan ook in terme van As_2O_3-gehalte aangedui word. Die praktiese implikasies is die volgende: om verdere oksidasie veral in 'n monster te verhoed, moet dit dadelik, by die neem daarvan, aangesuur word, anders gaan oksidasie voort. Die meeste monsters wat ingestuur word, word nie aangesuur nie. Verder bevat die meeste monsters vandag 'n mengsel van dipstowwe, wat uit emulsies en suspensies van tintetiese insekmiddels bestaan. Om by so 'n mengsel met arseen 'n suur te voeg, beteken dat die emulsie breek en die suspensie belemmer word. Vir die analiseer van die nuwere sintetiese middels moet dus geen suur bykom nie. Om dus die „2/3de berekening" in sommige gevalle toe te pas en in ander na te laat, sou onlogies wees. Om bogemelde redes het die „2/3de formule" totaal in onbruik geraak. Samehangende hiermee is tot 'n mate afgewyk van die sewedaagse sterkte van 0.16% As_2O_3. Volgens regulasies no. 136 (18.1.52) word 'n sterkte van 0.16 tot 0.20% As_2O_3 voor-

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geskryf en word aanbeveel dat die totale arseengehalte nie hoër as 0.20% moet wees nie.

Na die finale ontwerp van die arseendiptoetser is vir die res van die tweede tydperk baie min of feitlik geen verdere werk, wat die dipaspek betref, gedoen nie. Waar die tweede tydperk, met die inwerkingtreding van regulasies onder die Verenigingswet begin het, het dit in werklikheid ook geëindig, toe dieselfde regulasies in gedrang gekom het en toe daar tydelik, so is natuurlik in die begin gehoop, gestaak is met die toepassing daarvan. Die regulasie het naamlik bepaal dat die teenwoordigheid van 'n volgesuigde bloubosluiswyfie op 'n bees voldoende bewys is dat so 'n dier vir 'n tydperk van drie weke nie gedip is nie. Afdoende bewys is egter voor die hof in Oos-Londen gelewer, dat alhoewel diere gereeld gedip is, volgesuigde bloubosluiswyfies nog teenwoordig was. Vervolgings onder die regulasie is dus, hangende 'n ondersoek, tydelik (so is gehoop) gestaak.

III. Die Derde Tydperk: 1940 — 1959

Hierdie tydperk begin met die verskyning van, en dipproewe in verband met, die arseenbestande bloubosluise. Dit kenmerk die begin van die gebruik van sintetiesorganiese dipstowwe, om eerstens die bestande bloubosluis te bestry maar ook vir gebruik teen ander soorte bosluise sowel as die bestryding van alle parasiete op diere in die algemeen.

Die ontstaan van die arseenbestande bloubosluise kon volgens Bekker (1942 en 1953) deur hom sover as Mei 1937 naby Berlin en Maart 1938 naby Komgha teruggespoor word. Spesiale aandag is aan die dipping op die twee plase W.tchkrantz en Jessey gegee, voor vendusies aldaar gehou is, vandaar dat dit aan spesifieke datums gekoppel kon word. Alhoewel gereeld weekliks deeglik gedip is was daar nog volgesuigde bloubosluise op die diere teenwoordig met laasgenoemde verkoping, terwyl eersgenoemde afgelas is.

Nadat die bosluis 'n groot probleem veral aan die wesoewer van die Buffelsrivier geskep het en die plaaslike personeel onder dr. W. B. Allchurch (departementele korrespondensie) die grondigheid van die klagtes ondersoek het, is die hulp van beamptes van Onderstepoort ingeroep om die probleem te ondersoek.

Voorafgaande proewe is op Onderstepoort uitgevoer om die effek van die dipstof op die sogenoemde bloubosluis asook op sy lewensloop vas te stel, waarna grootskaalse veldproewe in die Oos Londen-gebied deur P. M. Bekker uitgevoer is. Hierdie resultate is gepubliseer deur Du Toit, Graf en Bekker (1941), waarvolgens blyk dat die bloubosluis (*Boophilus decoloratus*) sonder dat dit enige verandering in uiterlike voorkoms, lewensloop of leefwyse ondergaan het, nie meer deur middel van arseendipstowwe doeltreffend bestry kon word nie, terwyl dit nog die geval was met die gewone stam van hierdie bloubosluis elders.

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Volgens dié proewe is verskeie stowwe uitgetoets. Slegs nikotien was effektief en wel as 'n byvoegsel tot arseen (6.16% As_2O_3) om 'n konsentrasie van 0.04% nikotien te gee. Wat duidelik geblyk het uit die proewe is dat die nikotien nie so effektief was dat die bloubosluis in 'n paar dippings van die diere verdwyn het nie. Al word die diere in die korrekte sterkte gedip, dan kom daar nog gevalle voor waar die diere eers na die sewende dip geheel en al vry was van volwasse, volgesuigde bloubosluise.

Na verdere proewe beveel Bekker (1944) 'n byvoeging van 10 lb. 40% nikotiensulfaat op 800 gelling aan vir varsvulling en 1 pint op 100 gelling vir aanvullings om die natuurlike afname van die nikotien (40%, teenwoordig as nikotiensulfaat) in die dipbak teen te werk en te verseker dat die konsentrasie gereeld bo 0.04% nikotien in die 0.16% As_2O_3 dip is.

Omdat tabakekstrak (nikotien) egter weens oorlogsomstandighede skaars was, het Bekker (1942) 'n metode ontwerp om van afvaltabak, deur direkte uitloging in die dipbak, gebruik te maak. Ook het Bekker en Koch (1943) die kweek van 'n tabak met hoë nikotiengehalte vir die doel aanbeveel.

Terselfdertyd kon ook vasgestel word dat hierdie bosluis van plaas tot plaas en van distrik tot distrik deur middel van diere versprei is. Teen die einde van 1944 het die bosluis voorgekom vanaf Grahamstad in die suide, al langs die kus tot in Seoloeland en het later tot in Swaziland, Barberton en die oostelike laeveld versprei.

Verskeie ander werkers het ook die bestandheidsprobleem ondersoek waaronder prof. Barker (persoonlike mededelings) van die Rhodes Universiteit, A. B. M. Whitnall, mej. E. M. Fenwick, mej. B. Bradford. So het artikels verskyn deur A. B. M. Whitnall en mej. E. M. Fenwick (1945). A. B. M. Whitnall en mej. B. Bradford (Aug. 1945) het uitgebreide laboratoriumproewe beskryf om te bewys dat die bloubosluis wel arseenbestand is. Ook het Fiedler (1952) getoon dat die arseen- en BHC-bestande bloubosluis gelyktydig ook 'n merkbare bestandheid teen Toxafeen, Dieldrin en soortgelyke verwante verbindings besit het. Wat Toxafeen betref, het skrywer in proewe in samewerking met Blomefield, in 1949 gevind dat dit van die begin af nie geheel en al effektief was teen die bloubosluis nie. In 'n artikel oor die bestryding van bosluise in Suid-Afrika verwys Fiedler (1958) na werk van Blomefield (1954) en Whitehead (1956) in verband met verdere bestandheid van die bloubosluis en wel teen DDT.

Aangesien nikotien moeilik bekombaar was gedurende die oorlogsjare, moes 'n ander uitweg gevind word om die bloubosluis te beheer, veral as in aanmerking geneem word dat die vrektes van beeste aan rooiwater, gedurende die tydperk 1935 tot 1945 van 143+ tot 8375+ toegeneem het (departementele lêers). Gelukkig is vertroulike inligting uit die buiteland ontvang en is die moontlikheid van DDT en BHC as insektemiddels besef. Op die

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stadium is DDT al vir die geallieerdes op Klipfontein vervaardig en het die personeel goedgunstelik ingewillig om 'n hoeveelheid BHC spesiaal vir skrywer te vervaardig. Hierdie BHC is buite die laboratorium in bottels, waarin benseen in die son gechlorineer is, vervaardig. Van hierdie DDT en BHC het skrywer gedurende 1945 emulsies formuleer: nl. E30 (BHC) en E20 (DDT), waarvan laasgenoemde nog vervaardig en verkoop word deur die staatsfabriek K.O.P.

Voorrade van hierdie emulsies (kaseiën-harpuis-tipe, waarvan besonderhede binnekort gepubliseer sal word) is uit plaaslike produkte op Onderstepoort, veral met die hulp van J. R. Malan, vervaardig en was gereed vir gebruik in veldproewe in die begin van die somer 1945/46. Gedurende Februarie kon 'n begin gemaak word met die proewe wat uitgevoer is deur skrywer, en gepubliseer is deur Bekker en Graf (1946). Die eerste DDT-dipbak is gevul op 25.2.46, op die plaas Tayside, van mnr. J. W. A. Shaw, naby Kidds Beach. Die konsentrasie was 0.5% pp DDT en het wonderlike resultate op alle soorte bosluise gelewer, maar dit het ook 'n wonderlike som geld gekos om so 'n bak te vul. Ander dipbakke met DDT as 'n byvoegsel tot arseen (0.16% As_2O_3) op 'n 0.1% konsentrasie het ewe-eens uitstekende resultate gelewer. Die gevolgtrekking waartoe geraak is, was dat die arseenbestande bloubosluis doeltreffend met emulsies van BHC (0.005% gamma BHC) en DDT (0.1% pp DDT) bestry kon word, indien dit as 'n byvoegsel tot arseendippe gebruik word.

Op 30 Mei 1947 verskyn 'n verslag in die „Daily Despatch" (Oos-Londen) deur J. W. Shoebotham waarin hy 'n beskrywing gee van die eerste dipbak wat op die plaas „Shelford" van veeinspekteur L. G. Willioms gedurende Augustus 1945 gevul is met Gamatox-(BHC)-dipstof. Die plaas van Shaw, waarna reeds verwys is, sowel as Wiltonside, die plaas van C. H. Adcock, waar die eerste nikotien-proewe in 1940 uitgevoer is, is almal geleë in 'n radius van 5 na 8 myl. Besonderhede van hierdie proewe met BHC wat in Augustus 1945 begin is, is deur Thorburn (1947) gepubliseer en het hy ook tot soortgelyke gevolgtrekkings gekom. Dieselfde geld ook vir werk deur Whitnall en Bradford (1947).

As gevolg van vertoë van firmas, boere en veeartse om van arseendipstof ontslae te raak, is nuwe dipregulasies (no. 1215, Staatskoerant, 13 Junie 1947) gepubliseer. Hiermee is die alleengebruik van DDT-emulsies (0.2% pp) en BHC (0.01% gamma BHC) teen bosluise gewettig, alhoewel skrywer (wat die proewe uitgevoer het) nie meermee saamgestem het nie. Ook is DDT (0.1% pp) en BHC (0.005% gamma) as 'n byvoeging tot arseen (0.12% As_2O_3) en die twee gesamentlik, sonder arseen, toegelaat. Arseen (0.16% As_2O_3) is egter nog erken vir alleengebruik. Die regulasies (paragraaf 5) het egter bepaal: „Hierdie bevel bly van krag vir 'n tydperk van twaalf maande". Dit het ook bepalings bevat wat die staatsveearts gemagtig het om arseen te laat byvoeg indien BHC nie die bosluise doeltreffend beheer het nie.

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As gevolg van die ondoeltreffendheid van die alleengebruik van die DDT en van die BHC op bogenoemde konsentrasie teen ander soorte bosluise, is hierdie regulasies daarna nie weer hernu nie. (Nuwe regulasies in die verband het egter eers weer in 1952 verskyn.)

'n Verdere terugslag vir die BHC-dippe het gekom toe klagtes (sover bekend die eerste) van die eienaars van die plaas „Mistley, Lone Tree", naby Berlin (Oos-Londense gebied) teen die einde van 1947 en die begin 1948 ingedien is, nl., dat die BHC-dipstof ondoeltreffend is teen die arseenbestande bloubosluis. Dr. L. C. Blomefield (staatsveearts, Oos Londen) het hierdie klagte ondersoek en die diere vir elf agtereenvolgende dippings met weeklikse tussenpose, in dubbelsterkte (0.01% gamma) BHC-dipstof gedip. Sy bevindings was dat gedurende hierdie tydperk, 4.2.48 tot 14.4.48 die bloubosluis altyd teenwoordig was en dat die besmetting erger geword het. Vanaf 8.11.48 tot 12.4.49 het Blomefield en Bekker (ongepubliseerde resultate) verdere proewe op bogemelde plaas uitgevoer en gevind dat die bosluis werklik bestand was teen BHC, sowel as teen arseen, maar nie teen DDT nie, en nie beheer kon word selfs deur 'n 0.05%-gamma-BHC-sterkte te gebruik nie. Trouens 0.1% was ook nie eers geheel en al doeltreffend nie en dit in teenstelling met die feit dat 0.005% in die begin baie effektief was. Dieselfde tyd het Whitnall *et al* (1951) en (1952) die bestaan van 'n bloubosluis, wat teen BHC bestand is, bevestig.

In Julie 1949, in 'n voordrag gelewer voor die Suid-Afrikaanse Genootskap vir die Bevordering van die Wetenskap te Kimberley, het Bekker, Graf, Malan en Van der Merwe (1949) daarop gewys dat 0.01% gamma BHC, wat sonder arseen gebruik word, nie voldoende is vir algemene bosluisbestryding nie.

Na aanleiding, veral van werk wat deur Blomefield (ongepubliseer) gedoen is op die gebruik van 0.02% gamma BHC-konsentrasie is weereens regulasies uitgevaardig (no. 136 van 18.1.52 en 'n korreksie no. 2269 van 3.10.52) waarin weereens die alleengebruik, nou van BHC (0.03%/vir byvoeging 0.04% gamma), DDT-emulsies (0.25/0.40% pp), Toxafeen (0.25/0.375% gechlorineerde kamfien C.L.C.) en natuurlik arseen 0.16 tot 0.20% As_2O_3), gewettig is. Byvoegings tot arseen (0.16% As_2O_3) het nog geskied op dieselfde basis vir DDT-emulsie, nl., 0.1%, maar vir die benatbare poeier (wat 'n geruime tyd al verdwyn het) was dit op 'n basis van 0.2% terwyl vir BHC 'n byvoeging van 0.01% gamma aangedui word.

Tot op die huidige stadium was DDT die vernaamste dipstof waarmee die arseenbestande bloubosluis doeltreffend bestry is. Na aanleiding van bogemelde bevindings het Essenwein, Bekker en Ypsilanti (1958) proewe op die plaas Bush View (O.L.) van mnr. E. Martin Kruuse uitgevoer en tot die volgende gevolgtrekkings gekom: na 5 dippings in 'n 0.1% pp DDT + 0.03% gamma BHC was daar nog baie bosluise van alle stadia op die gedipte

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diere teenwoordig, d.w.s. onteenseglik 'n bestandheid teen DDT. Na 'n verdere ses weke was daar slegs 'n ligte besmetting op die gedipte diere, terwyl die kontroles oortrek was van bosluise. Hierdie verbetering het geleidelik toegeneem totdat na 2 maande daar slegs by uitsondering selfs 'n enkele bloubosluis op die gedipte kudde gekry kon word, in teenstelling met die swaar besmette kontroles. Op die 5 kontroles is 884 volgesuigde bloues getel en op vier gediptes kon slegs 18 volwasse bloues gevind word.

Alhoewel die bloubosluis dus ook al bestand is teen DDT, kan dit met gereelde weeklikse dipping, vir die huidige altans, in bedwang gehou word sonder dat dit enigsins in 'n probleem ontwikkel.

Bespuiting van diere wat in die vorige eeu prominensie in Grahamstad verwek het, het weer op die voorgrond getree; Blomefield in samewerking met Du Toit (1954) veral het proewe in dié verband uitgevoer, hoofsaaklik met die oog daarop om 'n metode te ontwerp om die nuwere dipstowwe, wat in die dipbak veranderinge en groot afname in sterkte ondergaan, vars te kan gebruik. So is een van die eerste spuitgange onder toesig van Blomefield in Oos Londen op A. M. Kretzmann se plaas, „Michaelton" naby Fort Jackson op 23 Oktober 1951 opgerig. Gelykertyd het private firmas proefspuitgange, waarvan daar vandag sowat 'n half-dosyn verskillende ontwerpe te koop aangebied word, ontwerp. Die grootste voordeel van die spuitgange is dat die spuitstof elke keer vaars opgemaak word. Dit is nie goedkoper as dipping nie en die benatting in die dipbak bly nog die beste manier vir die doel.

Die poging om die gebruik van die nuwere organiessintetiese dipstowwe op die korrekte sterkte te laat geskied is aanprysingswaardig (mits die benatting deeglik is), want in die dipbak is die prentjie nie te rooskleurig nie. Omdat die dipstof baie duurder is as arseen en ook vinnig afneem in sterkte, word die dipstof gewoonlik op te lae konsentrasies, wat in werklikheid nutteloos is, gebruik.

Gedurende 1954/55 het die dipseksie te Onderstepoort 'n opname gedoen om vas te stel in watter persentasie van die dipbakke dip op korrekte sterkte gebruik is. Die gebiede wat onder toesig was en waar ooskuskoors een of ander tyd voorgekom het, is gekies vir die doel, nl. die Ciskei, Transkei, Griekwaland-Oos, Natal, Soeloeland en die Oos-Transvaalse laeveld. Deur loting is een uit elke 10 bakke gekies en monsters ingestuur. Van die ongeveer 10,000 bakke is 968 dipmonsters ontvang. Die volgende tabelle gee 'n opsomming van die bevindings.

TABEL A

Groep	Tipe Dipstof Gebruik	Aantal Monsters	Aantal Korrek	% Korrek
A	DDT	280	4	1
B	BHC	218	15	7

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C	Toxafeen	103	10	10
D	Arseen	168	109	65
E	Arseen en DDT	60	7	17
F	Arseen en BHC	84	19	
G	BHC en DDT	35	1	3
H	Arseen en Toxafeen	8	0	0
I	Sonder effektiewe prinsiep	12	0	0
	Totaal	968	165	17

TABEL B

Opsomming van Kommentaar oor bosluisposisie by die 968 bakke

Gunstig	Ongunstig	Geen kommentaar
21%	41%	38%

Wat die nuwe dippe betref was die konsentrasies in 30 monsters korrek, d.w.s. slegs 21%, arseen 109, d.w.s. 65% van die betrokke aantal monsters. In die geheel was 165 monsters korrek, d.w.s. 17%. 62% van die dipbakke was met die organiese dipstowwe gevul en die res met arseen en arseenmengsels. Die haglike toestand word verder weergegee in tabel B waar gunstige kommentaar — wat bosluisposisie betref — vir slegs 21% van die dipbakke gelewer is. Onteenseglik weerspieël bogemelde dat bosluisbeheer met die koms van die sintetiese dipstof 'n terugslag ondervind het, veral omdat slegs 'n geringe persentasie van die dipbakke op sterkte was. Hierdie toestand was op die tydstip al vir meer as vyf jaar aan die gang, m.a.w. vanaf die tyd dat die nuwe dippe algemeen in gebruik gekom het.

Verdere klagtes het ook ontstaan toe boere in die omgewing van Pretoria vrektes as gevolg van rooiwater onder hulle vee ondervind het en dat hulle nie die bloubosluis met BHC-dipstof kon beheer nie. Proewe deur Bekker (1953) het bevestig dat hier 'n bloubosluis ontstaan het wat nie meer doeltreffend met BHC-dipstof bestry kon word nie. Ook het die proewe getoon dat die bosluis nog met DDT gemaklik beheer kon word. Bogemelde proewe is met hand-bespuiting gedoen. Aangesien die boer egter ook 'n 40 diere gedurende daardie jaar aan hartwater verloor het, en dit nie met BHC-dipstof, wat op die stadium 'n 0.0225% gamma-BHC-sterkte gehad het, kon beheer nie, het skrywer hom aangeraai om dadelik arseen (0.16% As_2O_3) in die dipbak by te voeg. Die arseen-BHC-kombinasie was dan ook doeltreffend teen die bloubosluis, wat daarop gedui het dat arseen alleen ook effektief sou wees. Die proef is herhaal deur die dipbak skoon te maak en net met arseen (0.16% As_2O_3) te vul. 'n Groep van 10 diere, waaronder 4 diere was wat tot op daardie stadium net

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met BHC bespuit was en dus oortrek was van die bloubosluis, is in die arseendipstof gedip en dopgehou. As kontrole is diere van die vorige arseen-BHC groep, op dié stadium vry van bloubosluise, met BHC (0.03% gamma) bespuit. Toe die proef beëindig is, was die diere van die BHC-groep oortrek van alle stadia van die bloubosluis, terwyl die arseen-groep skoon was.

Alhoewel geen spesiale aandag met die tweede proef aan ander bosluissoorte bestee is nie, het die eienaar bevestig dat hy vanaf 3 Maart 1953, met ingebruikname van arseendip, tot in Desember 1953, toe die besonderhede van die tweede proef vir publikasie ingelewer is, geen verliese aan rooiwater (deur die bloubosluis oorgedra) of hartwater (deur die bontbosluis oorgedra) gehad het nie.

Met die verskyning van die resultate van die eerste proef in 1953 is 'n hewige reaksie uitgelok van sommige firmas, wat tot van die Minister wou weet waarom sulke resultate gepubliseer is. Besonderhede van die tweede proef, alhoewel in Desember 1953 ingedien, is nog nie gepubliseer nie. Dit ten spyte kan ek meld dat op die melkplaas „Glen Farm", digby Pretoria, sedert 1953 elke week, winter en somer, in arseen gedip word en dat beide mnr. Carswell-Smith en nou mnr. Struben tot op die huidige datum nog nie weer klagtes gehad het oor bosluise en bosluisoorgedraagde siektes nie. Dit, soos baie ander getuienis, is onteenseglik bewys dat arseen en die dipbak geensins iets van die verlede is nie, maar dat 'n groot vraagteken hang oor die praktiese toepassing van die alleengebruik van die nuwere dippe in die dipbak. Die geskiedenis van verhoogde dipkonsentrasies met die verskyning van elke stel regulasies, is genoegsame bewys dat die gebruik van 0.01% gamma BHC, die werk van Whitnall *et al* (1951) ten spyt, net nie in die praktyk werk nie.

Bykomend hiermee moet die bevindings van Roulston en Schuntner (1958), dat die sterkte van die gamma-BHC-isomeer vinniger afneem as die ander isomere in dipstof, nie verontagsaam word, selfs ook nie waar spuitdipping met BHC toegepas word nie. Die werk staaf die bewerings en waarnemings deur skrywer elders in gekwoteerde verwysings gemaak, dat alhoewel die dipstof se sterkte chemies korrek mag wees, dit nog nie beteken dat die biologiese effek doeltreffend is nie.

In tussen is die Veesiekte Wet van 1911, soos gewysig, vervang deur Wet 13 van 1956 ("Wet op Dieresiektes en Parasiete"). Weereens moes die Afdeling Veearsenydiens sekere konsentrasies van dipstowwe verhoog. BHC-emulsies na 0.03/0.045%, suspensies na 0.035/0.0525% gamma; DDT na 0.30/0.45% pp; net arseen (0.16 na 0.20% As_2O_3) en Toxafeen (0.25/0.375% C.L.C.) het dieselfde gebly. Byvoegings tot arseen (0.16 — 0.20% As_2O_3) en ander verenigbare dipstowwe word toegelaat, veral met die oog op die bestande bloubosluis, nl. nikotien (0.05/0.075%), Asuntol (0.02/0.03%), DDT-emulsies (0.15/0.225% pp), Diazinon (0.03/

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0.045%) en Delnav (0.03/0.045%. Die volgende byvoegsels word toegelaat om die doeltreffendheid van arseendipstof te verhoog, nl. Toxafeen (0.25/0.375%) en BHC-emulsie (0.03/0.045%) en suspensie (0.035/0.0525% gamma). Die tweede syfer dui altyd die aanvullingskonsentrasie aan en die eerste die varsvulling. Vir spuitgange, elke keer vars gevul, word die volgende voorgeskryf: Toxafeen (0.25% C.L.C.), DDT-emulsie (0.30% pp); BHC-emulsie (0.03%), suspensie (0.035% gamma); Diazinon (0.05%); Asuntol (0.04%) en Delnav (0.05%).

Onder bogemelde is heelwat „nuwe gesigte" te wete: Asuntol (0,0 dietieltiofosfaat van 3 chloro-4 metiel-7 oxikumarien); Diazinon (2 isopropiel-4 metielperimediel-6 dietieltiofosfaat) en Delnav (2,3-dioxaan dietiel-S, S-b s (0,0-dietiel fosforoditioaat), almal tiofosfate. Besonderhede i.v.m. werk gedoen op die gebruik van Asuntol is gepubliseer deur Fiedler en Veldman (1957) en Fiedler (1958). Sover bekend het Cooper & Nephews (Delnav) en K.O.P. (Diazinon) nog nie besonderhede oor hulle produkte vir publikasie vrygestel nie.

Op „pad" is 'n verdere nuwe groep, nl. Karbamate, waarvan een t.w. Sevin (1 naftiel-N-metielkarbamaat) van K.O.P. eersdaags op 'n gebruikskonsentrasie van 0.075% vir spuitgange sal verskyn.

Voordat ek afsluit sou dit wenslik wees om kortliks te verwys na die aard van die dipstowwe wat op die huidige tydstip op die mark is:

1. Arseen: as arseniet-van-soda-poeier bevattende 80% As_2O_3 en patente vloeibare dipstof met 64% As_2O_3.
2. BHC: BHC benatbare poeiers bevattende 6 en 10% gamma BHC en ongeveer 43 en 75% totale BHC. Emulsies in die vorm van watermengbare olies met 'n gammagehalte van 12 na 13% en 'n totale BHC-waarde van 30 na 40%.
3. 75% Toxafeen watermengbare olies.
4. DDT emulsies en watermengbare olies waarin die pp-DDT-gehalte om en by die 20% is en totale DDT ongeveer 30 .
5. Asuntol: 50% Asuntol benatbare poeier.
6. Diazinon: 'n 70% Diazinon watermengbare olie.
7. Delnav: 'n 30% Delnav watermengbare olie.
8. Seven: 50% Sevin benatbare poeier.

Terwyl die ontwikkeling op die gebied van verdere nuwe parasitêre middels aan die gang is, „pantser" die bosluis hom verder met bestandheid teen die middels. Interessante werk in die verband is gedoen deur Whitehead (1958) deur die uittoets van 'n verskeidenheid van parasitêre middels en deur Thompson en Johnson (1958) deur vas te stel dat die bestande bloubosluis ongeveer tweemaal soveel van die -SH radikaal bevat.

Alles dui daarop dat die „geveg" met bosluise al interessanter, maar ook meer en meer ingewikkeld word, nie net vir die boer alleen nie, maar vir almal. Die skeikundige staan voor die probleem

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van bepalingsmetodes, veral as mengsels gebruik word. Werk in die verband deur Malan en Bekker (1958) gedoen, probeer help om praktiese metodes te ontwerp om vir DDT, Toxafeen, Malathion en Diazinon in vuil dipstof te toets.

Afgesien van bosluisbeheer kom die veearts ook in die gedrang van die oogpunt van toksisiteit beskou. So verwys Bekker (1958) na die breek van emulsies en die gevaar as die dipstowwe nie verenigbaar is nie; hoë mortaliteit kan in sulke gevalle voorkom en dit op gewone sterktes. As 'n emulsie breek is dit baie gevaarliker as wat arseen, op gewone dipsterkte, kan wees.

Waar die geskiedenis van dipping van beeste in Suid-Afrika begin het met baie probleme, is daar seer sekerlik vandag, na 70 jaar, nie juis minder nie. Mag spreker die wens uitspreek dat alle betrokke instansies lering sal trek uit die verlede en help om die neiging om te lae konsentrasies in gebruik te neem te bekamp, en terselfdertyd nie net as gevolg van handelsoorwegings, meer en meer nuwe middels op die mark te plaas nie as dit nie nodig is nie. Ondersoekingswerk kan en behoort natuurlik voort te gaan, maar dit is altyd wyselik om van die middels in reserwe te hou anders word die saak vir die bloubosluis dalk ook so ingewikkeld dat hy somar „besluit" om 'n bestandheid teen alle moontlike en onmoontlike middels op te bou.

Wat uiters belangrik is, is dat die benadering van die probleem, en die wyse waarop die middels aangewend word, in ooreenstemming met die praktyk gebring word; dan en dan alleen kan die korrekte gebruik van die nuwere middels in seën wees. So is deur korrekte gebruik die tsetsevleig in Soeloeland (du Toit, 1954) uitgeroei. In hierdie kampanje is ook van dipping van beeste in 'n arseen-DDT-mengsel (0.06% As_2O_3 + 0.1% pp DDT) in gebiede met min vlieë, waar vliegtuie nie gebriuk kon word nie, suksesvol gebruik gemaak. Dipproewe deur Bekker (nie gepubliseer nie) in Soeloeland het getoon dat die testsevlieg een van die eerste was om totaal te verdwyn by naturellebeesdipbakke waar die proewe uitgevoer is. Alle ander vlieë het op daardie tydstip nie net van die beeste af verdwyn nie, maar ook by die naturellekrale en hutte.

As die nuwere dippe se beperkinge in die dipbak besef word en arseen altyd as basis gebruik word, dan kan dipping baie waardevol wees om bosluise weer, soos op een stadium die geval was, doeltreffend te bestry. Arseen is goedkoop, stabiel en kan maklik getoets word. Die arseentoets kan gebruik word as maatstaf om gelykertyd met arseen die proporsionele hoeveelheid van die nuwe dipstof by te voeg.

As die spuitgang verder verbeter kan word, want die deeglike benatting van diere bly nog steeds in dié metode van dipstoftoediening 'n probleem, dan kan boere die nuwere dippe alleen sonder arseen in spuitgange gebruik, m.a.w. as dipstof korrek gebruik word da sal baie van ons huidige probleme oornag verdwyn.

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Opsomming

'n Opsomming oor die ontstaan en ontwikkeling van dipstowwe en dipping van beeste in Suid-Afrika word gegee vir 'n tydperk van 70 jaar, en is in drie periodes verdeel, nl. I. 1889-1910; II. 1910-1940, en III. 1940-1959. Gedurende die eerste tydbestek is veral werk gedoen in verband met siektes wat deur die bosluis oorgedra word en die ontwikkeling van 'n effektiewe dipstof, nl. arseen, om bosluise te bestry. Die volgende tydvak kan beskou word as die toepassing van opgedane kennis en uitroeiing van bosluise deur 'n beleid van dipping in arseen. Die laaste periode kan beskou word as die bestande-bloubosluis-tydperk en die ontstaan en ontwikkeling van die nuwe sintetiese organiese dipstowwe.

'n Poging word aangewend om na die bydraes van alle persone te verwys wat meegehelp het om die geskiedenis van dipping in Suid-Afrika te maak.

Summary

A summary is given of the origin and development of dips and dipping af cattle, over a period of 70 years, in South Africa, and is divided in three periods, viz. (1) 1889-1910; (2) 1910-1940; (3) 1940-1959. During the first period work was especially done concerning the tick as carrier of disease and the development of an effective dip, arsenic, to combat it. The second phase can be regarded as one of applying acquired knowledge and enforcing a policy of eradication of ticks by means of dipping in arsenic. The last period can be regarded as the resistant blue tick regime and the origin and development of the new synthetic organic dips.

An attempt is made to refer to all contributions of persons who helped in making dipping history in South Africa.

(A full-length but slightly popularized English version of this paper will appear in Veld, Vol. XX, No. 2 onwards published by Cooper & Nephews S. Af. (Pty.), Ltd., P.O. Box 4557, Johannesburg.

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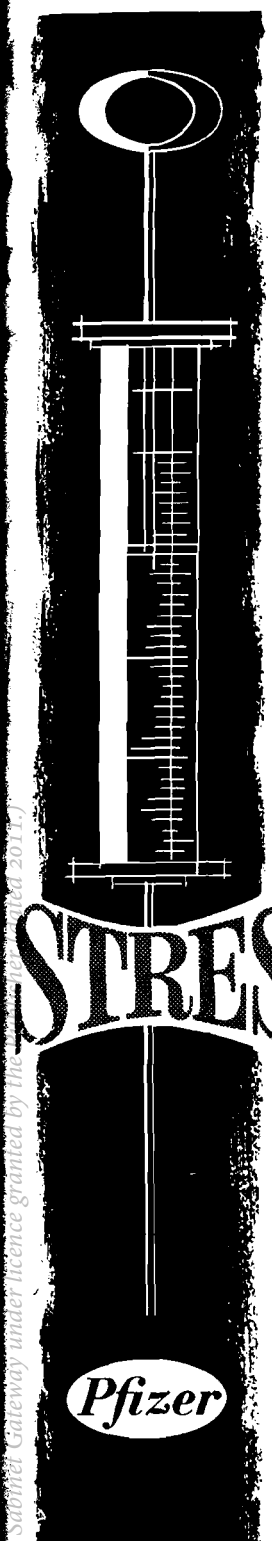
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Ear case T-33-4-1956

COCKER—Male—3 years of age

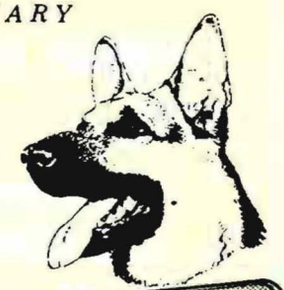
History: Inflammation of ear with odour and extensive head shaking.
Symptoms: Inflammation of external ear canal, typical pseudomonas odour, small laceration in external canal.
Diagnosis: TRAUMATIC OTITIS.
Treatment: Ear cleaned with chlorinated oil solution. METIMYD with NEOMYCIN applied twice daily for 10 days.
Results: Complete healing.



Eye case T-21-4-1956

COLLIE—Female—6 years of age

History: Corneal ulcer measuring 1/16" x ¼", following traumatic injury.
Symptoms: Light sensitivity, discharge, irritation.
Diagnosis: CORNEAL ULCER.
Treatment: METIMYD OINTMENT with NEOMYCIN applied twice daily for 12 days. On first and third days 5 cc. of foreign protein were given.
Results: Complete repair of ulcer with slight scar formation.



Skin case P-72-2-1956

GERMAN SHEPHERD—Female—2 years of age

History: Dry eczema of two months duration.
Symptoms: Dermatitis and alopecia involving left labial commissure and left external ear flap. Severe pruritus of involved areas.
Diagnosis: DRY ECZEMA.
Previous Treatment: Sodium caprylate solution and sulfa ointments.
Treatment: METIMYD OINTMENT with NEOMYCIN applied twice daily for one week.
Results: Pruritus ceased after the first application of METIMYD. Within one week all symptoms of dermatitis had disappeared.

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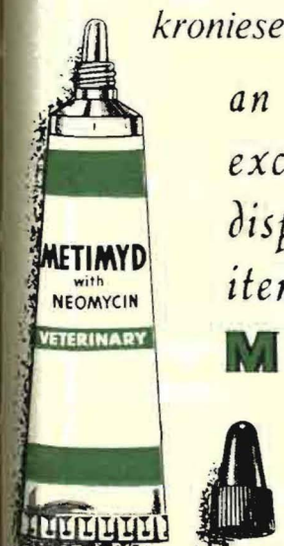
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"INTRA-ARTICULAR AND INTRA-THECAL PREDNISOLONE IN THE TREATMENT OF TRAUMATIC INFLAMMATION OF SYNOVIAL STRUCTURES IN EQUINES."

(Published in the Jl. S.A.V.M.A. 30(3) 1959, pp. 235-245.)

Author's reply to discussion of Paper

I regret I could not be present at the Congress and wish to thank Prof. Hofmeyr for having presented the paper for me. In reply to those who took part in the discussion, Prof. Hofmeyr states: "It was not exactly clear what was considered a "breakdown" or how long the cases were followed up." I did not use the word "breakdown." I used "recurrence" which indicates that the condition recurred and had to be retreated with a similar or another method of therapy. I stated that I began using Prednisolone in 1957. Many of the cases treated have been under observation since then, namely two and a half years, more recently treated cases have, of course, been followed up since 1957. Many of the cases treated continue to win races in the best company. One of them is probably the best sprinter in the country.

The results of Prednisolone intra-muscular injection and deep needle-point firing may be opposite, as Prof. Hofmeyr says. However, the use of Prednisolone as soon as possible after the accident reduces the duration and the intensity of the pathological phenomena. One would never think of firing an acute inflammatory lesion. Even where actual cautery is considered, the use of Prednisolone allows such intervention much earlier than would otherwise be possible, a laudable outlook. I use Prednisolone with this object. It is quite unnecessary to use X-ray examination as a routine except in cases where complications are a possibility, such as fracture of the sesamoid, carpal or tarsal bones. Even in these cases it is usually possible to make a diagnosis without X-ray examination. I use X-ray examination infrequently. I find it unnecessary.

I have indicated (par. 1, p. 239) that I have discontinued treating chronic cases with Prednisolone and use actual cautery with drainage alone or in combination with intra-articular corticosteroids. I make no claim that Prednisolone will cause resorption of an exostosis. In these chronic, frequently neglected cases, prognosis is considerably helped by careful local palpation for pathological change and abnormal physiological function. The synovia is always examined. If there is acute lameness, with blood-stained synovia, Prednisolone is used in preparation for actual cautery. If the synovia is opaque, without haemorrhage, actual cautery is proceeded with, but not before acute lameness has been treated by the usual methods stated in the paper.

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To finalise my reply to Prof. Hofmeyr's remarks, the local anatomical and physiological picture dictates the course I adopt.

REPLY TO DR. STEYN: I think the pathology of joints, tendon-sheaths and tendons in race-horses can quite reasonably be compared to similar conditions in athletes. I cannot agree that "the criterion of success was often relief of pain and discomfort". Atheletes wish to resume normal activity in the shortest possible time: it is similar with race-horses. In attaining this result cortico-steroid therapy has certainly not "failed dismally" (par. 4, p. 237) as stated by Dr. Steyn. It alleviates pain, shortens the inflammatory syndrome associated with acute traumatic synovitis and shortens the period of rest necessary to accomplish resolution. Consequently I consider we have made a noted advance in following the lead given by human orthopaedists in the treatment of synovitis in athletes.

Dr. Steyn admits that the beneficial effect of the cortico-steroids is based p.imarily on their "so-called" anti-inflammatory effect: why "so-called"? It is factual. Referring to paragraph 1, p. 243, Dr. Steyn's reasoning is unconvincing. The fact that the rapid disappearance of inflammatory changes and with it the disappearance of clinical symptoms of pain surely is an achievement which allows more rapid and complete resolution whether in slight or extensive damage to synovial structures. The first indication is the reduction of tension of the synovial capsule followed by reduction of the phenomena following the original damage. The longer pain and inflammatory phenomena remain, the more likely are we to be faced with unpleasant sequelae, atrophy, adhesion, fibrous tissue, exostosis and subsequent reduction of normal physiological function.

I cannot agree with Dr. Steyn that "the degree of lameness and the observable local reaction are a poor guide to the extent of the damage which has been sustained." In my opinion these are excellent guides, in fact, infallable. High grade lameness and acute local reaction are almost indications of acute sero-haemorrhagic synovitis, a far more serious condition than an acute serous synovitis.

In paragraph 3, p. 243 Dr. Steyn states: "When the chances of failure with any particular type of treatment are as high as 33 per cent., that treatment must be considered a bad treatment." This statement surely needs elucidation. What percentage of complete resolution followed other methods of therapy? What percentage of recurrences followed actual cautery? Are they lower than when a cortico-steroid is used? It is pleasing to note, however, that Dr. Steyn does not condemn cortico-steroid therapy in selected cases. I suggest that these selected cases are all acute and subacute inflammation of synovial structures; also certain chronic cases before applying actual cautery (par. 1, p. 239). I

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find no difficulty in selecting cases that will benefit from cortico-steroid therapy.

Dr. Steyn drawns attention to conformation as a predisposing cause to recurrences. I emphasized this sufficiently in paragraph 5, p. 237. It needs no further expansion. It is sufficient to emphasize that conformational defects will always cause predisposition to pathology of the legs in race-horses, whether it be to the original lesion or to recurrences following resolution. Otherwise what is the aim in breeding good legged horses?

Dr. Steyn mentions four horses which he treated (pars. 5, 6, 7, 8, p. 243) with cortico-steroid injections. According to his record eleven joints or tendon sheaths were treated, namely 3 thoroughpins, 4 knees and 4 fetlocks (11) of these six recovered, 4 improved, 1 did not improve (45%). Two of the horses treated had faulty conformation. a predisposing cause for recurrence. The two horses without faulty conformation recovered, apparently without recurrence. However, Dr. Steyn does not mention that firing also failed to produce any improvement in one of his cases of thoroughpin. Whether it was successful in his cases of recurrence in carpitis and arthritis of the fetlocks is not mentioned.

With reference to Dr. Steyn's remarks in paragraph 3, p. 244, the few cases mentioned cannot be conclusive in analysing the results of firing generally. If horses are fired early, before pathological changes involving organisation of connective tissue have taken place, there is little or no reduction in joint movement. Cortico-steroid therapy helps considerably in preventing this unhappy sequel to synovial inflammation. I can, for example, mention a horse, fired as a two-year-old, that won the biggest handicap in South Africa as a four-year-old, and another, fired at the same age, that won the Durban Gold Cup, while another won the Cape of Good Hope Derby. Practitioners engaged in equine practice have hundreds of such cases on their records. This may indicate that firing is an excellent method of treatment for certain pathological conditions, but the results do not allow us to conclude that the horses would not have been better if treated at the correct time with cortico-steroid injection.

With regard to the use of cortico-steroids in the presence of infection, septic bursitis and arthritis, I maintain that the procedure is justified for the reasons indicated in the paper (par. 7, p. 239). Its advantages outweigh its disadvantages. I use it with excellent results. I have been lucky enough to avoid the disasters which Dr. Steyn suggests.

REPLY TO DR. IRWIN: I have written a personal letter to ask what is meant by distending the capsule before withdrawing synovia. Perhaps Dr. Irwin means local distension, not general distension (see par. 5, p. 240). Local protrusion of a joint or tendon sheath is done by digital pressure.

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NAME OF ANIMAL **F. VETALENTA 2ND**

DATE	REMARKS
7·10·52	Calved (Heifer calf)
1·1·53	Served (B. Flash Prince)
9·10·53	Calved (Bull calf)
27·12·53	Served (B. Flash Prince)
17·1·54	" " " "
12·2·54	" " " "
	Injected 'Lutormone' — 1500 I.U. I/V.
28·2 54	Injected 'Protormone' — 10 c.c. I/m.
19·11·54	Calved (Bull calf)



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TORSION OF THE STOMACH IN THE DOG

E. M. HEARN and M. E. KEEP*.

(Paper presented to 54th Annual Congress, 26th August, 1959)

We feel justified in introducing this subject briefly, mainly because it is one which is rarely referred to in canine surgical literature. In all cases of torsion of the stomach rapid surgical interference is essential to save the patient, consequently the condition must be diagnosed immediately.

During the last three or four years we have seen seven cases, all of which have presented a fairly typical picture on examination. They have all been old dogs with a history of having been fed four or five hours previously. The most noticeable symptom is extreme distress, with obvious acute abdominal pain. The abdomen begins to swell, particularly on the left side, just behind the ribs, and respiration becomes progressively difficult, due to excess pressure on the diaphragm. In some cases retching occurs at this stage, but no stomach contents are vomited. In a very short time, the patient becomes cyanotic, with increasing tension in the abdomen and difficulty in breathing. Collapse and death follow if no treatment is commenced.

It must be emphasized that some immediate relief must be given if the acute stage has been reached when the patient is first seen. A long medium-bore exploratory needle is used to puncture the blown stomach, on the left side at the point of greatest curvature. Foul smelling gas is immediately released. A large bore needle or trocar should not be used as the pressure on the diaphragm must be only slowly released, otherwise death may ensue, apparently from shock. After the gas has been slowly withdrawn, the patient's colour and respiration will be seen to improve and the dog is now ready for attempted correction of the stomach torsion.

The left side of the dog is shaved and the skin cleaned. Anaesthesia is induced using thiopentone and immediately the dog loses consciousness, a McGill tube is introduced into the trachea and inflated. A stomach tube is also passed as far as the cardiac entrance of the stomach where, of course, it is obstructed — this also confirming one's diagnosis. The stomach tube is left in position.

A long incision, large enough to introduce one's hand, is made in the left flank just posterior to the ribs. On opening the abdominal cavity, the distended fundus of the stomach is seen, and usually,

*P.O. Box 1, Sandown, Johannesburg.

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running across it the greatly congested gastric vessels. The spleen, which is normally visible on the left side, cannot be seen. It has been found, in all the cases seen, that, looking anteriorly, the torsion is in a clockwise direction, causing complete blockage of the cardia, and strangulation of the gastro-splenic vessels. Hence, to correct the twist, the stomach must be rotated downwards, towards the operator, and this usually requires considerable manipulation with both hands. If a fair amount of gas has been withdrawn from the stomach before the operation, this manipulation will be found to be much easier.

As soon as the torsion is corrected, it will be found that the stomach tube passes easily into the stomach. Usually a quantity of gas and froth is immediately brought up. The spleen automatically returns to its normal position.

The abdominal wound is closed in the usual way, using interrupted sutures of No. 1 chromic catgut for the muscle layers and mattress skin sutures of braided nylon. When the dog comes round from the anaesthetic and sits up, the residual gas in the stomach is automatically brought up. Routine post-operative treatment includes a light non-bulky diet for a few days and daily penicillin injections.

Here are a few notes on actual cases treated:—

1. A twelve-year-old spaniel was first operated on for torsion in April, 1957, and recovered uneventfully. During this operation, which was the first of this kind we had attempted, it was found that not sufficient gas had been let out of the stomach before operation, and more had to be withdrawn using a long hypodermic needle directly through the stomach wall after the abdomen had been opened. A very blown stomach is almost unmanageable and cannot be turned, so it must be considerably reduced in size before operation.

In November of the same year the condition recurred in this dog, and, during the course of the second operation, the stomach was anchored in position by suturing its serous coat to the peritoneal lining of the abdomen by two or three gut sutures to try and prevent a further recurrence.

2. A nine-year-old Boxer bitch was presented with all the usual symptoms and already cyanotic. When the stomach was punctured prior to operation, it was found that very little gas escaped, although the tympany was very acute, and it was obvious that the needle was continually blocking. On opening the abdomen and puncturing the stomach wall with a larger bore needle, nothing but some froth oozed out. It was decided to open the stomach and empty it, and after packing off the stomach as carefully as possible, this was done. About three-quarters of a bucket of froth and fermenting stomach contents was removed, before the stomach was sutured and its torsion corrected.

3. An eleven-year-old Great Dane which was presented,

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enormously bloated. The operation was performed, the stomach tube having been felt right in the stomach before the abdomen was closed. Only a little gas escaped through the tube, however, and whilst the dog was still lying on its side, the stomach obviously still contained a lot of gas. The next morning, however, the abdomen was quite normal in size, the residual gas having passed during the night.

4. An aged Bull Mastiff was presented too late and died from respiratory failure on anaesthetization. In an attempt to stop this occurrence we now use oxygen and carbon dioxide connected to the McGill tube, so that at the first sign of respiratory failure the lungs can be ventilated using positive pressure.

Lastly, we should be very pleased to hear any discussion as to the possible causes of torsion. What set of conditions sets up the violent contractions of the stomach wall which result in a torsion? In some of the cases we have seen, there has been a history of some sort of excitement after a meal, but this is not a constant feature. The type of food given has varied, but in all cases there has been a considerable amount of fermentation of stomach contents. Whether this occurs *after* the torsion, or whether the fermentation *starts* violent peristaltic movement and consequent torsion, we do not know.

DISCUSSION.

DR. D. G. H. IRWIN (opener) after briefly recapitulating the topography of the stomach, continued:

"I had a lot of trouble trying to find out how the stomach, in fact, twisted. As the anterior end of the duodenum is fairly rigid, the stomach could make a backward somersault, which would cause rupture of either or both of the greater or the lesser omenta. I could also imagine a fulcrum or an axis through the ninth thoracic vertebra, in which case the stomach could twist clockwise (looking from underneath).

"I would like to say that the age of the animal is not necessarily advanced. It has been said that it may occur in any animal from the age of three to eight years and that it is usually confined to large dogs. If we can call this a disease of any one breed, then I think it would probably be the Great Dane. The only case which I have recognized in practice was in a member of that breed, although it has been seen, as we heard here this morning, in a spaniel. Heuscher's experience was that they were distributed as follows: One Alsatian, one Boxer, one Bulldog and four Deutsche Doggen (Great Danes).

"According to Heuscher *et al,* it is usually a case of a grossly distended stomach with a maximum amount of fluid (they insist on the fluid). The occurrence starts as a pendulum action, with an animal exercising shortly after having a large meal. The one I saw was vastly distended, in agreement with the speakers here this

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morning. Stungy *et al* reckon that there is not a great deal of distension because the rib cage prevents that.

"I like the notion of passing a stomach tube, so that immediately the stomach is twisted straight you can have an aspirating effect outwards so that you don't have to puncture the stomach to drain the fluid. I think it is possibly wise to anchor the stomach once having corrected it, just as in one of the methods described and which I know works well, for the replacement of abomasal displacement. I would be inclined, in theory, to plump for anchoring of the stomach, because once those ligaments have become loose enough to twist once, well, why shouldn't it happen again?"

DR. J. DORE: Mr. President, if I can recall from memory, I think I have seen six cases — three were Alsations, one was a Dobermann, one was a Dobermann cross. I think I have seen somewhere in the literature that this condition is usually found in large dogs. I was surprised to hear that a dog so small as a spaniel was a victim of stomach torsion.

One thing I did encounter in three of these cases was that there was a considerable enlargement of the spleen. Whether the spleen had acted as the pendulum for turning that whole stomach over, I don't know.

The other experience I would like to mention is the following: This dog came in merely for an ear treatment; it was a little bit restless on the table, it then went home by car and then the owner phoned me to say that the dog was very, very ill and swollen up. I told her to come straight in and when she arrived, approximately 20 minutes after she had left me, this dog was on the point of death. I did pass a fairly big trocar to relieve the gas (the dog was considerably swollen), but it died shortly afterwards. Probably I used too big a trocar to aspirate all the air from the stomach. I didn't mention the Chow—that was the first case that died within twenty minutes—that brings my total to six.

In regard to the operation, I found that it was necessary to remove the spleen in two of these particular cases. I have not in any of them anchored the stomach back into its normal position, but I have found that merely removing the spleen in these cases, has been sufficient to keep the stomach in its normal position and I have had no recurrence of the condition in any of the patients that I've had.

DR. MORFORD: I have not anything very much to add, except that my experience has been very much like Dr. Doré's. They have all been large dogs, except one, which was also a spaniel — but it was a very big spaniel. I find the same thing, that in most cases the spleen is very enlarged. We found that taking out the spleen seemed to have no ill effect on the dog and we have had no recurrences at all.

I think that this condition occurs very much more often than we see, because this is a very quick death. I think a large number of

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dogs die of this and are never brought to veterinary surgeons at all —I don't think there is time for that. I don't ever remember seeing it in England at all, although it probably occurs. Every now and again we get these cases cropping up and I do think it is quite common.

PROF. DE BOOM: I think the one predisposing factor is probably a lengthening of the lesser omentum, or pre-existence of an abnormally long lesser omentum. Probably in the older dog repeated overfeeding, especially if the dog is habitually a voracious feeder and the stomach is rapidly filled, causes tension on that omentum and thus lengthens it, or it may already congenitally have a fairly long omentum.

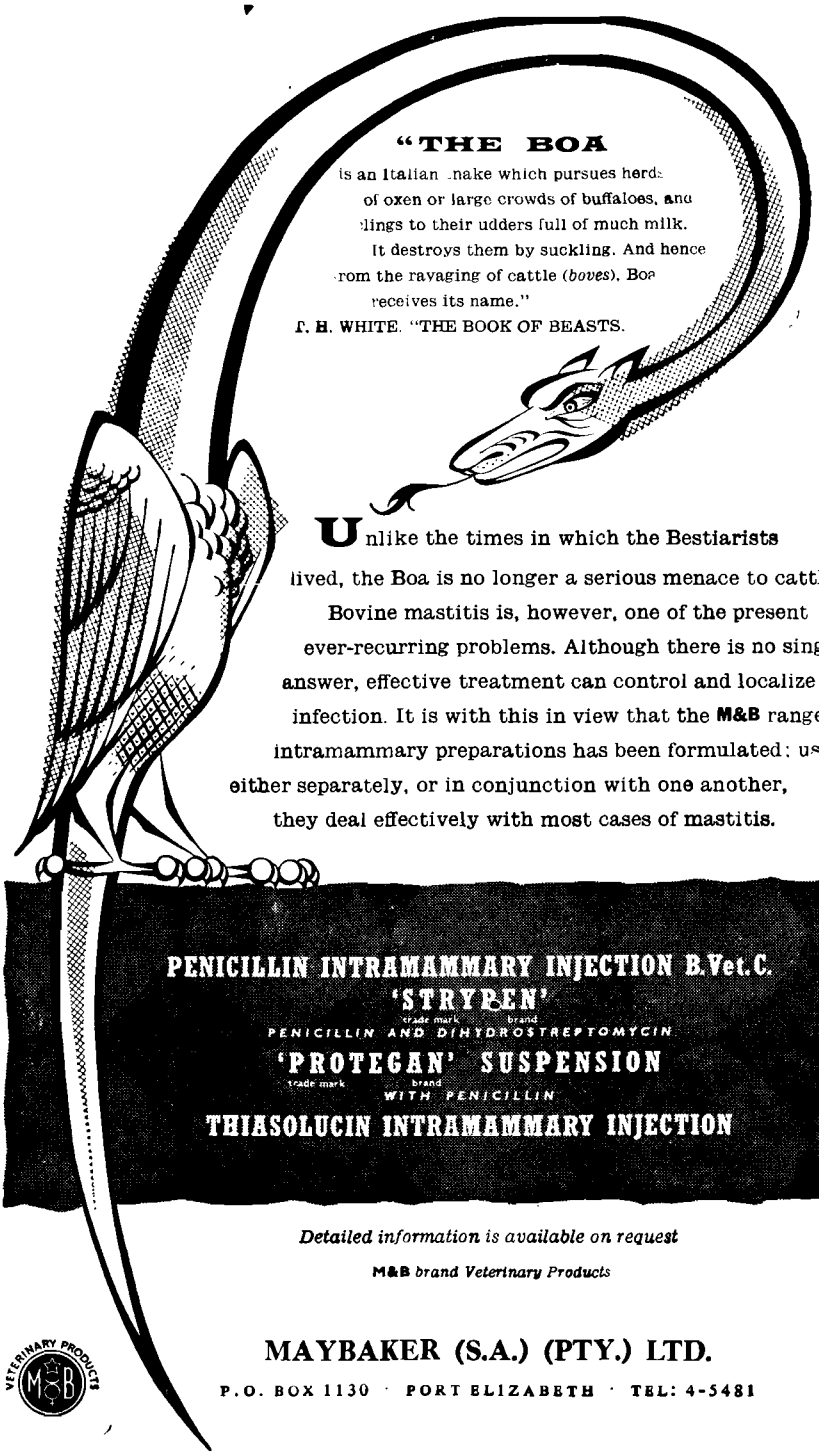
I wish to recall a few experiences we have had with police dogs. It was invariably the case that they had been fed, and then taken out to practise. They had had to jump down from the lorry which transported them, from quite a height, and there the trouble started. The other case I know of was where the dog had also had a fairly big meal and then started playing with the children, rolling on the grass. I think that mealie meal feeding could have a lot to do with it; it is quite a heavy, cement-like substance, which rapidly fills the stomach. I think that big meals, fed rapidly — in other words, hungering the dog for a long time and then giving it big meals — is definitely a predisposing factor.

On this question of the enlargement of the spleen, I can imagine a certain amount of enlargement of the spleen when you start feeding, due to filling of the splanchnic area, but undoubtedly the greatest enlargement must be due simply to the fact that the splenic vein, which is quite big and lies just at the point of torsion, is closed off after torsion of the stomach.

DR. KEEP: I must admit that we have not seen this excessive enlargement of the spleen. There is no doubt that we have seen enlargement, but I think if you can correct the torsion and then wait for a short while, you will find that, by correcting the torsion, you do relieve the congestion of the splanchnic vessels, and the spleen probably will (in our experience, it certainly does) reduce in size to such an extent that you can close the dog up without having actually to amputate the spleen. I am very glad that this point was made and that mention was made of amputation of the spleen. We have not met with that and now we shall know what to do if we do get stuck over the spleen.

MR. PRESIDENT: I don't think it is really incumbent on me to say anything, but there is just one remark I feel I must make, and that is, to remind you of the danger of biliary fever in your splenictomised dogs.

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"THE BOA

is an Italian snake which pursues herds of oxen or large crowds of buffaloes, and clings to their udders full of much milk. It destroys them by suckling. And hence from the ravaging of cattle (*boves*), Boa receives its name."

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ADVANCES IN "GEELDIKKOP" (*Tribulosis Ovis*) RESEARCH.

2.—FIELD INVESTIGATIONS — THE MOBILE LABORATORY AND EXPERIMENTAL FACILITIES.

J. M. M. BROWN.*

(*Received for publication October,* 1959)

As indicated in the first paper of this series the toxic principles present in *Tribulus terrestris*, the plant believed to cause the characteristic symptoms of "Geeldikkop", have proved singularly elusive and all attempts to isolate such agents so far have met with failure.

Because the disease occurs in areas far removed from the research laboratories, much of the early field work depended on dosing and grazing experiments with the plant. Chemical work on the plant was performed on specimens brought back to their laboratories by the investigators concerned. During recent years it was realised that much of these detailed studies would have to be carried out in the field on the affected farms and at times when outbreaks of the disease were occurring. Not only was it essential that the chemists concerned with the isolation of active principles from the plant work on freshly collected material and also be able to study daily variations in the plant itself, but facilities were also required for a comprehensive study of the chemical pathology of the disease.

On consideration of the feasibility of investigations along these lines, it was obvious that the difficulties which would have to be surmounted were considerable and in some cases even formidable.

Temporary laboratory facilities would have to be created for the various workers at some point in a vast area of this country, where, although the roads were reasonably good, distances between individual farms and between these and the nearest towns were considerable. The area is poorly served by railways and postal communication. To add to these difficulties the appearance of the disease during the summer months is most erratic and the focal points of outbreaks were continually shifting. Daily temperatures at this time of the year are usually very high and subject to marked fluctuations. In many parts of this area the water has a very high mineral content which renders its use in chemical work most problematical.

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Investigations of the nature envisaged were finally made possible through the very generous loan to this Institute, by the Trustees of the Stock Diseases Research Fund, of a mobile laboratory. The specifications for this vehicle, which was built at a cost of approximately £4,500, were drawn up by a joint committee of the personnel from Onderstepoort and the Council for Scientific and Industrial Research, responsible for the Geeldikkop research project. The laboratory was completed in almost record time, the chassis and body being assembled by a private firm in Johannesburg and the interior fittings were designed, assembled and installed in the C.S.I.R. workshops in Pretoria.



Fig. 1. *The Mobile Laboratory.*

The unit, which is constructed upon a bus chassis, has an empty weight of 14,780 pounds and is capable of carrying a load of 3,220 pounds, an important factor when the large amount of equipment needed for these investigations is considered. The cab offers comfortable seating accommodation for at least three persons. The laboratory is entered through double doors on the left side and has an internal area of 105 square feet. It is air-conditioned, providing working temperatures of 60-80°F. even on the hottest summer days. Although not yet installed, it is intended to include a heating system to provide comfortable working conditions during cold weather.

Adequate bench space is provided for at least three persons, and the bench surfaces are of acid proof, heat resistant material. Considerable storage space is provided by lockers, situated below the benches (one of which is fire proof) and at eye level running the length of the laboratory on each side. External lockers are provided for camping equipment, tools and other such items.

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A large refrigeration unit incorporating a deep freeze occupies part of the space beneath the bench on one side and electricity is provided by a generator situated at the rear of the vehicle. This 10kw. generator, which delivers 220 to 250 volts A.C., makes the unit independent of local supply of electric current.



Fig. 2. *View of the interior of the Mobile Laboratory.*

Laboratory heating facilities are further provided in the form of butane gas, the cylinders being placed in one of the lockers below the benches. Two electrically operated high vacuum water pumps are installed for chemical work. The laboratory is supplied internally with water from a large tank holding 50 gallons slung beneath the chassis. This water is pumped manually into a smaller roof tank, and is available at a sink set in one of the benches.

The mobile laboratory was used for the first time in the late summer of 1958 in investigations on Geeldikkop in the Vosburg district. This trip, although more in the nature of a trial run to test out the potentialities of the unit, yielded much new and valuable information concerning the disease. The unit was used again a few months later during investigations into abortion amongst Angora goats in the Somerset East area.

From the experience gained on these two investigations we were able to exploit the potentialities of the unit to the fullest in the recent investigations on Geeldikkop in the early summer of 1958

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and in January of this year, in the Victoria West district. It had become obvious that the most satisfactory manner of using the laboratory was by establishing it in some centre which was the focal point of severe outbreaks of the disease and then using independent motor transport for the purposes of collecting plant specimens and material from sick animals. The most suitable site for placing the laboratory was considered to be on a showground, which is to be found in most of the major centres in this country. Such an arrangement not only allows of the use of the local power grid and water supply (the former was found to be more economical than operating the generator installed in the unit) but also the use of pens and stalls for the accommodation of experimental animals. Additional laboratory and storage space can be found in the various halls and pavilions found on such premises. This latter consideration was important, for the large amount of work done and the variety of tasks performed by the various members of the staff demanded much extra space. The mobile laboratory then serves as the centre of operations, from which power may be drawn and where specialised chemical work (e.g. entailing the use of the vacuum pumps or distillation apparatus) may be done and sensitive electrical equipment operated.

Besides the facilities offered for the present investigations, the unit provides wide scope for detailed studies in other fields, e.g. virology, bacteriology, pathology and many others. Indeed, during the last two investigations, such studies were actually made, a small incubator, sterilizers and a frozen section microtome being included in the portable equipment. A large capacity pressure cooker could substitute quite well for a small autoclave.

The problem of obtaining distilled water was overcome by including in the equipment carried a very efficient de-ionizing apparatus, but it was still found essential to pass fairly pure water, such as rain water, through the de-ionizing columns. The mineral content of the underground water in many parts of the Karoo was so high that the efficiency of the de-ionizing resins fell rapidly when such water was used.

The nature of the work conducted during the three investigations mentioned entailed carrying a considerable amount of equipment, all of which could be comfortably stored in the laboratory portion of the unit during transit. Because of the scarcity of postal and railway communications and the distance of the affected areas from the home laboratories, careful planning of the programme was required. Chemicals and other consumable stores sufficient for the requirements of the work had to be carried and various eventualities which might arise had to be considered. For instance, it was usual to include small supplies of chemicals from which many of the commonly used reagents could be prepared should the need arise. Besides being familiar with the normal maintenance and servicing of all the equipment used it was also essential that the team members

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be able to effect minor repairs to this equipment, and that spare parts be carried where possible.

On arrival a telephone was installed, allowing easy communication with the farmers in the affected areas as well as with the home laboratories or other centres. The local farming community was informed of the advent of the research team by means of messages broadcast during the "Regional News Services". Accommodation for the staff naturally depends on circumstances, but during the past investigations it has been found most convenient to use hotels or boarding houses in the centre where the laboratory has been established.

In lighter vein may be mentioned some of the difficulties which are inevitably encountered when the mobile laboratory arrives at the town where it is to be stationed. This event, as would be expected, always arouses considerable interest and curiosity in the local population and one or other member of the team usually has to assume the rôle of public relations officer. Innumerable visitors arrive to be conducted over the scene of activities, foremost of which are those who invariably know all about the disease being investigated and have discovered cures for it years ago. The farming community must be convinced that the unit is not an itinerant clinic and that the organic chemists present in the team need not be conversant with the intricacies of veterinary obstetrics or the control of internal parasites. Officials of the local school wishing to arrange educational visits for their scholars must be patiently dealt with. "Young hopefuls" must be advised on careers in veterinary science, medicine or chemistry and a watchful eye kept on the local Hottentot or Bantu population who are invariably attracted by the sight of *post-mortem*...examinations.

Although the potentialities of this mobile unit in veterinary field investigations are enormous, it must be stressed that the expenses involved in such work are considerable and its use must be justified by the economic importance of the condition to be investigated.

During the most recent investigation, a considerable amount of experimental work on Geeldikkop was performed, involving dosing experiments with sheep using the various substances isolated from Tribulus and other potential icterogenic agents. Not only were detailed chemical pathological studies on blood, urine and faeces carried out, but for the purposes of the work it was also essential to have accurate measurements of food and water consumption and urine or faeces excretion over a twenty-four hour period. As the laboratory was established on the showgrounds at Victoria West, the sheep were conveniently housed in the sheltered sheep pens available and the judging enclosures were used for exposing the sheep to sunlight during tests for photosensitization. Wethers were used in all the experiments and faeces were conveniently collected using the usual type of collecting bag, employed in metabolic studies, strapped to the hindquarters of the animals. The urine was collected

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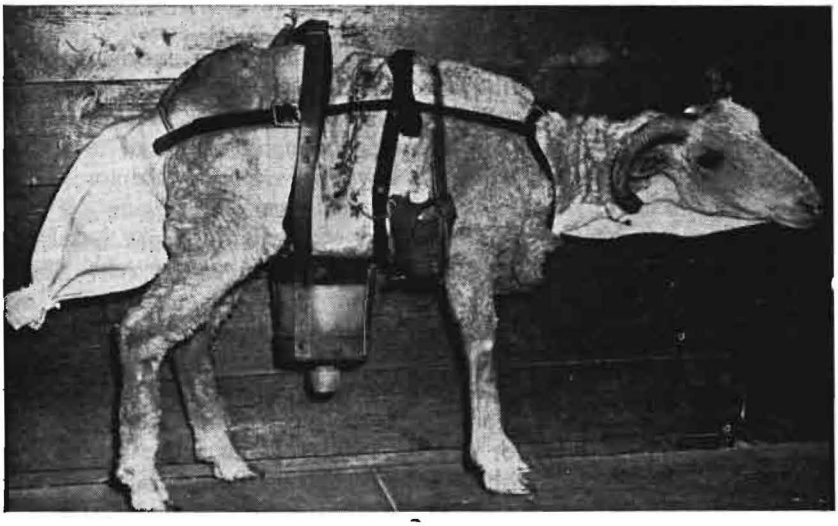


Fig. 3. *An experimental sheep used during these investigations, showing urine and bile collection bottles and faeces collection bag in place.*

in specially constructed polythene collecting bottles. These urine collectors were constructed as illustrated in figure 4, and were made from round two litre polythene bottles, the bottom halves of which were removed. A polythene funnel of the appropriate dimensions was

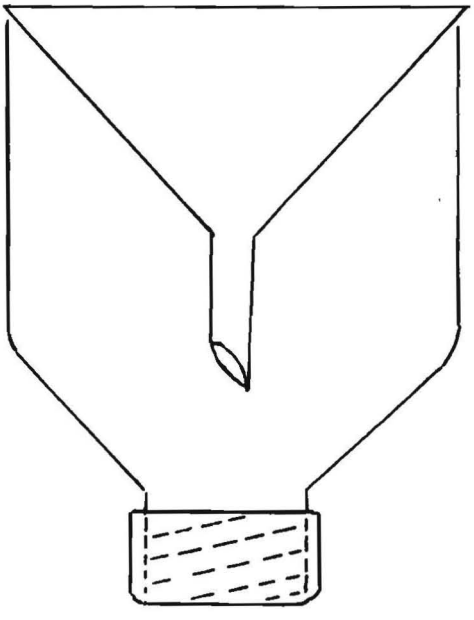


Fig. 4. *Sketch showing construction of urine collection bottle.*

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then fused on to the top half of the bottle as illustrated. This serves to prevent loss of urine when the animal lies down. The collecting bottle was then held in place beneath the belly of the animal by means of a suitable harness, and was usually drained at six-hourly intervals through the screw-cap below.

For later studies, not performed during these field investigations, an external biliary fistula was created in experimental animals by introducing and securing a length of small bore (2 mm.) polythene tubing into the common bile duct and bringing it to the exterior through a small stab wound adjacent to the laparotomy wound through which the operation was performed. The bile was then allowed to flow into a polythene bottle of 250 ml. capacity, suspended against the right side of the sheep by means of a suitable harness. The external biliary fistula, bile and urine collection bottles and faeces bag are shown in position on the sheep in figure 3.

Full details of the observations made during these studies, the chemical pathology of the disease and details of the experiments mentioned will be presented in the appropriate succeeding papers in this series.

Summary.

A brief description of the mobile laboratory used in the recent field studies on Geeldikkop, its potentialities in work of this nature and an insight into some of the organization required for such investigations is presented. Apparatus for the collection of urine and bile during experiments in these investigations is briefly described.

Acknowledgements.

The permission of the Director of Veterinary Services for the publishing of this paper is gratefully acknowledged. The assistance of Mr. R. J. J. Briel (Senior Technical Officer, Department of Physiology, Onderstepoort) in the design and construction of the urine and bile collection appartus is much appreciated. The assistance and advice of Mr. K. H. C. Riley (Senior Mechanical Assistant, Onderstepoort) in carrying out investigations of this nature and the personal enthusiasm and organizing ability of Technical Assistants P. J. de Wet and D. F. Gouws were of extreme value.

The considerable amount of time and thought, which Dr. J. R. Nunn (National Chemical Laboratories, Pretoria), Professor R. Clark (Department of Physiology, Onderstepoort) and many other persons, too numerous to mention, have put into the design and development of the mobile laboratory is remembered with grateful thanks.

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ADVANCES IN "GEELDIKKOP" (*Tribulosis Ovis*) RESEARCH.

3.—THE EPIZOOTOLOGY OF "GEELDIKKOP."

J. M. M. BROWN.*

(*Received for publication, Oct., 1959.*)

As was indicated in the initial paper in this series[1] the classical "Geeldikkop" syndrome is a disease of small ruminant animals, characterized by a severe photosensitization, intrahepatic cholestasis and in most cases a severe icterus, severe renal lesions, uraemia, blood electrolyte disturbances and certain blood cell dyscrasias. It occurs in most parts of the Karoo, the North West Cape Province and Southern Orange Free State amongst sheep grazing on the low shrub, bushes and pioneer grasses typical of the vegetation of these areas, and is believed to be caused by the annual plant *Tribulus terrestris*.

For the purposes of this paper the condition is to be distinguished from the closely related photosensitization syndrome known as "dikoor" (Thick ears), a condition occurring in the central and North Eastern Free State, the Transvaal Highveld and certain areas of Natal, chiefly amongst lambs grazing on *Panicum* species, and described by Steyn [2,3,4], Quin [5] and Rimington and Quin [6].

It must also be distinguished from the photosensitization syndrome seen sporadically amongst sheep grazing on oats and lucerne and also from the ill-defined condition known as "lugdikkop" encountered in certain areas of the Karoo. As a clinical entity typical "Geeldikkop" is quite distinct from Enzootic Icterus, notwithstanding our present belief that the two conditions may be very closely related as regards their aetiology.

"Geeldikkop" is a disease which makes its appearance and subsequent disappearance in a most insidious manner. It usually makes its appearance in the summer months within a few days after the first summer rains have fallen and especially if these are followed by spells of hot dry weather. The disease is usually prevalent in the affected areas from November to March, the peak incidence occurring in the latter part of December and January, depending on the rainfall.

Outbreaks of the disease vary considerably in their extent, severity and duration from time to time. It may appear on isolated farms in a particular district, or only in certain camps on such farms.

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Whole districts within what are regarded as the endemic areas are often unaffected, and often a few years may pass between outbreaks in such areas. On the other hand the disease may make its appearance in frightening proportions affecting vast areas in the parts of the country mentioned, with barely a single flock escaping its ravages, and appearing in districts where apparently its has been unknown before. An outbreak on such a scale occurred during the late summer of 1958 and January of 1959, whereas the outbreak during the previous summer was of a comparatively mild and restricted nature.

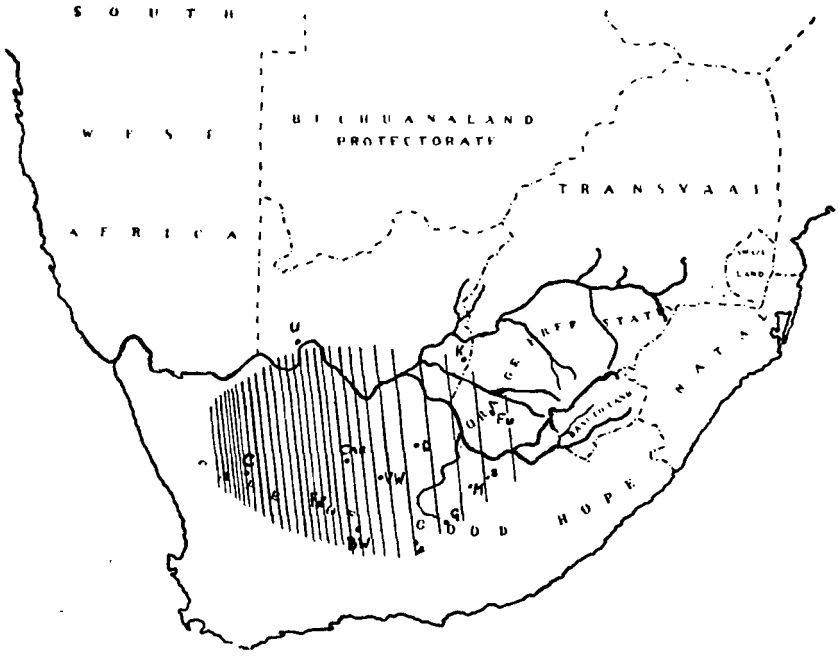


Fig. 1.—Map showing approximate incidence of "geeldikkip" in the Union.

KEY

A	Aberdeen.	G	Graaff-Reinet
B W	Beaufort West	K	Kimberley
C	Calvinia.	M	Middelburg.
Car	Carnarvon.	S	Steynsburg.
D	De Aar.	U	Upington.
Fa	Frazerburg.	V W	Victoria West
Fu	Fauresmith.		

Parallel shading indicates severity of geeldikkop.

The areas which have been involved in outbreaks described in the literature, reviewed in the first paper[1] of this series, are shown in figure 1. They include the greater part of the Karoo, Cape Midlands, North Western Cape Province and Southern Free State. It is of considerable interest to note that outbreaks of the disease were described by the Colonial Veterinary Surgeon and his staff, at the turn of the century in the Khoup and Beaufort West districts,

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while Quin performed much of his experimental work on the disease at Celeryfontein, south of Fraserburg and at Burghersdorp. From the information at our disposal it appears that the disease is almost unknown in these areas during the present times. The focal point of the outbreaks seems to have shifted to the Northwest, and the areas in and around the triangle formed by Victoria West, Vosburg and Britstown are by far the worst and most regularly affected. Most of the present investigations have been conducted over the last five years in these areas. The disease also never seems to have appeared again on the scale decribed by Theiler [7] in the Southern Free State from 1916 to 1918.

During the late summer of 1958 and January of 1959, in one of the most severe outbreaks of the disease known, it made its appearance on a great scale in the Hofmeyr and Teviot districts of the Cape Midlands on farms where it was unknown before. These areas lie some two hundred miles east of the usual focal centre of the disease. Few cases of the disease were reported at this time from the intervening areas.

A point of considerable interest is that today we regard the vast tract of country South and South East of Fraserburg, adjoining the Khoup as being the chief area in the Union where Enzootic Icterus occurs. "Geeldikkop" as it is known to us has been entirely supplanted by this disease in these areas.

The area in which "Geeldikkop" now appears annually in the severest outbreaks is, as mentioned earlier, the triangular tract of country bounded by Victoria West, Vosburg and Britstown. It forms part of the so-called Central Upper Karoo, and occupies the central part of the upper plateau south of the Orange River.

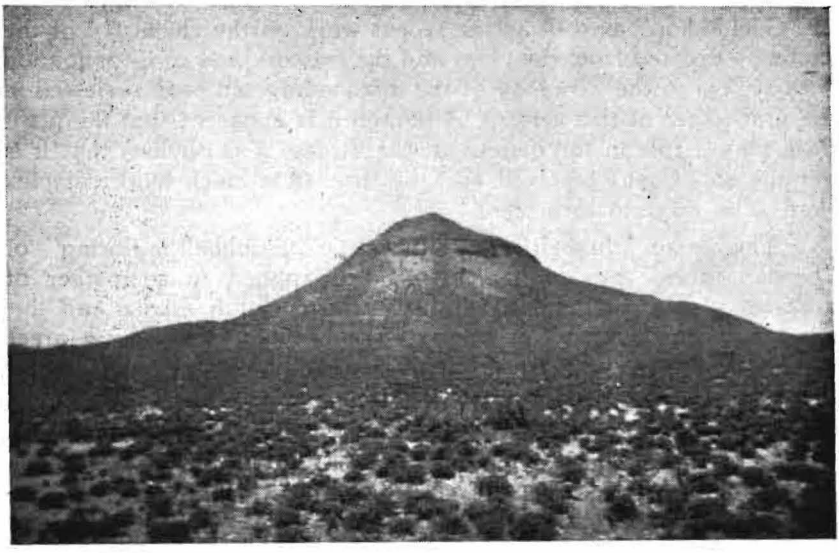


Fig. 2. *Typical veld of the Central Upper Karoo.*

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Altitudes range from 3,500 feet to 5,500 feet above sea level, and the annual rainfall varies from seven to ten inches, most of which falls in the latter part of summer. It is mostly flat country wherein dolerite hills and low ranges of hills and mountains are scattered, especially to the South West. In general the plains are of a stony nature being covered with shale, sandstone and calcareous tufa, although in parts they are covered to a shallow depth with red sandy loam, and along the dry rivers and watercourses wide silt flats or flood plains are often seen. The hills and mountains are often more grassy than the plains. The vegetation consists of a wide variety of the hardy Karoo bushes, the dominant species on any particular farm often being one or more of the following: *Eriocephalus*, spp, *Salsola* spp, *Pentzia* spp, *Pteronia* spp, *Nestlera* spp or *Chrysocoma* spp. Grasses, where they occur, are usually the hardy pioneer types like various *Eragrostis* and *Aristida* spp.

Trees are almost completely absent except around homesteads, and such shade that is available is provided by rocky outcrops, or by taller shrubs such as various *Lycium* and *Rhigozum* species, which occur on the flood plains or on and around the hills. Further West the country becomes more arid and featureless, the soil poorer and the rainfall less. Further East it becomes more grassy and complete grassveld areas may be found in protected places.

The cause of "Geeldikkop" for many years has been considered to be the well known annual plant *Tribulus terrestris* L. (Zygophyllaceae), the "Devil's thorn" or "dubbeltjie", which grows most abundantly in the areas where the disease makes its seasonal appearance. It is, however, also widespread and abundant in many areas of South Africa where the disease does not occur. The early work which led to the incrimination of this plant as the causal agent of "Geeldikkop" as well as the recent work on the chemistry of the saponins isolated from the plant and the present lines of investigation with regard to the aetiology of the disease have all been reviewed in the first paper of this series.[1] Although it is apparent that the plant does play a rôle in the genesis of the disease it is obvious that it is not the sole agent involved and the disease is much more complex than was hitherto suspected.

The name "dubbeltjie", "duwweltjie", "dubbeltjie-doring" or "Devil's thorn" has unfortunately been applied to a number of different plants growing in various parts of South Africa and this has led to some confusion amongst the farmers especially in areas where the disease only occurs sporadically, and as a result some of these plants have been incriminated as the cause of "Geeldikkop". The best known of these plants are the following:

(a) *Emex australis* Steinh (Polygonaceae): This plant which is quite distinct from *Tribulus* in appearance has at various times been regarded as the cause of "Geeldikkop", and many farmers in the Cradock district especially, still believe that it is. According to Watt[8] this has been disproved.

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(b) *Emex spinosa* (Polygonaceae): Also known in Xosa and Zulu as "in-Kunzane" and in Afrikaans as the "Dubbeltjiesblaar". This plant is believed to have a purgative and diuretic action and is used by the Xosa's as a vermifuge for horses. It does not cause "Geeldikkop".

(c) *Oxygonum sinuatum* (Polygonaceae).

(d) *Alternanthera repens* (L.) Link (Amarantaceae): Both these latter two plants are well known weeds, the last named being usually referred to as the "Khaki-dubbeltjie". They do not appear to have been proved toxic in any way and it is very doubtful whether the last-named plant would be eaten at all, owing to its very fine thorns borne in abundance, which cause a marked burning sensation on piercing the skin or mucous membranes.

Various other plants have from time to time been blamed by farmers as the cause of "Geeldikkop". It is believed by some that the plant *Hertia* (*Othonna*) *pallens* O. Kuntze ("Springbokbos") is the cause of outbreaks of the disease which sometimes occur in the late Winter and Spring. Preliminary dosing experiments conducted recently on rabbits using the powdered root bark of the plant and alcohol extract of the finely ground plant have so far yielded no significant findings. Van der Walt and Steyn[9] reported that *Asaemia axillaris* (Thunb) Harv. (Compositae) the "vuursiektebos" may be responsible for some outbreaks of the disease in the Karoo, but no further work has been conducted on this plant. *Pteronia pallens* L.f. the "Scholtzbossie" or "Witbossie" is thought by many farmers to be the cause of "Geeldikkop" and by others to be the cause of Enzootic Icterus. We have found, in preliminary dosing experiments conducted on rabbits, that after prolonged dosing it may give rise to a chronic catarrhal cholangitis and cirrhotic changes in the portal tract. No evidence of an intrahepatic cholestasis, such as seen in Geeldikkop was found.

Tribulus terrestris L. (Zygophyllaceae) also known in certain areas as "Môrester", "volstruisdoring", thsêhlo" (Sotho) "sekanama" (Tswana) is a laxly branched prostrate herb, which in its early growth stages is of upright habit attaining a height of about three to four inches. During its later stages of growth it sends out numerous long prostrate stems which may attain a length of six feet or more, becoming densely covered with leaves, small yellow flowers and the peculiar sharp "dubbeltjie" or thorny fruit. It usually makes its appearance shortly after the first of the summer rains have fallen, in November or more generally in December. It is an extremely hardy plant and is most frequently encountered on fertile alluvial soil of the flats, around homesteads, kraals, gates, watering points and "tramped out" areas on the farms. The seed is widely distributed by flood waters and germinates with the first moisture. If further rains should fall within short intervals the plants develop rapidly and the long prostrate stems form a dense carpet on the ground where the plant is abundant.

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In its early stages of growth the plant is exceedingly well eaten by sheep, who show a marked preference to it above some of the other apparently more tempting bushes and shrub which also

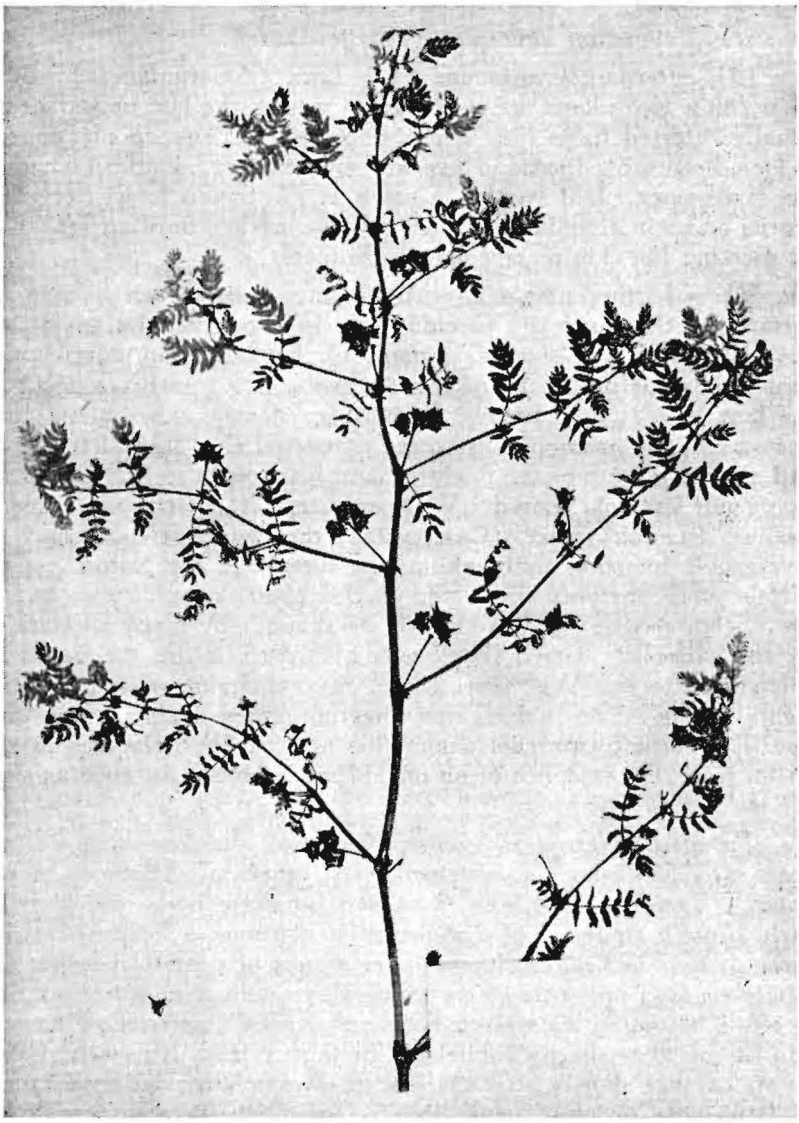


Fig. 3. *Tribulus terrestris L. (Zygophyllaceae) — a fruiting specimen.*

abound in these areas. A very well known fact, and one which we have also observed frequently, is that sheep will, when the plant is becoming rather scarce after heavy grazing, scrape the soil away from the roots of the plant with their forefeet, to eat even these. It

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is still eaten in its later growth stages, when the long recumbent stems have formed; but it does not appear to be quite so attractive to sheep as in the earlier stages, possibly because of the presence of the large numbers of thorny fruits which it bears.

The plant has always been considered to be an excellent fodder crop by farmers, except during the periods when Geeldikkop makes its appearance. Quin in one of his earlier papers on the subject [10] states that Tribulus cannot be regarded as a poisonous plant in the ordinary sense of the term. It is of a high nutritive value, except when "Geeldikkop" makes its appearance. In a further paper Quin and Rimington [11] state that. "the well-grown plant is recognized as an excellent stock feed and tests have confirmed that it possesses a high nutritive value". In the light of our recent findings these views should be reconsidered. During the course of the recent field investigations into "Geeldikkop" and the heavy outbreak of abortions amongst Angora goats in the Karoo and Cape Midlands, a very large number of normal animals were examined, subjected to numerous diagnostic tests, and various determinations, which will be described in later papers in this series, were performed on blood specimens from them. Many of these animals were also slaughtered for histopathological studies. It was found that nephrosis of varying degrees of severity was common amongst these animals as was also a peculiar type of liver lesion. In its earliest forms this lesion was seen as a large circumscribed area of necrosis lying about midway between the dorsal and ventral lobes of the liver, surrounding the entrance of the portal vein into this organ. The necrotic area was extremely soft in consistence and in advanced cases much of the necrotic tissue had been replaced by fibrous tissue. In a number of extreme cases, the liver tissue in this area had virtually disappeared and the organ appeared to consist of two distinct lobes joined by a thin isthmus of fibrous tissue.

During the course of experiments performed on normal healthy Karoo sheep in which the various saponins and sapogenins extracted from Tribulus were tested for toxicity, certain interesting findings in this respect came to light. It was found that when 20 gms. of crude Tribulus saponins were dosed to sheep in split doses over a three-day period, necrobiotic changes in both the liver and kidneys, highly reminiscent of those described, were produced. Of the two aglycones tested, Ruscogenin proved to be a severely hepatoxic, nephrotoxic and haemolytic, while Diosgenin produced only mild degenerative changes in both the liver and kidneys. These experiments will be reported on in detail in one of the following papers of this series.

As these saponins are present in the young plant in concentrations of up to 2% of the dry weight of the plant and as the plant is eaten most ravenously at this stage, it should be considered as a potential hazard to animal health in spite of its apparent good food value. From the experiments cited it is obvious that either the saponins, their aglycones or degradation products of either must

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be absorbed in order to produce the lesions noted.

The fact that Tribulus is so widespread in the areas where "Geeldikkop" is prevalent is due in no small measure to overstocking and other grazing malpractices. As mentioned earlier it is to be seen growing in abundance in parts of the veld which have been severely tramped out. Ample evidence of the deterioration of the grazing is also provided by the large patches of *Rhigozum trichotomum* ("Driedorings") *Hertia pallens* and *Asclepias* spp. which are to be seen on many of the farms in these areas. In a paper published in 1935, Quin and Rimington [11] made a special note of the rôle overstocking was playing in the increasing incidence of "Geeldikkop" and advocated grazing control measures and the propagation of suitable trees and shrubs for control of the disease. The need for such measures is even more acute today than it was then if the sheep and wool industry of this country is to be saved from disaster.

In its early stages of growth, *Tribulus terrestris* has been responsible for much confusion amongst the farming community as to whether it is a single species or not. When conditions are favourable for rapid growth the small upright plants are overall green in colour, but when subjected to dry hot conditions they become stunted and slow growing and assume a dull reddish brown colour. The leaves of the plant in this state often appear much smaller than those of the normal fast growing plant. This had led to the belief amongst farmers that the green healthy plants are a non-toxic species, while the reddish brown wilted forms are a separate toxic species. As will be seen shortly, the latter plants are certainly more toxic than the former, but it has been established that these different forms are physiological variants of the same plant.

A considerable diversity of opinion exists amongst the farmers in these areas and also among the past workers on the problem as to at what stage of growth and under what conditions Tribulus becomes toxic. Theiler [7] noted that the disease appeared shortly after the first summer rains especially if these were followed by a spell of drought, and also that the disease disappeared after the first frosts had occurred. He noted also that the plants growing on the veld containing much limestone were more toxic than elsewhere and that the disease was more prevalent along rivers and low lying parts of the farms than on the hills and mountains. He concluded that the plant was toxic in its flowering stage. In 1928 Quin[12] cited experiments in which seven out of ten sheep grazing on the brown wilted "dubbeltjie" developed the disease, while ten controls grazing on the green luxuriant plant all remained healthy. However, in a paper appearing in 1929, he described further experiments in which sheep developed the disease while grazing on all growth stages of the plant. Further outbreaks of the disease ceased abruptly after heavy rains had fallen. Subsequently dosing of plant material collected after these rains produced sudden death associated with methaemoglobinaemia. In his subsequent publications [5,10,11,14], he states that only

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the wilted plants are toxic. In one of these [11] the climatic factors leading to wilting of the plant are discussed, and mention is made of the hot dry wind (usually westerly) which often follows light showers of rain and is one of the most important factors in wilting of the plant. The Karoo farmers often refer to a wind of this nature as a "Geeldikkop" wind. Another factor which he believed to contribute towards the wilting of the plant, was the presence of a small insect larva, resembling a common mealie weavil, in the stem of the plant. This infection of the plant coupled with short periods of drought lead to rapid wilting. This may hold good for such infected plants but is by no means an important factor as only a comparatively small percentage of plants on any farm is so affected.

In a later publication[15] he stated that the wilted plants about one to two inches in height were the most toxic. In the same paper he noted that these plants were non-toxic when quite dry. In an earlier paper [5] he made a similar observation, stating that "poisonous Tribulus once removed from their habitat lose their toxicity without delay." These statements are of considerable interest, for Enslin and Wells [16] showed that yields of crude saponins varying from 0.5 to 2 per cent. by weight could be obtained from the dry plants. As we have so far failed to induce the disease with these saponins or their degradation products, and as no other toxic compounds worthy of note in this regard have been extracted from the plant, this may be taken as further evidence that although the plant is connected in some way with the aetiology of the disease, it is by no means the sole agent responsible. Henrici advanced the theory that a zinc deficiency in certain soils was responsible for the appearance of the saponins in the plant, but put forward no satisfactory evidence to support this view [17].

As a result of numerous observations made during field investigations carried out over the last five years the following facts have emerged: (a) the disease occurs chiefly amongst sheep grazing the small upright wilted plants; (b) it makes its appearance within a few days after sufficient rain has fallen to cause rapid growth of the plants, followed by conditions conducive to wilting; (c) the disease appears to be as severe and the incidence as heavy on any type of soil; (d) it is more prevalent on the plains where tribulus grows in abundance than on the hills and ridges where the plant is scarce; (e) the saponins present in the plant may vary qualitatively with its stage of growth or state of health, e.g. the saponin provisionally designated Saponin C, a ruscogenin rhamnoside, was found to be present in greatest amounts during the stage of incipient wilting of the plant and absent from the fully wilted plant.

It is a well known fact, and one also noted by Theiler [7] that when sheep are moved to a different camp the moment symptoms appear amongst them, the disease often disappears dramatically. It is rather difficult to understand how plants on one side of a fence could be toxic, yet non-toxic on the other side when conditions

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throughout both such camps are often quite identical. At this stage of the research work we are still forced to speculate on the reason for this. The explanation would seem to be in one or both of the following: (a) a very short period of toxicity of the plant and extreme lability of the icterogenic agent; (b) a latent period following ingestion of the toxic factor before the typical symptoms of the disease appear. By the time affected sheep show signs of the disease the toxic factors have disappeared from the grazing in the camp to which they are moved. This view seems supported by observations that the disease often appears in successive waves in flocks which remain in camps where it breaks out, with sporadic cases occurring in between each large outbreak. It must be remembered that seeds of the plant germinate throughout the summer months whenever sufficient moisture is available and fresh crops of the plant are continually appearing to become subject to the conditions which render it capable of producing the disease.

The disease probably affects all breeds of goats and sheep and possibly also some of the smaller wild ruminant game species. The finely bred Merino sheep and Angora goats appear to be most susceptible. There is at the moment no valid reason why other breeds of sheep or goats should not develop the disease, but there are on the other hand some good reasons why the disease might pass unnoticed by the farmer or stock attendants in these animals, when many of them might be suffering from it. In order to understand this, the salient features of the disease should be considered, namely: (1) It is characterized by the sudden onset of a severe but transient photosensitization reaction and a more or less severe icterus of somewhat longer duration. The photosensitization lesions are noticed on those unpigmented areas of the body poorly covered with hair or wool and consequently exposed to sunlight, notably the skin of the face and ears, the nostrils, lips and eyelids, the axillae and groins, the volar aspect of the carpus and plantar aspect of the tarsus, the lower extremities of the limbs, coronets, scrotum and mammae. These lesions, being the most dramatic, are the first and usually the only outward signs noticed by the farmer. Should the skin of the animal concerned be pigmented, especially black as in the case of the Black Head Persian sheep or Karakul, the skin will be protected to a marked degree and such cases will probably pass unnoticed by the farmer. Careful examination of cases in the former breed, will usually reveal the lesions on the limbs, and in both breeds the icterus will be seen. The fine skins of the Merino and Angora goat possibly also offer less resistance to penetration of the light rays concerned, than the coarser and thicker skins of the hardier indigenous breeds of sheep and the "Boer" goat.

(2) There appears in most cases of the disease a severe nephrosis or nephritis which is associated with severe electrolyte disturbances and uraemia. At least thirty per cent. of affected animals succumb to these conditions and at the time of death the photo-

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sensitization has often disappeared, the icterus is frequently negligible and there is usually a severe gastro-intestinal stasis. A diagnosis of conditions like Enterotoxaemia, Enzootic Icterus, rumen stasis and other plant poisonings is often made, the primary condition not being recognized, especially if the animal has not developed the photosensitization lesions.

(3) Many cases appear to die up to one month after the appearance of the primary symptoms from severe adrenal hypofunction and electrolyte imbalance, in the absence of any renal lesions. These animals are usually markedly emaciated and dehydrated and apart from obvious changes in the adrenals little else of note is visible on autopsy. The death of such animals is attributed to any number of conditions except "Geeldikkop".

(4) As will be seen in the following paper in this series, the disease does not always follow a typical course. In some cases the icterus may be negligible from the onset of the disease or entirely absent in a few cases. On the other hand the overall picture of the disease in a particular flock may be confused by the presence of cases of Enzootic Icterus as well as the "Geeldikkop". The following case history of an outbreak of these diseases in one particular flock, serves not only to illustrate this point but is very clear evidence of the close relationship between the two diseases. During the course of our field investigations into "Geeldikkop" in the Victoria West area in December of 1958, we were notified that the disease had broken out in a severe form on a certain farm lying roughly twelve miles north of Loxton. On careful inspection of the farm and flock it was found that although Tribulus was present in the camps, it was extremely scarce, and the affected sheep were found to be suffering from and dying in large numbers from the typical acute haemolytic syndrome of Enzootic Icterus. No cases of "Geeldikkop" were apparently present. One week later the farmer concerned notifed us rather indignantly that our diagnosis was incorrect and that his sheep were definitely suffering from "Geeldikkop". A further visit was paid to the farm and it was found that almost all the sheep which had become ill subsequent to the first visit were suffering from the photosensitization syndrome and intrahepatic cholestasis, typical of "Geeldikkop". With the exception of a few lingering cases, Enzootic Icterus had virtually disappeared. The only visible change in the grazing was that the veld had, if anything, become somewhat drier, and the occurrence and appearance of the Tribulus plants did not seem to have altered materially since the initial visit. No movement of the sheep to different camps had taken place and no obvious reasons for the change in the nature of the disease could be determined.

As will be seen in a later paper in this series dealing with the pathology of "Geeldikkop", many of the cases from other farms regarded as being typical of the disease, by virtue of the clinical and chemical pathological findings, show in addition to the usual

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features of "Geeldikkop" histopathology, certain changes which have hitherto been considered as being pathognomonic of Enzootic Icterus. There seems to be little doubt that these two enigmatic diseases are closely related.

"Geeldikkop" occurs in small ruminant animals of all ages and there is also no apparent sex difference in the incidence. According to Quin [12] Quin and Rimington [11] and Theiler [7] the disease occurs particularly in lambs under twelve months old. During our recent investigations the disease was seen in animals of all ages from those just starting to graze to adult animals and aged animals. One case was seen in a very pampered pet hand-reared ewe stated by the owner to be at least fifteen years old. No figures are available, but the general impression gained during the recent investigations was that the majority of cases encountered were in sheep from 9 months to 3 years of age. This is in no way significant as the majority of sheep in the Karoo flocks are of this age.

Animals which recover may develop the disease again later in the same season or in the succeeding years. There appears to be no doubt that the statement often made by farmers, that once a sheep has developed the disease, it will never do so again, is quite erroneous. There is indeed no valid reason why this should be so, seeing that the disease is essentially a poisoning.

Although the disease is encountered in animals of all ages, the fact that it is reported to occur chiefly in lambs of under twelve months old is most interesting. A condition known as "Alveld", which occurs in Norway amongst lambs of the same age group, and essentially similar to "Geeldikkop", but caused by the plant *Narthecium ossifragum* (Lilliaceae) has recently been described by Ender [18]. From our experimental work to be described in a later paper, we have concluded that the icterus of "Geeldikkop" and the icterus of *Lippia* poisoning are due to a biochemical lesion of the enzyme systems responsible for the conjugation and excretion of bile pigments by the liver. This lesion is assumed to be due to an antagonistic or competitive action of the toxic agents involved on the enzyme systems concerned. During recent years a considerable mass of literature has appeared on the enzymes and other intermediate compounds concerned in the conjugation of bilirubin. This work has done much to elucidate the nature of the defects concerned in neo-natal icterus and congenital non-obstructive, non-haemolytic icterus. For instance, Schmid *et al* [19] have recently demonstrated that the congenital jaundice seen in certain strains of Wistar rats is due to a defect in the glucuronyl transferase system. They state that: "Since conjugation of bilirubin appears to be essential for its excretion in the bile, this enzymatic defect in glucuronide formation is believed to be the cause for the impaired pigment excretion and the retention of free bilirubin in the plasma". The same authors, and also Lathe and Walker [20] have suggested that the same conditions occur in human neo-natal jaundice. The latter workers describing in vitro conjugation experiments with fresh liver slices, found that liver

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slices from new-born human infants did not conjugate bilirubin. Dutton and Greig [22], describing similar work, found a glucuronyl transferase to be normally present in the liver of the sheep, and that conjugation activity was absent in the livers of early guinea pig foetuses and new-born mice. They were able to demonstrate that enzyme levels rose considerably in the livers of full term guinea pig foetuses, while the enzyme levels in murine livers reached the adult values in from 3-4 weeks after birth. This interesting work does not appear to have been pursued further in sheep, but it seems reasonable to assume that the glucuronyl transferase levels in the liver slowly builds up in the same manner, until the adult values are attained. At the moment then it seems reasonable to assume that lambs are more susceptible to the action of these icterogenic agents than are adult sheep by virtue of imperfectly developed conjugation systems, which are more completely overwhelmed by the icterogenic aglycones than the conjugation mechanisms in older sheep.

"Geeldikkop" as induced by Tribulus does not seem to occur to any extent in domestic animals other than the small ruminant. Quin[15] mentions that it is seen occasionally in cattle. During the recent investigations into the disease a few reports were received from farmers to the effect that fowls eating Tribulus growing around homesteads at the time of "Geeldikkop" outbreaks developed icterus, and necrosis of the comb followed by sloughing of this structure. As none of these birds were seen by us, thes ereports could not be verified.

In conclusion, it is not out of place to mention briefly some of the economic aspects of this disease. The annual havoc wrought by "Geeldikkop" in the sheep and wool industry of this country does not appear to have ever been properly assessed. There is no doubt that the losses caused by it every year in the areas where it occurs are quite devastating and during the last three years many of the smaller farmers have been brought to the verge of bankruptcy following the continual decimation of their flocks. No exact figures have been compiled as to the incidence of the disease in individual flocks, but general impressions gained place this on the average of from 10 to 30 per cent. of each flock affected. The mortality rate in affected flocks shows considerable variation according to the severity of the particular symptom complex seen, the care and treatment accorded sick animals, the nutritional state of the flock, and the presence of other debilitating conditions like verminosis. Up to 80 per cent. of the affected animals may succumb on certain farms, while on others the majority of the animals may recover, depending on the care they have received.

Not only is the mortality rate on the average high enough to constitute a serious financial loss to the farmer and the sheep and wool industry, but the subsequent loss of condition, production and impairment of reproductive ability seen in recovered animals is a severe annual drain on the economy of the industry. Recovered sheep invariably develop a "break" in their wool and indeed often shed

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their entire fleece. Many take months to improve in condition again and during this period of convalescence show a marked lowering in fecundity. Sporadic deaths are encountered up to at least a month after the disease has disappeared from the flock, due to nephritis and adrenal dysfunction.

During the course of investigations into outbreaks of the disease in the Hofmeyr district in January 1959, nineteen farmers were requested to complete a small questionnaire in this regard. At this time the disease had only been present in the district for about three weeks and according to reports received continued its ravages for at least a further three weeks. The farms and flocks in this area are in general smaller than in the Britstown, Vosburg and Victoria West areas, and thus the losses were felt more acutely than in the latter districts. The following table indicates the approximate losses incurred by those nineteen farmers within about three weeks:

Total of sheep that were sick ..... 1860 (Approx. value £5,570).
Total of sheep that had died ..... 935 (Approx. value £2,800).
Mortality rate .... Approximately 33%.

The approximate values given are, if anything, on the modest side and do not take into consideration losses which will be incurred after recovery such as lowered production. Some of these flocks were inspected by us and it was quite apparent that at least another third of the cases still showing symptoms of the disease would terminate fatally. The losses amongst stud breeders are of an even higher order. On one farm in the Loxton area four extremely valuable rams were seen in a serious condition. The worst of these was one valued at five hundred pounds.

Summary.

Some of the more important features of the epizootology of "Geeldikkop" are presented. Its seasonal occurrence and geographical distribution are discussed. Some information concerning the plant believed to cause the disease is given and other plants suspected by farmers of being responsible are considered. The toxicity of Tribulus under various conditions is discussed. The incidence of the disease in small ruminant animals in respect to breed, sex and age is reviewed and some thoughts advanced as to why the disease is usually seen chiefly amongst young animals and the more finer breeds of sheep and goats. The close relationship between "Geeldikkop" and Enzootic Icterus is noted and some indication of the economic importance of the disease is given.

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Secretary of the Teviot branch of the National Wool Growers' Association, will long be remembered.

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THE CONTROL OF THE ITCH MITE OF SHEEP (*Psorergates Ovis*) WITH DIPS CONTAINING DELTA BHC.

R. DU TOIT AND O. G. H. FIEDLER.

(*Received for publication, 3rd November,* 1959.)

The finding by the authors[1] that the itch mite of Merino sheep (*Psorergates ovis* Womersley) possesses a pattern of insecticidal susceptibility entirely different from that of other ectoparasites such as scab mites, lice, keds, ticks and blowflies, appears to be of considerable interest. Whereas these latter species respond to control by the gamma isomer of BHC, the itch mite, however, shows an extremely high degree of natural tolerance to this otherwise potent isomer but is very susceptible to the delta and epsilon isomers. In addition a distinct antagonistic action between the gamma and delta isomers upon *Psorergates* was noted whereby the effectiveness of the delta isomer is reduced in the presence of the gamma isomer. This phenomenon offers an explanation for the inefficiency of ordinary BHC dips against this mite. The gamma-delta ratio of technical BHC is normally 2 to 1, i.e. an insufficient amount of the delta isomer is present in the dipwash at the usual effective gamme concentration; the action of the delta isomer is further decreased by the antagonistic effect of the comparatively large quantity of gamma upon it.

It was the aim of the experiments described in this paper to establish a suitable ratio, or what might be termed the optimum proportions, of these two isomers to produce a dipping preparation which would prove effective both for the itch mite as well as for lice, keds, and ticks.

As a first step the efficacy of the delta isomer upon the itch mite had to be determined by experiments *in vitro*. Serial dilutions of the pure delta isomer in liquid paraffin gave the following mean periods for complete immobilisation of itch mites immersed in such solutions:

delta concentration	immobile after
5,000 ppm.	10½ minuutes
2,500 ppm.	17 minutes
1,250 ppm.	55 minutes
625 ppm.	3¾ hours
313 ppm.	over 6 hours

The interaction of the two competing isomers was tested thereafter

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by adding equal parts of the gamma isomer to the delta solutions. It was noted that the periodss for immobilisation were prolonged.

delta and gamma conc.	immobile after
5,000 ppm. of each	16½ minutes
2,500 ppm. of each	29½ minutes
1,250 ppm. of each	3 hours
625 ppm. of each	over 6 hours

In a second series the delta concentration was kept constant at 1,250 ppm. and increasing quantities of the gamma isomer were added to it. The results showed that the addition of even a low percentage of the gamma isomer produced a pronounced antagonistic effect, which effect had to be considered when the initial delta concentration was fixed for the first dipping trials.

	Composition	delta-gamma ratio	immobile after
1.	1,250 ppm. delta	—	55 minutes
2.	1,250 ppm. + 125 ppm. gamma	10:1	97 minutes
3.	1,250 ppm. + 625 ppm. gamma	2 :1	173 minutes
4.	1,250 ppm. + 1,250 ppm. gamma	1 :1	186 minutes

As the resulting figures governing the period up to the onset of immobility follow a hyperbolic curve, it becomes possible to interpret the action of intermediate delta-gamma ratios not included in this series. In previous experiments it had been determined that those concentrations of a killing agent dissolved in liquid paraffin, which resulted in total immobilisation of the mites within six hours, proved to be effective in practice, whereas those compounds or concentrations of effective compounds not killing or affecting the mites within this period, also failed to control the parasites *in vivo*. It can be deduced therefore, from the tests given above that the pure delta isomer possesses a killing potential well below the concentration of 600 ppm., and a mixture of the gamma and delta isomers of equal parts has its lower limit at a concentration of 1,000 ppm. delta. It must be borne in mind, however, that certain solvents and emulsifiers might increase the effectiveness of an emulsion and, secondly, that the active ingredient is removed selectively from an emulsion dip with the result that this selective removal produces a higher concentration of the active ingredient by accumulation in the greasy film on the skin and around the wool fibres. All the ectoparasites of woolled sheep thus come into contact with the compound at a concentration considerably higher than that present in the dipping tank. This should also hold true for the delta BHC and it can be expected, therefore, that concentrations far below 1,000 ppm. would still be effective *in vivo*, particularly when the gamma content of the preparation is low.

Preliminary dipping trials with several experimental preparations in the form of emulsions or wettable powders proved that this assumption was correct. All formulations tested were effective and

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resulted in a complete kill of the mites below a concentration of 1,000 ppm. delta.

The test preparations used consisted of two emulsions with a delta-gamma ratio of 1 to 1 and 3 to 1, respectively, and a wettable powder belonging to the last category. The dipping experiments were conducted as supervised field trials at Onderstepoort, on different farms in the Transvaal and in the Karoo Midlands of the Cape Province.

The following experimental procedures were observed. If possible, flocks were inspected prior to shearing and well infested animals, showing typical clinical symptoms and the presence of the parasite in skin scrapings, were selected and ear-tagged. (The same method was followed when sheep with longer wool had to be treated.) If an inspection prior to shearing was not possible, test animals for earmarking had to be selected by examination of skin scrapings only, immediately before dipping. The dipping tanks, with capacities of between 600 and 1,000 gallons were filled with an initial delta strength of 1,000 ppm. Samples of the dip wash were taken for analysis prior to the commencement of dipping and again after every 50 or 100 sheep. The ear-tagged animals were sent through the bath singly or in groups of two or three at the time when the dipwash was sampled. No replenishment of insecticide or water was made during these preliminary trials and dipping was carried on as far as possible until about three-quarters of the dip-wash had been removed. This experimental procedure permitted a direct correlation to be made between the insecticidal strength and its biological efficiency. Results were read after several months and for the last time prior to the next shearing. If the treatment was not successful the remaining infestation could always be detected by the persistence of the clinical symptoms and by finding the mites in scrapings. It was found during the course of the trials, that dipping results can be ascertained in infected ear-tagged sheep about three months after treatment, and any focus of mite infestation surviving the application will show up distinctly in the flock until the following shearing.

The preliminary field trials without replenishment of the dip-wash during dipping gave the following results:

1. The delta wettable powder with a delta-gamma ratio of 3 to 1 ensured eradication of the itch mite with a single dipping at a delta concentration well below 500 ppm., provided that the sheep were recently shorn. The preparation failed entirely, however, at considerably higher concentrations when the wool was longer (over three months). Control of keds and lice was always good. The wettable powder was excluded from further tests on account of its presumed poorer penetration ability through the wool.

2. The BHC emulsion with a delta-gamma ratio of 1 to 1 gave good results down to about 400 ppm. delta. Eradication of keds, lice and scab mites was still achieved with a concentration of about

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TABLE I.—DIPPING TRIAL WITH A DELTA BHC EMULSION (RATIO OF THE DELTA-GAMMA ISOMERS 4:1) AND MERINO SHEEP CARRYING FIVE MONTHS' WOOL.

Number of Sheep	Dipwash in tank		Delta concentration in ppm.		Gram delta in tank	Gram delta removed per 50 sheep	Gram delta removed per sheep	Percentage exhaustion rate	Gram delta per gallon in tank	Gram delta removed per gallon	Selective removal
	Galls.	Litres	Analysed	Computed							
0	1,500	6,805	992	6,755	——	——	——	4.5	——	——	——
50	1,388	6,290	957	957	6,010	745	14.9	11.0	4.3	6.6	1.54
100	1,275	5,780	926	921	5,330	680	13.6	11.3	4.2	6.0	1.35
150	1,163	5,275	860	875	4,260	710	14.2	13.3	4.0	6.6	1.57
200	1,050	4,760	794	824	3,925	695	13.9	15.0	3.7	6.2	1.68
250	938	4,255	774	764	3,250	675	13.5	17.2	3.5	6.0	1.71
300	825	3,745	697	692	2,590	660	13.2	20.3	3.1	5.9	1.90
350	713	3,235	631	614	1,985	605	12.1	23.4	2.8	5.4	1.93
400	600	2,720	524	535	1,455	530	10.6	26.8	2.4	4.7	1.96
450	488	2,215	463	460	1,020	425	8.7	29.9	2.1	3.9	1.85
500	375	1,700	402	390	663	357	7.1	35.0	1.8	3.2	1.78
550	263	1,190	351	325	387	267	5.5	41.6	1.5	2.4	1.60

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100 ppm. gamma. It was felt, however, that this ratio of isomers was not suitable in practice as the initial gamma concentration of 1,000 or 8,000 ppm. was far too high to be safe. Toxic symptoms of BHC poisoning were observed in a number of animals in poor condition.

3. An emulsion with a delta-gamma ratio of 3 to 1 represented a definite improvement upon the former two formulations. No toxic symptoms were observed at the highest concentration applied, viz. 1,000 ppm. delta and 333 ppm. gamma, even in young lambs. Complete kill of itch mites still occurred at a concentration of about 250 ppm. delta in short woolled sheep, and at the same time lice and keds were controlled successfully by the equivalent gamme strength.

It was concluded from this test series that the optimum proportion of the two important isomers in an all-purpose sheep dip is a ratio of about four parts of delta to one part of gamma. Such a combination would eliminate any possible toxic hazards, the antagonistic influence of the gamma isomer would be further reduced and finally, the dipping material would be more economical, if the initial delta concentration could be decreased. An improved BHC formulation of this nature was obtained and all further experimentation was carried out with this particular emulsion.

Since previous dipping tests with medium and long woolled sheep gave doubtful results with the BHC formulations tested when the delta concentration was allowed to fall below a certain limit (at the time undetermined) dipping trials with individual well infested animals, carrying wool about 2 to 2½ inches long were conducted. The new BHC preparation with a 4:1 delta-gamma ratio* was used to determine the lowest effective concentration of the delta isomers still capable of eliminating itch mites with a single treatment. The tests revealed that with a single treatment eradication was not achieved at a concentration of 400 ppm. delta. Larvae and nymphs of the mites were found in the scrapings about six weeks after the treatment. The same low delta strength, however, was successful when a second dip was supplied two to four weeks later.

As the aim was to achieve complete control of the itch mite as well as of all the other parasites, it was decided that the concentration of the delta isomer should never drop below 600 ppm. in future trials and that the initial concentration would be fixed at 800 ppm. The gamma concentration would then remain between 200 and 150 ppm., which is known to assure a complete kill of all gamma-susceptible ectoparasites. The replenishing necessary to maintain this level was established in two separate trials before the experiments designed to determine the efficacy of the dip were commenced. In accordance with the selective removal of the active ingredient, the replenishing rate had to be 1¾ times higher than the initial strength of the bath, and replenishment became due when about one-third of

*This preparation, with the factory symbol CX99, was supplied by Cela, Ingelheim, Germany.

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the dipwash was removed. To demonstrate the procedure an example of such a dipping trial is given in Table I, where 550 sheep carrying five months' wool were immersed for only about 20 seconds each. The average rate of selective removal until half of the dipwash was carried out, was 1.65 times higher than the dipping concentration.

Two large-scale field tests were conducted before the preparation was released for user trials. Flocks comprising 2,400 and 2,250 sheep were dipped in a tank with a capacity of 800 gallons. The first flock consisted of recently shorn Merinos (two to four weeks after shearing), whereas the second flock carried wool of $2\frac{1}{2}$ to 3 months. The itch mite infestation as judged by the appearance of the fleeces was between 5 and 15 percent., respectively. To ensure that infection was present, however, well-infested animals were ear-tagged and sent through the bath when replenishment was due. Chemical analysis of dip samples taken before and after replenishing showed that the insecticidal concentration remained between the desired limits.

The flocks were inspected 3 and 10 months after dipping and again 15 months later and scrapings were taken from the marked animals. Both treatments were a complete success. No signs of mites nor any clinical symptoms were found during any of these inspections. The skin of the heavily infested animals had regained its normal colour and consistency, none of the 4.650 sheep showed the slightest disturbance of the fleece. Thereupon, a number of user trials were conducted in different parts of the Union, comprising between 25,000 to 30,000 sheep. The treatment was applied by the farmers themselves in each case and the flocks were inspected after 6 to 12 months by one of the authors. The results appeared to be entirely satisfactory. Until September 1959, about 2,000,000 Merino sheep were dipped against infectious itch in this particular BHC preparation and only eight reports have been received so far where the owners claimed the eradication of the mites was not achieved. All these cases were followed up; three farmers were found not to have observed the recommended rate for replenishment and, therefore, had dipped a number of their sheep in a concentration hopelessly below strength. Three other users had mistaken lumpy wool (mycotic dermatitis), that had developed about three months after treatment, for a recurrence of the mite infestation and only in the remaining two cases could no obvious reason for the failure of the dip be found.

Conclusions:

The trials conducted by the authors in the Union of South Africa have demonstrated that a BHC emulsion with a ratio of the delta to gamma isomers of 4 to 1 constitutes a suitable means of exerting adequate control of all ectoparasites of sheep with the exception of blowflies and BHC-resistant ticks. It was shown furthermore that such a preparation is capable of eradicating the itch

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mite with a single treatment provided it is applied at the correct strength. Finally, it must be stressed that for best results only recently shorn sheep should be treated.

This last remark applies not only to achieving complete cure of the itch mite infestation, but this procedure should be observed in all cases as the fine wool of the South African Merino tends to suffer and to decline in quality when brought into intimate contact with the water of a dipping bath alone and especially so in water in which dirt and foreign matter tend to accumulate.

Summary.

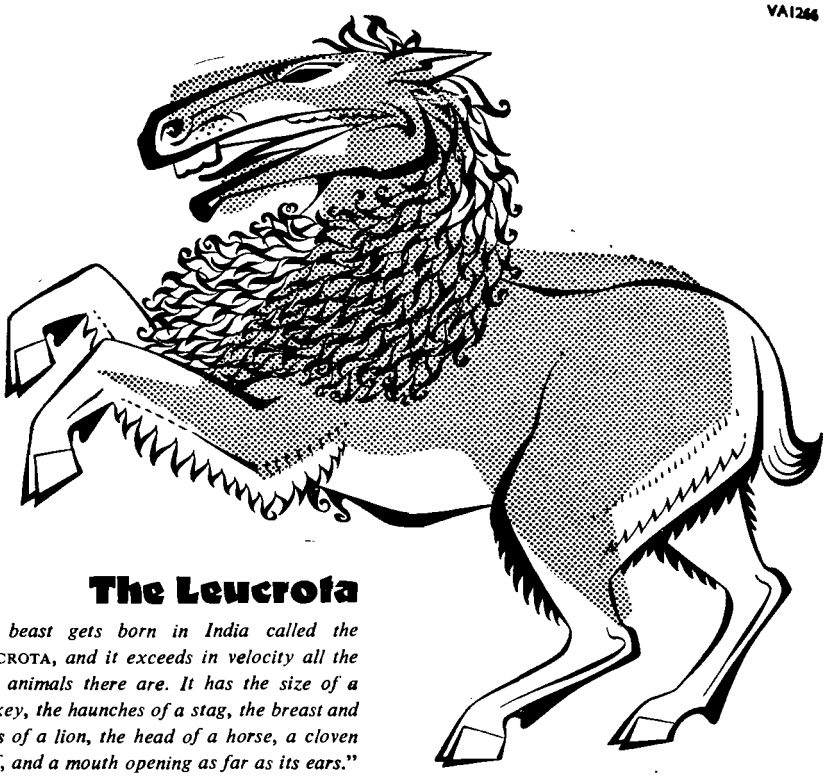
Experiments are described wherein *Psorergates ovis* mites were subjected to serial dilutions of the delta isomer of BHC and also to varying combinations of the delta and gamma isomers in a bland mineral oil in order to demonstrate the pattern of insecticidal susceptibility possessed by this mite species as also the antagonistic effect of the gamma isomer upon the delta. From the findings it is concluded that a ratio of 4 parts of the delta to 1 part of the gamma isomer represents a combination for dipping purposes well suited to eradicate *Psorergates* mites as well as gamma susceptible ectoparasites such as scab mites, keds and lice. Based upon these findings a dip containing the delta and gamma isomers of BHC in the ratio of 4:1 was tested under field conditions and it was shown that a single dipping would eradicate infectious itch provided the wool at the time of dipping is short, not over ½ an inch, and the concentration of the delta isomer is not allowed to drop below 600 ppm.

A table is included indicating the extent to which the delta isomer is removed selectively during a typical dipping trial on sheep carrying 5 months' wool.

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ON THE DIFFERENTIAL DIAGNOSIS OF THE THIRD STAGE (INFECTIVE) LARVAE OF *Chabertia ovina* and OESOPHAGOSTOMUM spp. (*O. columbianum and O. venulosum*) WITH SPECIAL EMPHASIS ON THE CHARACTERISTIC LENGTHS OF *C. ovina.*

G. L. Muller*

During the course of routine examinations of numerous larval cultures, some difficulty was experienced in differentiating between the infective third stage larvae of *Chabertia ovina* and Oesophagostomum spp. (*O. columbianum* and *O. venulosum*), using the low power magnifications which are usually used for such routine examinations. With the use of higher magnification, involving of necessity time and care, significant differences are apparent between the third stage larvae of these three species, and these differences are described[1-5] although Morgan[6] states that he could not differentiate between the third stage larvae of *C. ovina* and *O. venulosum.* However, these methods of differentiation are slow, tedious and time consuming, especially when large numbers of larval cultures have to be examined.

It is felt that a comparison of measurements of certain characteristics of the larvae would simplify the differentiation of these species of larvae. The significant characteristic measurements quoted are:-

a) Total length of the larva from the oral extremity to the tip of the tail sheath.
b) Length from anus to tip of tail sheath.
c) Length from anus to tip of larval tail.
d) Length from tip of larval tail to tip of sheath.
e) Length of oesophagus.
f) Length of intestinal cell group.
g) Number of intestinal cells.

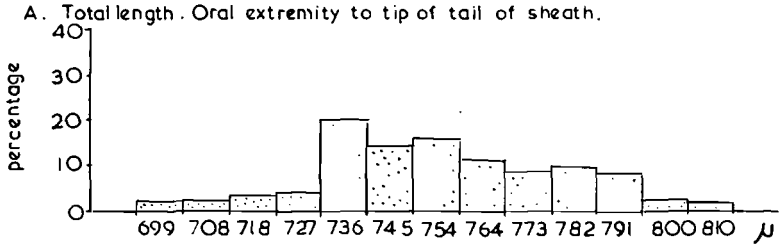
A wide divergence of measurements was noted for the various characteristic lengths of the third stage larvae of *C. ovina* by the various authors, whereas the measurements quoted for *O. columbianum* and *O. venulosum* are all more or less within the same range groups, and these figures are quoted, although a considerable number of *O. columbianum* larvae were checked for the purposes of confirmation. To correlate the measurements of *C. ovina,* 210 larvae were studied and examined. These *C. ovina* larvae were obtained by incubating eggs, harvested from gravid *C. ovina* females, for 7 days at 26°C.

* State Veterinarian, Mossel Bay.

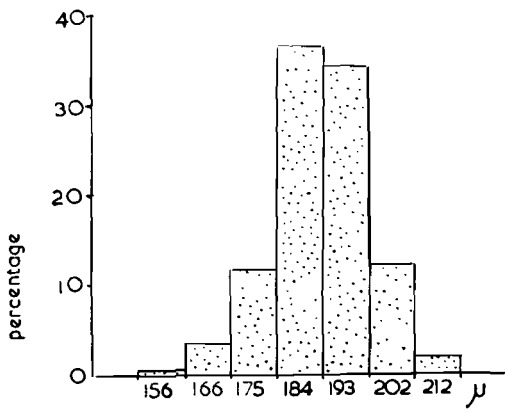
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CHABERTIA OVINA ESSENTIAL MEASUREMENTS IN MICRONS μ.
THIRD STAGE (INFECTIVE) LARVAE (210 LARVAE)

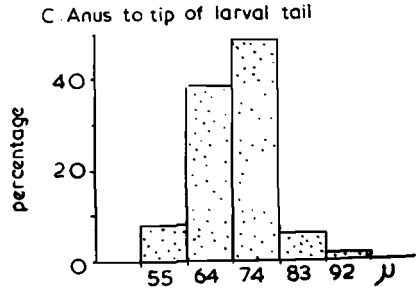
A. Total length. Oral extremity to tip of tail of sheath.



B: Length from anus to tip of sheath

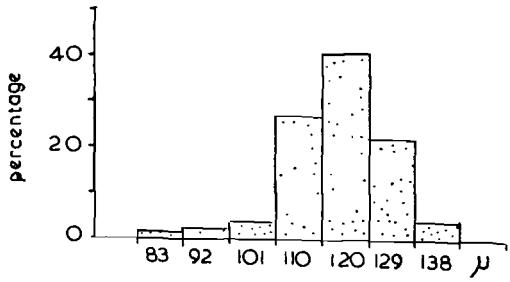


C. Anus to tip of larval tail

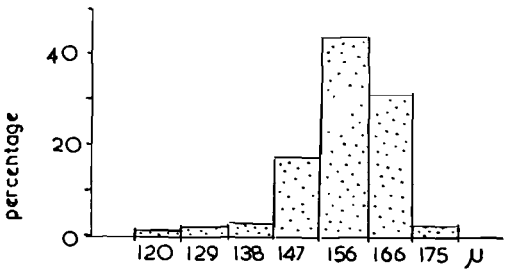


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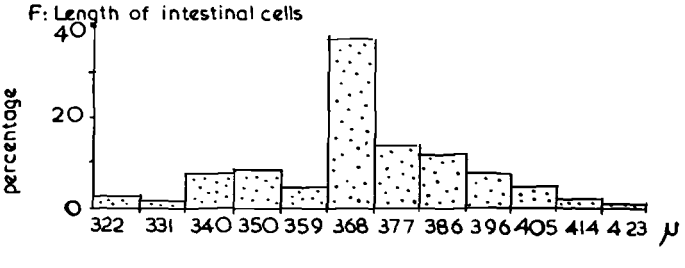
D. Tip of larval tail to tip of sheath.



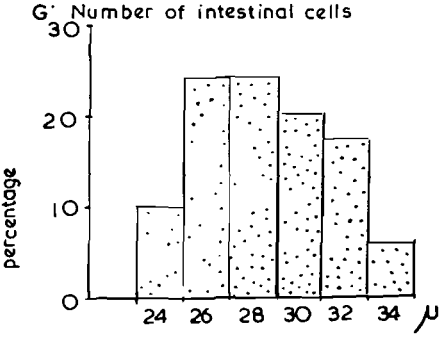
E: Length of oesophagus.



F: Length of intestinal cells



G: Number of intestinal cells



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General

The average total length of the larvae of both species of *Oesophagostomum* is greater than that of *C. ovina* although there is an overlap at the extremities. The larvae of *C. ovina* were found to range from 699 microns (μ) to 810 microns, with the majority (87.6%) within the limits 736μ to 791μ, while for those of *O. columbianum* a range of 771μ to 923μ[2] and 791-849μ[3] is given. Measurements made by the writer of third stage *O. columbianum* larvae favour the former measurements. The total length range for third stage *O. venulosum* larvae is given by Goldberg[4] as an average of 899μ while Wetzel and Marholdt[5] give a range of 760-1140μ with an average of 941μ.

The range of measurements of the characteristics of the larvae of *C. ovina* are shown in the accompanying histograms, whilst the tables below set out the comparative measurements of *C. ovina, O. columbianum* and *O. venulosum*. All measurements are given in microns (μ).

Discussion

From the measurements obtained from the various authors it is obvious that there is a considerable variation in the range of the characteristic measurements of the three species with overlapping at the extremes. For the purpose of rapid identification at low power routine examinations, the author has found it practicable to differentiate the Chabertia and Oesophagostomum spp. simply on:-

Total Length: All larvae with the special characteristics of *C. ovina, O. columbianum* and *O. venulosum* with a measurement of under 791μ, can be classified as *C. ovina*, while those above 791μ should be regarded as either one or other of the two spp. of *Oesophagostomum*. Should doubt arise when a larvae is observed at the extreme end of length range, then the following characteristics are useful in making a positive diagnosis:-

(a) Length from Anus to Tip of Sheath: Those larvae below 202μ classified as *C. ovina* and those with a measurement of over 202μ as *Oesophagostomum* spp.

(b) Length from Anus to Tip of Larval Tail: Those below 74μ are *C. ovina*, and above, *Oesophagostomum* spp.

(c) Length from Tip of Tail to Tip of Sheath: Below 129μ *C. ovina*, and above, *Oesophagostomum spp.*

(d) Oesophageal Length: The average oesophageal length including the range of the majority precludes differentiation of the three species.

(e) Intestinal Cell Group Length: Those larvae measuring over 350μ should be considered as belonging to *C. ovina*, while under 350μ may be regarded as *Oesophagostomum spp.*

(f) Number of Intestinal Cells: Here the number of intestinal cells helps differentiation insofar as *O. columbianum* has

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TABLE 1. TOTAL LENGTH: ORAL EXTREMITY TO TIP OF SHEATH.

Author	*C. ovina* A.	*C. ovina* B.	*O. columbianum* A.	*O. columbianum* B.	*O. venulosum* A.	*O. venulosum* B.
Mönnig	——	772 aver.	791-849	830 aver.	——	——
Dikmans and Andrews	710-789	747 aver.	771-923	840 aver.	——	——
Muller	699-810	736-791	800-958	856-920	——	——
Goldberg	——	——	——	——	——	899 aver.
Wetzel and Marholdt	724-889	786 aver.	——	——	760-1140	941 aver.

TABLE 2. LENGTH FROM ANUS TO TIP OF SHEATH.

Author	*C. ovina* A.	*C. ovina* B.	*O. columbianum* A.	*O. columbianum* B.	*O. venulosum* A.	*O. venulosum* B.
Mönnig	——	172 aver.	204-235	223 aver.	——	——
Dikmans and Andrews	175-220	192 aver.	193-235	213 aver.	——	——
Muller	156-212	175-202	202-248	212-239	——	——
Goldberg	——	——	——	——	——	242 aver.
Wetzel and Marholdt	——	208 aver.	——	——	——	241 aver.

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TABLE 3. LENGTH FROM ANUS TO TIP OF LARVAL TAIL.

Author	*C. ovina* A.	*C. ovina* B.	*O. columbianum* A.	*O. columbianum* B.	*O. venulosum* A.	*O. venulosum* B.
Mönnig	——	67 aver.	——	74 aver.	——	——
Dikmans and Andrews	60-70	65 aver.	60-80	71 aver.	——	——
Muller	55-92	64-74	64-83	74-83	——	——
Goldberg	——	——	——	——	——	70 aver.
Wetzel and Marholdt	——	69 aver.	——	——	——	76 aver.

TABLE 4. LENGTH FROM TIP OF LARVAL TAIL TO TIP OF SHEATH.

Author	*C. ovina* A.	*C. ovina* B.	*O. columbianum* A.	*O. columbianum* B.	*O. venulosum* A.	*O. venulosum* B.
Mönnig	——	105 aver.	——	149 aver.	——	——
Dikmans and Andrews	——	127 aver.	——	142 aver.	——	——
Muller	83-138	110-129	129-174	138-156	——	——
Goldberg	——	——	——	——	——	172 aver.
Wetzel and Marholdt	——	139 aver.	——	——	——	165 aver.

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TABLE 5. LENGTH OF OESOPHAGUS.

Author	*C. ovina* A.	*C. ovina* B.	*O. columbianum* A.	*O. columbianum* B.	*O. venulosum* A.	*O. venulosum* B.
Mönnig	——	172 aver.	157-172	165	——	——
Dikmans and Andrews	160-178	166 aver.	150-170	159	——	——
Muller	120-175	147-166	129-184	138-166	——	——
Goldberg	——	——	——	——	——	168 aver.
Wetzel and Marholdt	——	173	——	——	——	171 aver.

TABLE 6. LENGTH OF INTESTINAL CELL GROUP.

Author	*C. ovina* A.	*C. ovina* B.	*O. columbianum* A.	*O. columbianum* B.	*O. venulosum* A.	*O. venulosum* B.
Mönnig	——	353 aver.	337-360	345	——	——
Dikmans and Andrews	——	——	——	——	——	——
Muller	322-423	340-396	331-460	405-414	——	——
Goldberg	——	——	——	——	——	——
Wetzel and Marholdt	——	——	——	——	——	——

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TABLE 7. NUMBER OF INTESTINAL CELLS.

Author	*C. ovina* A.	*C. ovina* B.	*O. columbianum* A.	*O. columbianum* B.	*O. venulosum* A.	*O. venulosum* B.
Mönnig	——	——	——	——	——	——
Dikmans and Andrews	24-32	——	16-24	——	——	——
Muller	24-34	26-32	20-26	22-24	——	——
Goldberg	——	——	——	——	30-34	32
Wetzel and Marholdt	——	32	——	——	——	——

KEY: A = Total Range. B = Range of Majority.

TABLE 8. COMPARATIVE TABLE OF AVERAGE MEASUREMENTS BY DIFFERENT AUTHORS.

Larval Character in μ	*Chabertia ovina* Mönnig	*Chabertia ovina* Dikmans and Andrews	*Chabertia ovina* Wetzel and Marholdt	*Chabertia ovina* Muller	*Oesophagostomum columbianum* Mönnig	*Oesophagostomum columbianum* Dikmans and Andrews	*Oesophagostomum columbianum* Muller	*O. venulosum* Goldberg	*O. venulosum* Wetzel and Marholdt
Total Length	772	747	786	736-791	830	840	856-920	899	941
Anus to S.t.	172	192	208	175-202	223	213	212-239	242	241
Anus to L.T.t.	67	65	69	64-74	74	71	74-83	70	76
L.T.t. to S.t.	105	127	139	110-129	149	142	138-156	172	165
Oesophagus	172	166	173	147-166	165	159	138-166	168	171
Genital Cell Group Length	353	—	—	340-396	345	—	405-414	—	—
No. of Cells in Genital Cell Group	—	24–32	32	26-32	—	16–24	20-26	32	32

KEY: S.t. = Sheath tip. L.T.t. = Larval Tail tip.

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an average range of 16-24 triangularly shaped cells, and *O. venulosum* 32 pentagonal cells, while *C. ovina* is characterized by 26-32 rectangular, ill-defined cells.

Summary

1) The infective third stage larvae of *Chabertia ovina, Oesophagostomum columbianum* and *Oesophagostomum venulosum* may be differentiated and fairly accurately classified from a consideration of:-

(a) Total length. (b) Length from Anus to tip of Sheath. (c) Tip of Larval Tail to Tip of Sheath. (d) Number and shape of intestinal cells especially between *C. ovina* and *O. columbianum*. For routine examination TOTAL Length is the main criterion for identification.

2) This differentiation may be carried out under ordinary low power magnification of the microscope, thus saving time and tedium, especially when the examination of a large number of routine cultures is involved.

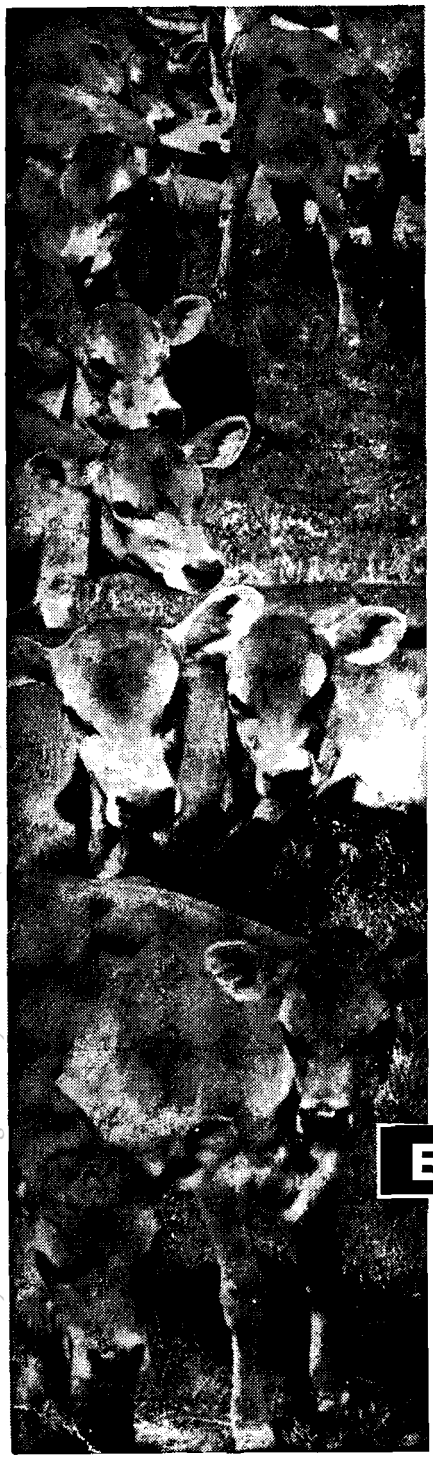
Acknowledgement

The Director of Veterinary Services is thanked for the facilities made available to carry out these investigations, and for kind permission to publish this article.

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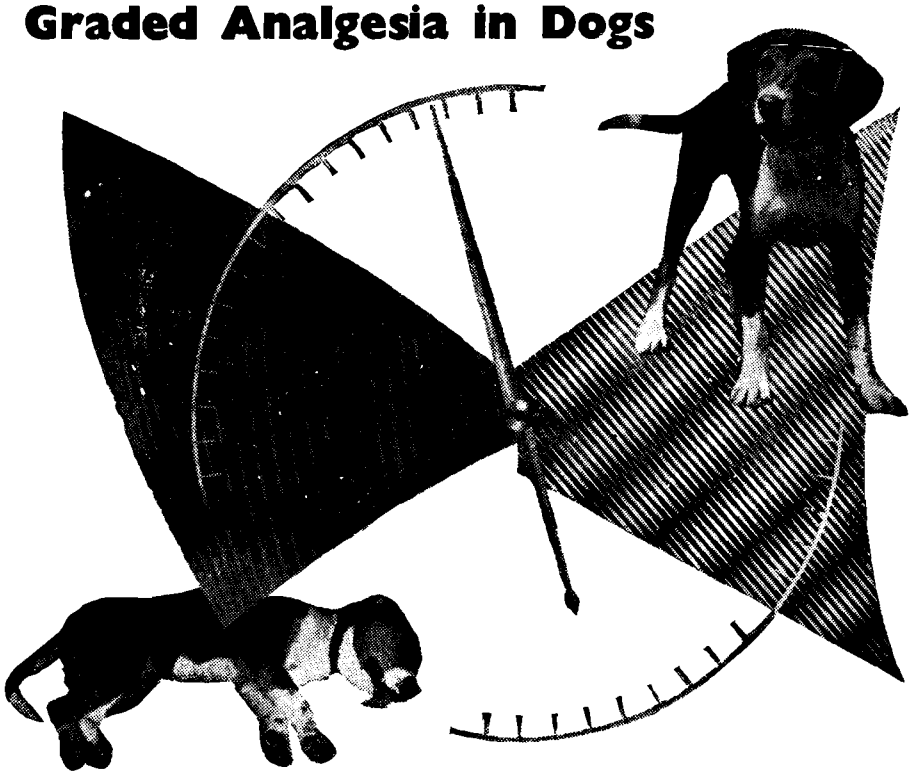
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A NOTE ON THE COMPARATIVE EFFICACY OF PHENOTHIAZINE OF DIFFERENT PARTICLE SIZE UNDER SOUTH AFRICAN CONDITIONS

by

P. W. Thorold, C. J. Bosman and J. A. F. Baker*

(*Received for publication, 3rd September,* 1959)

Work in Australia, Britain and the United States of America with fine particle phenothiazine has indicated that it is more effective than coarser material against certain nematodes, i.e. *Trichostrongylus* spp., *Cooperia* spp., *Haemonchus contortus, Ostertagia circumcincta,* and *Nematodirus spathiger*[2,1,8,4]. It was shown[3] that *Oesophagostomum columbianum* may be less affected by fine particle size (less than 10 mu.) than the trichostrongyles, but that for a high degree of efficiency against *Oesophagostomum* a large proportion of particles should be less than 20 mu. Later it was reported[6] that finer particles (under 5 mu) were more efficient against trichostrongyles of the abomasum and small intestine, but that larger particles (5-20 mu) were more efficient against the species *Oesophagostomum venulosum* and *Chabertia ovina* in the large intestine.

As no information was available of results to be expected when treating South African stock, certain preliminary trials were undertaken. In these, three liquid phenothiazines (permanent suspensions) were used. Prior to making up the liquid products the phenothiazine powders were subjected to particle size determination using the Sharples Micromerograph with the results given below:-

PARTICLE SIZE DISTRIBUTION

Percentage by Weight less than

	2	5	10	20	30	40	50	70	Microns
Material X	0	6	20	64	82	90	95	100	
Material Y	0	22	71	100					
Material Z	3	42	78	(All under 17 microns)					

Materials "Y" and "Z" were from the same source of technical phenothiazine.

Dosing Rate: In Experiments I and II the sheep (70-90 lbs.) were dosed with 2-fluid ounces of liquid phenothiazine products containing 10-grammes phenothiazine per fluid ounce, that is, each sheep received 20-grammes of phenothiazine. The sheep were Merino type native-owned sheep on over-grazed, sour veld —

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there was a marked improvement in condition following treatment.

Experiment I.

Groups of twenty adult sheep were used in this trial. Egg counts were made weekly for three weeks before and two weeks after dosing, according to the method of Gordon[3] and Whitlock[7,5], and faeces cultured as described by Whitlock. These sheep were infested with *Haemonchus, Oesophagostomum, Bunostomum, Trichostrongylus* and *Strongyloides.*

TABLE 1.

	Material X	Material Y	Material Z	Controls
Mean e.p.g. before dosing ...	3,833	4,133	3,299	3,199
Mean e.p.g. two weeks after dosing	822	322	110	3,107
% Reduction e.p.g.	78.6%	92.0%	96.7%	—

Experiment 2.

Four groups of 25 sheep, further divided into sub-groups of 5, known to be carrying a good infestation of nodular worms, were selected for dosing with each of the three phenothiazine suspensions. Faecal egg counts and larval cultures were undertaken on combined specimens from each sub-group of 5. Moreover, one sheep from each group was followed individually and that from group "Z" was sacrificed for post mortem examination. The contents of the abomasum, small and large intestines, were each diluted in one litre of water and then examined in 100 ml. aliquots against a dark background for nematodes, with negative results. Detailed data are presented in tables 2, 3 and 4.

RESULTS:

The data from these two trials, comparing three phenothiazines differing in particle size, show that fine particle size phenothiazine (material "Z" — all under 17 mu) is not only just as efficient as the coarser grades against a good infestation of *Haemonchus* and light loads of *Bunostomum* and *Ostertagia*, but considerably more effective against *Trichostrongylus* than the coarsest material. Against *Oesophagostomum* the finest material was extremely effective.

Experiment 3.

The same three phenothiazine materials were dosed to 4-6 months old calves at 1-fluid ounce/100-lbs., with the following reduction in the faecal egg counts:-

Material "X"—61%; Material "Y"—72%; Material "Z"—87%.

The species encountered in these calves were:-

Bunostomum phlebotomum, Cooperia spp., *Haemonchus contortus* and *Oesophagostomum radiatum.*

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TABLE 2. DATA — 25 SHEEP EACH DOSED 2-oz. MATERIAL "X"

Date	EGG COUNTS *E.P.G. Strongyloides*	*E.P.G. Others*	LARVAL DIFFERENTIATION % *Haemonchus*	*Oesophagostomum*	*Bunostomum*	*Trichostrongylus*	*Ostertagia*
BEFORE DOSING							
22.4.59	140	1,220	30	56	2	9	3
27.4.59	53	1,340	50	30	2	16	2
4.5.59	280	1,259	51	35	2	10	2
Mean —	157	1,273	44	40	2	12	2
Conversion to E.P.G.			560	509	25	153	25
AFTER DOSING							
11.5.59	13	86	0	12	2	85	1
18.5.59	166	133	0	14	1	85	0
Mean —	89	108	0	13	2	85	0.5
Conversion to E.P.G.			0	14	2	91.5	0.5
Calculated % Reduction in E.P.G.			100	97	92	60	98
INDIVIDUAL SHEEP							
BEFORE DOSING							
22.4.59	100	1,500	7	78	1	12	2
27.4.59	100	1,700	14	74	4	8	0
4.5.59	No sample						
Mean —	100	1,600	10.5	76	2.5	10	1
Conversion to E.P.G.			176	1,208	40	160	16
AFTER DOSING							
11.5.59	33	66	0	16	0	80	4
18.5.59	66	33	0	10	5	85	0
Mean —	49	49	0	13	3	82	2

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TABLE 3. DATA — 25 SHEEP EACH DOSED 2-oz. MATERIAL "Z"

BEFORE DOSING

Date	EGG COUNTS *E.P.G. Strongyloides*	EGG COUNTS *E.P.G. Others*	LARVAL DIFFERENTIATION % *Haemonchus*	*Oesophagostomum*	*Bunostomum*	*Trichostrongylus*	*Ostertagia*
22.4.59	66	1,006	21	52	7	16	4
27.4.59	106	1,359	46	32	3	17	2
4.5.59	133	1,546	27	62	2	7	2
Mean —	101	1,304	31	49	4	13	3
Conversion to E.P.G.			404	639	52	170	39
			AFTER DOSING				
11.5.59	33	13	0	0	0	100	0
18.5.59	33	13	0	0	0	100	0
Mean —	33	13	0	0	0	100	0
Conversion to E.P.G.			0	0	0	13	0
% Reduction			100	100	100	92	100
			INDIVIDUAL SHEEP				
			BEFORE DOSING				
22.4.59	100	2,500	35	48	2	11	4
27.4.59	200	2,000	29	47	1	20	3
4.5.59	—	2,800	20	66	0	12	2
Mean —	100	2,433	28	54	1	14	3
Conversion to E.P.G.			681	1,314	24	341	73
			AFTER DOSING				
11.5.59	66	100	0	4	3	92	1

Post Mortem Examination on 11.5.1959 — No worms found, see text.

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TABLE 4. DATA — 25 SHEEP EACH DOSED 2-oz. MATERIAL "Y"

BEFORE DOSING

Date	EGG COUNTS *Strongyloides* E.P.G.	EGG COUNTS *Others* E.P.G.	LARVAL DIFFERENTIATION % *Haemonchus*	*Oesophagostomum*	*Bunostomum*	*Trichostrongylus*	*Ostertagia*
22.4.59	53	1,286	30	46	3	16	5
27.4.59	93	1,526	44	34	2	18	2
4.5.59	144	1,815	49	27	6	16	2
Mean —	97	1,542	41	36	3	17	3
Conversion to E.P.G.			632	555	46	261	46
			AFTER DOSING				
11.5.59	33	19	0	0	3	95	2
18.5.59	73	13	0	0	0	100	—
Mean —	53	16	0	0	2	97	1
Conversion to E.P.G.			0	0	0	16	0
% Reduction in E.P.G.			100	100	100	94	100
			INDIVIDUAL SHEEP				
			BEFORE DOSING				
22.4.59	100	600	23	48	6	19	4
27.4.59	No sample						
4.5.59	100	2,100	40	42	2	14	2
Mean —	100	1,350	32	45	4	16	3
Conversion to E.P.G.			432	608	54	216	41
			AFTER DOSING				
11.5.59	66	66	0	5	0	95	0
18.5.59	66	133	0	0	4	96	0
Mean —	66	99	0	3	2	95	0

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Experiment 4.

In this trial phenothiazine, Material "Y" was compared with tetrachlorethylene emulsion and Government Nodular Worm Remedy, with and without pre-stimulation closure of the oesophageal groove by copper sulphate solution. Each group consisted of ten sheep which were in very poor condition, and hence it was considered prudent to reduce the doses from those usually prescribed.

The worms present were:-

Bunostomum, Haemonchus, Oesophagostomum, Trichostrongylus, and *Strongyloides.*

TABLE 5.

Groups	1. Material "Y"	2.	3. Tetrachlorethylene 33⅓% Emulsion	4.	5. Government Nodular Worm Remedy	6.
Dose	1¾-ozs.	1¾-ozs.	15 ml.	15 ml.	No. 2 spoon (1.8 gm)	
Pre-stimulation	No.	Yes.	No.	Yes.	No.	Yes.
% Reduction e.p.g.	86%	86%	36%	60%	47%	53%

RESULTS:

It is evident that pre-stimulation did not improve the efficacy of the phenothiazine, greatly enhanced that of Tetrachlorethylene Emulsion, but was of doubtful significance in the case of Government Nodular Worm Remedy.

DISCUSSION:

The results of these preliminary trials, under South African conditions in both cattle and sheep, reveal that the phenothiazine preparation — material "Z" — with 42% particles less than 5 mu, 78% less than 10 mu and 22% 10-17 mu, was very efficacious. This would confirm other workers' findings that phenothiazine preparations containing a large preparation of particles, by weight, under 10 mu and all under 20 mu, is a more effective anthelmintic than coarser preparations, particularly for combating trichostrongyles. The work reported here suggests that fine phenothiazine is also very effective against *Oesophagostomum* under South African conditions. The fact that there may be an increased and more rapid absorption of fine particle phenothiazine did not seem to be of any great significance in these trials as no toxicity was observed. It will be noted that in Trial 4 even after the closure of the oesophageal groove prior to dosing, no toxicity nor photosensitization was encountered.

CONCLUSION:

These pilot trials confirm the effcacy of fine particle phenothiazine against those worms of sheep and cattle which are affected by Phenothiazine.

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ACKNOWLEDGEMENTS:

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DIE HISTOPATOLOGIESE DIAGNOSE VAN GOUSIEKTE.

J. D. SMIT.*

Alhoewel gousiekte reeds gedurende die pioniersdae van veeartsenykunde in S.A. bekend was, bly dit nog steeds 'n aktuele en ekonomies belangrike probleem. Veral die korrekte diagnose van die siekte lewer dikwels veel probleme. In die vroeë literatuur is gousiekte beskryf as 'n hartversaking as gevolg van 'n opvallende dilatasie van veral die linker ventrikel. 'n Geleidelike verplasing van die spiervesels deur fibroblaste en kollagene vesels het gelei tot 'n verlies in elastisiteit en saamtrekbaarheid met dilatasie tot uiteindelike gevolg. Theiler, du Toit en Mitchell[7] beskryf 'n opvallende dilatasie van albei ventrikels en atria van die hart, die wande van die ventrikels was abnormaal dun met 'n baie ferme konsistens en 'n ligte grys tot rooskleurige voorkoms. Steyn[5] beweer dat in 'n groot aantal natuurlike asook eksperimentele gevalle hy in die reël geen letsels van diagnostiese belang kon vind nie. Hy beweer dat in geen enkele geval makro- of mikroskopiese letsels in die hartspier gesien is nie.

Alhoewel groot getalle van verdagte gousiekte-uitbreke jaarliks voorkom was die bevestiging van die diagnose altyd 'n groot probleem. Die diagnose kon wel in 'n sekere persentasie van gevalle bevestig word deur die voorkoms van 'n fokale kroniese miokarditis. Om 'n enigsins betroubare diagnose te kan maak moet 'n groot aantal sneë, geneem van verskillende dele van die hart, ondersoek word. Selfs dan was alleen die positiewe diagnoses betroubaar, want 'n negatiewe ondersoek het nog nie met sekerheid letsels in die res van die hart uitgesluit nie.

Die diagnose word nog verder bemoeilik deur die feit dat verskillende plante betrokke is by die ontstaan van gevalle van gousiekte. Vroeër is aangeneem dat alleen *Pachystigma pigmaeum* verantwoordelik is. Onlangse waarnemings[1] het bewys dat ook ander plante verantwoordelik kan wees. Deur omstandigheidsgetuienis en daaropvolgende eksperimentele werk is bewys dat *Pavetta harborii* gousiekte kan veroorsaak[2]. Volgens veldondersoeke van histologies positiewe gevalle van gousiekte moet ons vandag aanneem dat ook harige goorappel, *Pygmaeothamnus zeyheri* var. *rogersu*, *Pachysugmathamnus* (Natalse gousiektebossie) en moontlik ook *Pygmaeothamnus chaemaeodendrum* var. *setulosis* (Natalse harige goorappel) gousiekte kan veroorsaak. Die diagnose word dus al meer ingewikkeld en het al meer probleme opgelewer.

In 'n poging om 'n meer bevredigende en betroubare metode van diagnose te vind is 'n sistematiese ondersoek van die hart onder-

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neem. Uit hierdie ondersoek het geblyk dat sekere dele van die hart baie meer konstant die letsels toon. Die letsels het altyd in die apex van die hart voorgekom. In vroeë gevalle was dit meesal net beperk tot die spits en het geen letsels in die res van die hart voorgekom nie. In meer uitgesproke gevalle het die letsels ook in die linker vertrikelwand en in die septum voorgekom, en in uitgesproke gevalle ook in die regter ventrikelwand. Geen letsels kon vasgestel word in die voorkamer nie. Veral is ook aandag gegee aan die omgewing van die a-v-knoop maar ook hier kon geen letsels vasgestel word nie. In die meeste gevalle is die letsels baie fokaal en is in werklikheid alleen 'n minimale deel van die hart-apex aangetas. Die vraag ontstaan dus: wat is die oorsaak van die hartversaking? Vroeër is aangeneem dat dilatasie as gevolg van oorkompensasie die oorsaak is. Dit kan egter alleen die geval wees by hoë uitsondering. In die meeste gevalle is daar geen sprake van dilatasie nie. Dit was opvallend dat daar so 'n groot aantal purkinje-vesels voorkom in die apex van die hart, juis in die gebied waar gewoonlik die mees opvallende fibrose voorkom. Dit is ook bekend dat die hartimpulse deur die spits gaan. In sommige spesies gaan dit hoofsaaklik van die hartkamerwand deur die apex in die septum op en in die bok hoofsaaklik vanaf die septum deur die apex na die ventrikelwand. Na aanleiding van die verspreiding van die histologiese letsels in die hart wil dit dus voorkom of die patogenese van die hartversaking verklaar kan word deur 'n versteuring van die impuls waar dit deur die beskadigde apex gaan. Die fokale kroniese miokarditis beskadig heelwaarskynlik die geleidingsvermoë van die purkinje-vesels. In sommige van die sneë wil dit voorkom of daar wel degenerasie van die purkinje-vesels voorkom. Hierdie versteuring lei dan tot 'n toestand van akute hartfibrillasie en uiteindelik tot hartversaking. Hierdie veronderstelling word gestaaf deur die kliniese bevindings, en ook deur voorlopige kardiografiese ondersoeke[4]. Akute hartblok as gevolg van versteuring in die meganisme van impulsgeleiding in die hart kom ook by ander toestande voor. In die mens word dit beskryf by gevalle van fokale miokarditis in die omgewing van die a-v-knoop in die tussenskot by die oorgang van voorkamer na kamer. In gevalle van difterie is dit nie 'n onbekende letsel nie[3]. In eksperimentele gevalle van *Schizotrypanum cruzi*[6] kom 'n diffuse interstisiële miokarditis voor, maar veral opvallend is die fokale letsels wat voorkom aan die basis van die regter ventrikelwand. Dit word beweer dat die miokarditis die aurikulo-ventrikulêre sisteem aantas en die geleidingsvermoë versteur. Dit lei dan ook tot 'n akute hartblok. Dit kan dus met 'n redelike mate van sekerheid aanvaar word dat die apex van die hart die swak skakel in die patogenese van gousiekte is, en akute hartblok die direkte oorsaak van dood. Van 'n diagnostiese standpunt gesien is die apex dus die belangrikste deel van die hart om te ondersoek.

Die histologiese beeld by gousiekte gesien en veroorsaak deur die verskillende plante, verskil ook in 'n mate. Die relatiewe giftigheid van die plante bepaal tot 'n groot mate die graad van akuutheid

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van die letsel. In die geval van *Pavetta harborii* kry ons 'n taamlike akute letsel, wat redelik diffuus versprei is deur die hartspier. Die letsel bestaan essensieël uit 'n subakute tot kroniese miokarditis, met heelwat vervanging van spierbundels deur fibroblaste. Gewoonlik is nog nie veel kollagene weefsel neergelê nie. 'n Mate van rondesel- (limfosiet) infiltrasie kom wel voor, asook 'n mate van kongestie. In gevalle van gousiekte veroorsaak deur harige goorappel kry ons 'n meer kroniese beeld, met letsels taamlik diffuus versprei deur die hart. 'n Opvallende kollagene neerlegging kom voor. Feitlik geen kongestie en selinfiltrasie word gesien nie. Die letsel by *P. thamnus* is tussenin, met meer kongestie en selinfiltrasie maar ook alreeds heelwat neerlegging van kollagene vesels.

Dit is veral in gevalle van *P. pygmaeum* afkomstig van Ventersdorp, waar die letsels heelwat afgewyk het van die erkende beeld van gousiekte. Baie akute letsels het hier voorgekom, met bloedings, edeem, kongestie en aktiewe spierdegenerasie. Histologies het die beeld meer gedui op gifblaarvergiftiging. Dit was eers na 'n deeglike plaaslike ondersoek dat die diagnose van gousiekte aanvaar is. Nog 'n ongewone verskynsel was 'n fokale hiperemie van die rumenslymvleis. Dit was so opvallend dat 'n tentatiewe diagnose van arseen-vergiftiging gemaak is. Hierdie verskynsel is nog alleen hier opgemerk en ek kan geen verklaring daarvoor aanbied nie. In hierdie gevalle kon die diagnose van gousiekte alleen gemaak word na 'n sistematiese ondersoek van die hart. Die tipiese gousiekteletsel het alleen in die apex voorgekom en deur hierdie deel van die hart te ondersoek kon 'n definitiewe diagnose gemaak word.

Summary.

A brief review of the "gousiekte" literature is given. The various plants responsible for cases of "gousiekte" are mentioned. A comparative description is given of the histopathological lesions produced by the various plants.

The pathogenesis of the heart failure is discussed. The conclusion is drawn that dilatation of the heart is not the explanation for the heart failure.

The importance of the constant occurrence of focal lesions in the apex of the heart is stressed and it is emphasized how important this portion of the heart is in the histological diagnosis of the disease.

The theory is postulated that the focal myocarditis in the apex is responsible for an acute heartblock resulting in acute fibrillation and heart failure.

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AN OUTBREAK OF WHITE MUSCLE DISEASE IN LAMBS.

R. C. TUSTIN*.

Introduction:

A small outbreak of white muscle disease (or stiff lamb disease) was recently encountered in a flock of German Merino sheep. As this is the first recorded outbreak of the disease in South Africa it is deemed worthy of note.

White muscle disease is a condition in which degenerative and necrotic changes of striated muscle and/or of the myocardium occur. It is considered that a Vitamin E deficiency plays a rôle in the aetiology of the condition but recently it has been shown that a selenium deficiency may also be incriminated as an aetiological or predisposing factor. The disease has been known for many years in Europe and North America where it usually occurs in lambs after they have been turned out to pasture in the spring and where their dams have received a poor diet.

Hartley and Dodd[1], in a review of muscular dystrophy in New Zealand livestock, describe the condition in various age groups of sheep, viz. congenital form, in lambs up to three months of age, in hamels 9 to 12 months old and in ewes.In New Zealand the disease in lambs up to three months of age occurs in the late winter and spring, and has been seen in flocks where the ewes in the later stages of pregnancy and early lactation have been grazed on autumn saved legume dominant pastures and supplemented by excellent quality lucerne hay, and also where ewes have been wintered on swedes or turnips and moved at lambing to lush legume dominant pasture. The legumes involved have usually been red and white clovers but lucerne and subterranean clovers have also been incriminated. Most outbreaks have occurred on highly improved, well-managed farms, often after heavy applications of fertilizers and where there is ample lush green feed available for the nursing ewes and lambs.

The majority of outbreaks in New Zealand are seen in lambs at pasture and are apparently unassociated with exercise. On some properties, however, where there might or might not have been a few cases on pasture, driving has precipitated a severe outbreak. Tocopherol therapy appears to be beneficial in lambs affected at pasture, but of little value in those following driving.

Whiting, Willman and Loosli[2], showed that a low level of tocopherols in the milk of ewes fed a ration of lucerne and beans was the cause of muscular dystrophy observed in the lambs. The condition was cured by administering tocopherols to the lambs but it was not prevented by *prepartum* supplementation with tocopherols.

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Recently it has been found that selenium appears to have a definite protective effect on white muscle disease in lambs. Muth, Oldfield, Remmert and Schubert[3], have shown that when ewes were fed a basal ration consisting of Ladino clover hay originating from a severely affected area and 0.25 lb. of oats per ewe per day plus 0.1 ppm. selenium (calculated for the total ration) their lambs were almost completely protected. Lambs of ewes fed the basal ration plus alpha-tocopherol supplements were not protected. In a control group fed the basal ration with no supplements 11 lambs out of 15 developed lesions. In another group of ewes receiving a ration of Ladino clover hay and alfalfa hay grown in relatively non-affected areas and 0.25 lb. of oats per ewe per day one lamb out of 18 became affected. Analyses of the hays fed in the experiment indicated levels of less than 0.1 ppm. selenium.

Trials conducted in New Zealand by Hartley, Grant and Drake[5], suggest a protective action of selenium when given to the lambs in naturally occurring outbreaks of muscular dystrophy.

Arising from the idea that subclinical muscular dystrophy might be one of the factors for the slow growth rate of lambs in the South Island of New Zealand—a condition known as "ill thrift"—Mclean, Thompson and Claxton[6], investigated the possible effect of selenium supplements on the growth rate of various classes of lambs. The improvement in live weight gains was regarded as highly significant.

History of the Outbreak:

During September, 1959, two live German Merino lambs were presented for examination. One was unable to stand; in the other the disease was not so far advanced. It showed an arching of the back when standing, disinclination to move and stiffness, especially of the hind limbs, when walking. Both lambs were about a month old.

The owner of the animals, who farms near Kroonstad, reported that he had had five cases of the disease in his German Merino flock which consisted of 45 ewes. The first lambs in this flock were born in May and by the beginning of September 52 lambs had arrived, but the disease had occurred only during the previous month in lambs of about a month of age. The ewes and lambs were grazing on veld near the house and received supplements of lucerne day, groundnut hay, yellow mealie meal milled on the farm and a proprietary lick, to which salt and bonemeal had been added. The daily ration of mealie meal to the ewes amounted to approximately three-quarters of a bucket. Due to the very dry conditions at the time, the natural grazing was very poor.

At the time of writing five lambs had shown symptoms of the disease. Two had died, one of which had been sick for about six days before death and the other for about 15 days. One had recovered spontaneously after showing stiffness for 12 days. The other two were brought to Onderstepoort for examination and were autopsied. According to the owner the ewes were in fair condition and had been on the farm since June, 1955. These were the first cases of this disease that he had experienced.

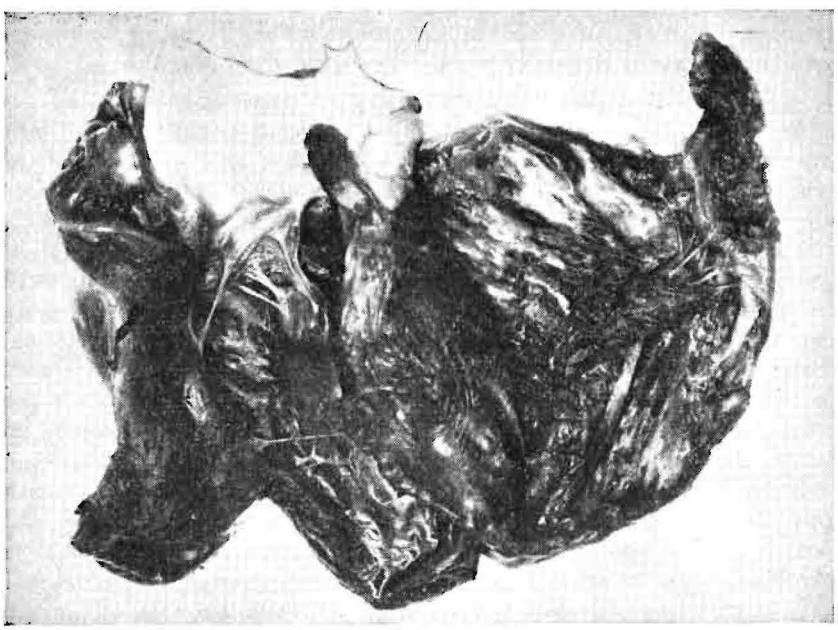
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The farmer also runs a flock of 500 Merino ewes on the farm. These lambed during April and May and were given supplementary lucerne hay, cowpea hay and ensilage for a short while after lambing. They had subsequently been grazing only on veld. No cases of the disease occurred in this flock.

Pathology:

Both lambs were in poor condition and appeared small for their age.

The lamb which was still able to walk, had shown signs of the disease for about five days and on *post mortem* typical bilaterally symmetrical lesions were present particularly in the thigh and shoulder muscles. The affected muscles were a uniform pale colour with a granular appearance. Parts of some muscles appeared normal and the differentiation between the two was distinct and marked In the wall of the right ventricle wh.tish yellow areas and longitudinal streaks were present beneath the endocardium. These extended approximately one millimetre into the heart (see figure). The kidneys were a pale yellow-brown colour with numerous very small white foci present in the cortex.



Histologically the affected muscles showed changes varying from hyaline degeneration to necrosis with fragmentation of the fibres. Some segments of fibres showed a light deposition of calcium. A proliferation of the nuclei of the muscle fibres was present in parts and a light infiltration of macrophages and lymphocytes occurred

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between the muscle fibres. In isolated areas a slight increase of connective tissue was present. The heart lesions proved to be a calcification of the subendocardial layer with a light focal infiltration of lymphocytes. In odd segments of muscle fibres of the tongue hyaline degeneration was observed. Severe fatty degeneration and cloudy swelling was present in the kidneys with a focal deposition of calcium in a granular form occurring between the tubules, in the epithelial cells, in Bowman's capsules and in the glomeruli. The odd glomerulus appeared atrophic and fragmented. The liver showed a moderate centrilobular fatty infiltration.

The muscular lesions of the other lamb, which had been sick for 10 to 12 days, were also bilaterally symmetrical and the thigh and shoulder muscles were particularly affected. The lesions appeared as dull white longitudinal streaks in the muscles. The heart was not affected. The appearance of the lesions histologically was similar to that of the above case except that the deposition of calcium was more severe. The kidneys showed severe fatty degeneration but no calcium deposition.

DISCUSSION.

While outbreaks of white muscle disease have not been recorded before in sheep in South Africa, sporadic cases, other than in the present outbreak, have been encountered with increasing frequency and the disease is probably more widespread than is realised.

The use of legumes in relation to the disease is stressed, as it is considered that they might contain anti-vitamin E substances which are responsible for some outbreaks. Whether this is the case, or whether, in the light of recent investigations, it is purely a trace element deficiency, or whether both vitamin E and selenium play a rôle, has yet to be determined. Mclean *et al*[6] state that the explanation of the mode of action of selenium in producing a growth response must be rather speculative at the present time, that there can be little doubt that traces of selenium are required by the animal for normal metabolism, that vitamin E and selenium are interrelated in their metabolic functions and that vitamin E cannot replace the need for selenium. The growth responses which they obtained in lambs might therefore be taken as an indication of specific selenium deficiency, one manifestation of which is a slowing up of growth rate and another, a predisposition to muscular dystrophy and possibly other conditions in which the metabolism of vitamin E is involved.

Other products with an anti-vitamin E action which should be mentioned are the unsaturated fatty acids found in fish liver oils and fish and meat meals.

SUMMARY.

A small outbreak of white muscle disease in lambs is described. The rôle of vitamin E and selenium in the aetiology of the condition

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is briefly discussed. The main constituents of the diet of the ewes and lambs were lucerne hay and groundnut hay.

ACKNOWLEDGEMEMNT.

Thanks are due to the Director of Veterinary Services for permission to publish this article.

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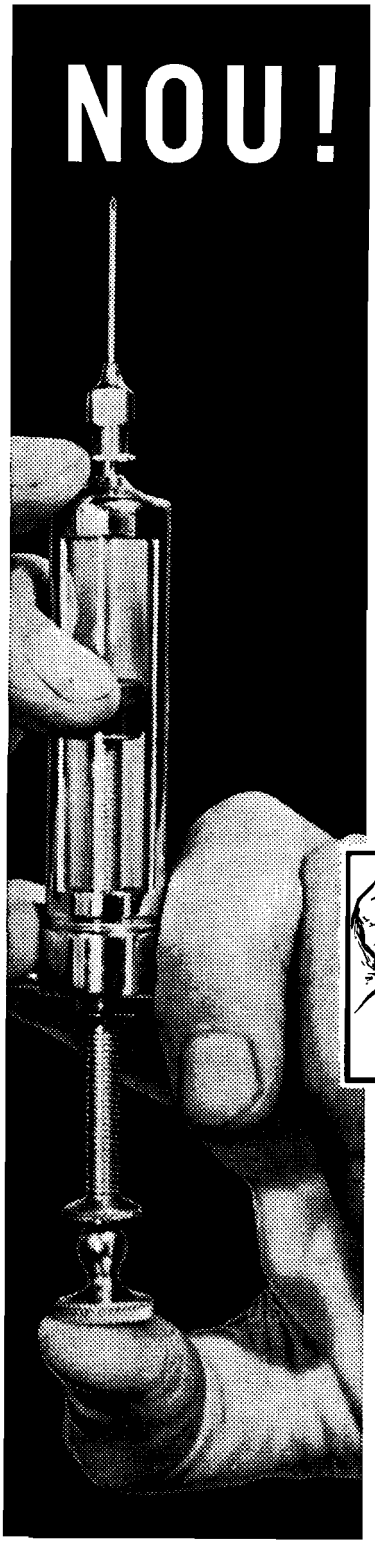
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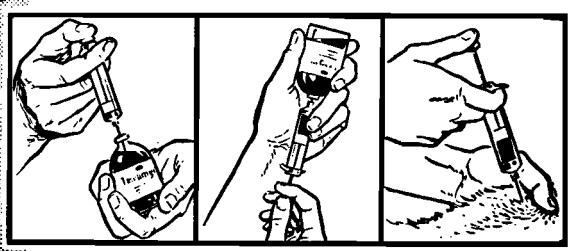
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THE EFFECTS OF (a) SOLUBLE PHOSPHATE AND (b) COBALT ON THE UTILISATION OF UREA NITROGEN BY CATTLE.

N. BARRIE* and R. CLARK‡.

Introduction:

The ability of ruminants to utilise urea nitrogen through the activity of the ruminal flora is well known and experiments in South Africa with urea and molasses as a supplement to winter rations have been reported.

The efficient use of urea nitrogen depends on many factors, chief among which is the simultaneous presence of an adequate source of readily available carbohydrate. The large scale synthesis of microbial protein must also depend on the availability of other elements such as phosphorus and sulphur while cobalt is known to be essential for many microbial activities.

Insoluble phosphates, such as bone meal and bone flour, are not directly available to the ruminal organisms. Although the soluble phosphate in the rumen is maintained at a high level by the saliva[3] it was considered that this might not be optimal to support large scale protein synthesis. Previous (unpublished) trials had demonstrated that South African molasses contained sufficient sulphur and the addition of that element was without effect. It was therefore decided to test out the effects of (a) substituting soluble phosphate for bone flour and (b) adding cobalt, when feeding a urea and molasses supplement.

Methods:

The following mixtures were prepared

1. Molasses ..... 78 %
   Degelatinised bone flour ..... 6.25 %
   Urea ..... 7.0 %
   Co (As $CoSO_4$) 0.003%
2. As for Mixture I but without cobalt.
3. As for Mixture I but with disodium phosphate substituted for degelatinised bon flour to equal P value.
4. As for Mixture 3 but without cobalt.
5. Molasses 93%
   Degelatinised bone flour ..... 7 %

Eight head of cattle between 1½ and 2½ years of age were used in each group. They were kept in open paddocks, with poor quality grass hay and water available *ad lib*. Each group received 5lb. silage

*The mixtures were prepared and generously donated by Messrs. Cooper and Nephews, S. Af. (Pty.) Ltd.

†Senior State Veterinarian, Heidelberg, Tvl.

‡Dept. of Physiology — Onderstepoort.

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per head per day and one pint of one of the above mixtures per head per day. The experiment lasted for 15 weeks and the results were based on the live weights of the animals before and after the experimental period.

RESULTS

Gr.	Supplement	Av. Init. Wt. (lb.)	Av. Final Wt. (lb.)	Diff. (lb.)
1.	Mol. + B.F. + Urea + Co.	565	643	+78
2.	Mol. + B.F. + Urea	562	623	+61
3.	Mol. + Na_2HPO_4 + Urea + Co	561	659	+98
4.	Mol. + Na_2HPO_4 + Urea	560	647	+87
5.	Mol. + B.F.	561	533	—28

The loss in weight shown by the animals in Group 5 as compared to the gain shown by all groups receiving urea, leaves no doubt that the urea nitrogen was utilized.

Statistical analysis of the results obtained in the first four groups revealed the following:

(a) There was a significant difference (0.1% level) between groups 1 and 2, i.e. the addition of cobalt had a demonstrably beneficial effect when the phosphorus was given as bone flour.

(b) There was also a significant difference (1.0% level) between groups 2 and 4, i.e. soluble phosphate gave a better response than bone flour when no cobalt was added.

(c) There was no significant difference between groups 1 and 3, nor between groups 3 and 4.

DISCUSSION.

Consideration of the above results led to the query as to whether the disodium phosphate was not contaminated with traces of cobalt but this was found not to be the case.

Preliminary inspection of the results indicated that soluble phosphate gave a better result than bone flour and that the addition of cobalt improved the mixture in both cases. As stated above, however, no statistical difference could be shown between bone flour plus cobalt and disodium phosphate without cobalt, nor could the beneficial effect of cobalt be proved when disodium phosphate was used. There are two possible explanations for these facts.

1. That the apparent differences are in fact real but that the groups were too small to allow of statistical proof.

2. That the limitation of the nitrogen intake precluded any extra significant weight gains, despite the possible potentially additive beneficial effects of soluble phosphate and cobalt.

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INVESTIGATION INTO THE TOXICITY OF CARBOCAINE (BOFORS) — A NEW LOCAL ANAESTHETIC

C. F. B. Hofmeyr*

(*Received for publication, 17th December,* 1959)

Carbocaine is an amide (1-Methyl-2-piperidine carbon)-2-6 dimethylarilide hydrochloride. It is highly stable and can be boiled for several hours.

It is claimed not to have any influence on the action of sulphonamides or antibiotics.

In this study the vervet monkey (*Cercopithecus aethiops*) was used.

Firstly it was decided to test the cumulative effect of Carbocaine by giving intramuscular injection of the Carbocaine half-hourly until signs of toxicity appeared. The results are indicated on Table I.

The next experiment was the determination of the STD_{50} intravenously i.e. the smallest quantity of Carbocaine by intravenous injection giving rise to clear side effects.

TABLE II.

20% CARBOCAINE INJECTED INTRAVENOUSLY INTO V. CEPHALICA IN LESS THAN 5 SECS.

SUBJECT Sex		Weight	Dose	SYMPTOMS
1)	♀	3.2 Kg.	7 mgm/Kg.	Slightly aggressive. Movements not very exact.
2)	♀	2.8 Kg.	8 mgm/Kg.	Slightly aggressive.
3)	♀	2.2 Kg.	8 mgm/Kg.	Very aggressive. Scratches the body.
4)	♀	3.2 Kg.	9 mgm/Kg.	Very slight incoördination.
5)	♀	3.6 Kg.	9 mgm/Kg.	Fair degree of incoördination and dizziness.
6)	♀	2.0 Kg.	9 mgm/Kg.	Very slight incoördination.
7)	♀	2.6 Kg.	9 mgm/Kg.	Threatening attitude.
8)	♀	2.05 Kg.	10 mgm/Kg.	Fair degree of incoördination.
9)	♀	1.9 Kg.	10 mgm/Kg.	Lack of coördination and of balance.
10)	♀	2.0 Kg.	10 mgm/Kg.	Slight incoördination.
11)	♀	2.0 Kg.	10 mgm/Kg.	Somewhat aggressive.
12)	♀	3.3 Kg.	10 mgm/Kg.	Some incoördination and dizziness.
13)	♀	1.9 Kg.	10 mgm/Kg.	Lack of coördination and of balance.
14)	♀	2.5 Kg.	11 mgm/Kg.	Lack of coördination and of balance.
15)	♀	1.7 Kg.	12 mgm/Kg.	Threatening attitude. Slight incoördination.
16)	♀	2.1 Kg.	12 mgm/Kg.	Slight dizziness and incoördination.
17)	♀	2.4 Kg.	12 mgm/Kg.	Slight aggressiveness. Incoördination and dizziness.
18)	♀	2.0 Kg.	13 mgm/Kg.	Incoördination, dizziness and excitement.
19)	♀	2.4 Kg.	14 mgm/Kg.	Convulsions, salivation. After eight mins. almost recovered but still aggressive.

* Department of Surgery, Faculty of Veterinary Science, Onderstepoort.

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TABLE I.

1. Cumulative Effect s Injections into Gluteus or Triceps

SUBJECT Sex	Weight	Dose	No. of injections at half-hour intervals until signs of toxicity appeared.	Time elapsed between last injection and signs of toxicity	Time taken for symptoms to disappear	SYMPTOMS
1) ♀	2.8 Kg.	20 mgm/Kg.	3	4 min.	21 min.	Incoördination. Dizziness.
2) ♀	3.2 Kg.	20 mgm/Kg.	3	4 min.	14 min.	Incoördination.
3) ♀	2.9 Kg.	20 mgm/Kg.	3	4 min.	22 min.	Incoördination and inability to walk.
4) ♂	2.9 Kg.	20 mgm/Kg.	3	5½ min.	13 min.	Incoördination and dizziness.
5) ♀	2.9 Kg.	20 mgm/Kg.	3	2½ min.	24 min.	Clonic spasms. Marked salivation. Great depression.
6) ♂	3.2 Kg.	20 mgm/Kg.	3	1 min.	Died after 32 min.	Clonic spasms. Marked salivation. Great depression. Death.
7) ♀	3 Kg.	20 mgm/Kg.	3	7 min.	15 min.	Incoördination. Clonic spasms.
8) ♀	2.9 Kg.	20 mgm/Kg.	2	24 min.	15 min.	Incoördination.
9) ♀	3.6 Kg.	20 mgm/Kg.	3	8 min.	18 min.	Fair incoördination.
10) ♀	2.3 Kg.	20 mgm/Kg.	3	1 min.	11 min.	Incoördination.

Discussion

(1) It is clear that a cumulative effect is demonstrated in one case after two injections at half-hourly intervals and in the other nine after three injections. There was one death. In the surviving cases the symptoms disappeared quickly i.e. between 11 and 24 minutes but not as quickly as in the second experiment where a single toxic dose was administered.

(2) Table II indicates aggressiveness in some cases. These monkeys are often inclined to adopt threatening attitudes on release after being handled. These cases however gave the impression that there was definite stimulation of the C.N.S. by the drug. Slight but clear indications of toxicity were evident in three cases out of four with the dose at 9 mgm/Kg. and more clearly marked signs were shown by five out of six receiving a dose of 10 mgm/Kg.

The STD_{50} has therefore been determined at 9 mgm/Kg. of Carbocaine intravenously for the vervet monkey (*Cercopithecus aethiops*).

Summary

1. Cumulative effect was demonstrated in the vervet monkey (*Cercopithecus aethiops*) at the second intramuscular injection of Carbocaine (Bofors) at half-hourly intervals of 20 mgm/Kg. in one case and in nine at the third injection.
2. The STD_{50} has been determined at 9 mgm/Kg. of Carbocaine intravenously.

Acknowledgements

The author wishes to record his appreciation to Messrs. Westdene Products (Pty.) Ltd. for making the Carbocaine available and for arranging the loan of the monkeys from the Poliomyelitis Research Foundation to whom further thanks are due.

To Mr. D. de Vos, Chief Technical Officer of the Department of Surgery, the author is indebted for customary efficient assistance.

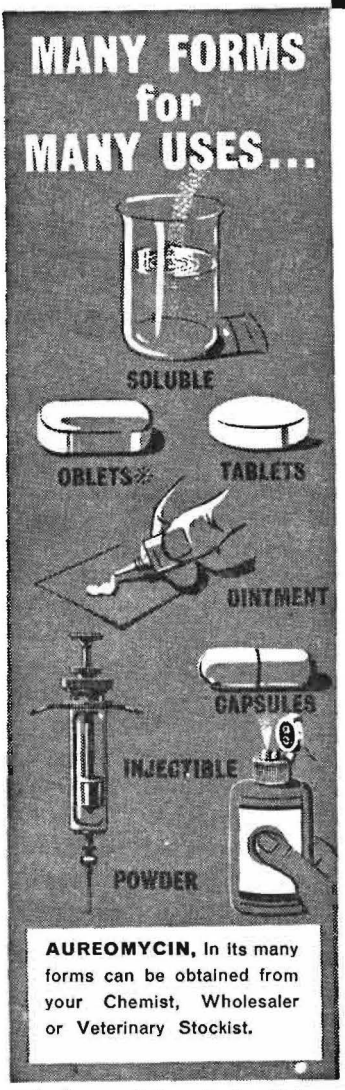
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LYMPHOID POLYPUS AT THE INTERNAL NARES OF A DOG, AND ITS SURGICAL REMOVAL.

D. H. G. IRWIN*.

Polypoid growths occur in the nostrils and external nares of animals, and arise from a proliferation of all layers of the mucous membrane. They may be in the nature of a papilloma or an adeno-fibrous hyperplasia of the mucosa and must be distinguished from angioma, lipoma, osteoma, ecchondroma, sarcoma and carcinoma. Nasal growths may occur in rhinosporidiosis, schistosomiasis, blasto-mycosis, tuberculosis and actinomycosis.

Judging from the literature it would appear that the polypus described below is unusual in its histology, its location, and its surgical relief.

Subject:

This was an eight-year-old male wire-haired Fox Terrier, weighing 13 Kg.

History:

About 5 months previously the owners noticed a respiratory sound only when the dog was breathing through the nose whilst exercising. This gradually became louder over the course of four months, when veterinary assistance was obtained. The practitioner suspected mechanical obstruction, and referred it to the Faculty Hospital for surgical correction.

Examination:

The nasal, lacrimal and malar bones were quite symmetrical. The teeth appeared healthy and the palate even and normal. No tenderness was elicited and no nasal or ocular discharge was present. Lymph nodes were not enlarged. Auscultation after rest revealed nothing unusual, but after exercise a nasal stridor occurred when the mouth was closed, being a little more marked on expiration. Both nostrils appeared equally affected.

X-ray examination revealed no abnormality.

General anaesthesia was induced by pentothal sodium (0.25 gm i.v. quite quickly) and the trachea was intubated to maintain anaes-thesia with nitrous-oxide-oxygen. By illuminated mirror examination a tumour about 1.2 cm diam. spherical in shape and red in colour was observed dorsal to the soft palate.

Surgical Treatment:

* Dept. Surgery, Onderstepoort.

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The following day the patient was reanaesthetized as described for the examination.

The endotracheal tube was taped to the lower jaw and a gag employed. With the dog in the ventral position the soft palate was incised from its free edge about 4 cm rostrad.

This disclosed a semi-pedunculated spherical body about 1.2 cm diam. attached to the dorsal surface of the naso-pharynx on a transverse plane immediately rostrad to the hamuli of the pterygoid bones. It was excised with tonsillectomy scissors. Haemorrhage was controlled by adrenalin soaked fibrin-foam and pressure, along with parenteral Haemostem[1]. The soft palate was sutured with 00 gut.

Within a few minutes the dog was sitting up, and showed mild pharyngeal discomfort. Convalescence was short, the patient being discharged two days later and 7 weeks later the owner reported improved habitus and vigour with no respiratory distress.

Histopathology:

Professor K. C. A .Schultz of the Department of Pathology reported a superficial inflammatory oedema with fibrosis and hyperplasia of the lymphoid tissue. The epithelial lining showed metaplasia.

DISCUSSION.

Previous reports on removal of polypi describe the use of a polypus snare for use via the nostril or alternatively, approaching the nostril by trephining the maxilla or nasal bone.

Summary:

The nature of nasal polypi is briefly recounted. The examination and removal of a hyperplastic lymphoid polypus situated dorsal to the soft palate in a dog is described.

ACKNOWLEDGEMENTS.

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Thanks are due to Prof. K. C. A. Schulz, Department of Pathology, for the histological examination.

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CONTRACTS AS THEY AFFECT A VETERINARIAN IN HIS PRACTICE.

By ANON.

Every citizen is presumed to know the law.

> *"Ignorance of the law excuses no man;*
> *"not that all men know the law, but*
> *"because 'tis an excuse every man*
> *"will plead, and no man can tell how to*
> *"confute him."*

One can say that no man can possibly know the law be he a scientist or a lawyer. In this short article my aim is to give as simply as possible some legal principles which should be borne in mind by a Veterinarian.

Purchasing Fixed Property whether it be his house or anything else.

In our law, ownership of fixed property only passes on registration of transfer in the name of the purchaser in the Deeds Office. The purchaser would be wise, therefore, never to part with any money for portion of the purchase price of fixed property except against registration of transfer in the Deeds Office in his name. The usual way of achieving this is to deliver to the seller's conveyancer a suitable guarantee, e.g. one issued by a Bank, Building Society or Insurance Company. Fixed properties are frequently purchased on the instalment plan. A purchaser should, however, only resort to this method of acquiring fixed property if the seller is of undouted financial standing. The reason for this is that should the seller go insolvent before transfer is registered in the name of the purchaser, the latter will lose the property and have the cold comfort of proving a concurrent claim in the insolvent estate.

Purchasing a Practice:

In purchasing a practice one should be advised by a qualified Attorney who should, in turn, be assisted by a Chartered Accountant in fixing the goodwill. Very great caution should be taken to see that an amount paid in respect of goodwill is not excessive. If outstanding debts are taken over, care should be taken to see that a suitable guarantee is given in regard to the recoverability of these debts. If goodwill is paid for, then the seller should agree to what is known as the Restraint Clause preventing him from practising in competition with the purchaser. This clause is what is known as a Restraint of Trade Cause. Before the Court will uphold the validity of any such clause, the Court must be satisfied that the clause is reasonable, both as regards the period and area. In the case of a veterinarian practising in a country district, his area would no doubt

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be considered as necessarily being larger than that of say, a medical practitioner. Nevertheless, great care must be taken to see that the area is not unnecessarily large and the period must be limited in time. From a perusal of numerous decided cases both in this country and in the United Kingdom, one would say that it would be unwise for the period of restriction to be longer than three years.

Books of Account:

Proper books of account should of course be kept for obvious reasons. In rendering an Income Tax Return it should be borne in mind that under Section 11 (2) (r) of the Income Tax Act No. 31 of 1941 as amended, an allowance not exceeding £100 is provided for in respect of medical and dental expenses and under Section 11 (2) (p) up to £150 in respect of entertainment. (Somewhat difficult to reconcile with the status of a professional man — nevertheless it can be availed of.)

The Relationship between a Veterinarian and his Assistant:

If a contract is entered into between a veterinarian and his assistant, it would be wise to determine precisely the duties to be performed by the assistant and the hours to be worked by him. If he uses his own motor car, care must be taken to lay down the precise terms and conditions under which the motor car will be used and paid for. It is customary in such circumstances for a log book to be kept.

The period of notice of termination to be given by either party should be set out and a reasonable restraint clause inserted to prevent the assistant from going into practice in opposition to the veterinarian on termination of the contract.

Partnerships:

When a partnership is entered into between Veterinarians, the document requires to be carefully prepared by a competent attorney. Apart from necessary matters such as the sharing of profits, provision for the death or disability of any partner, such matters as the use of motor cars and the payment therefor, should becarefully provided for. Any necessary Restraint Clause should be included. A partnership contract should include a clause providing for the appointment of an Accountant to supervise the books and to prepare the annual Balance Sheet.

Testamentary Dispositions:

It seems to be a popular fallacy that if a person dies intestate, the State takes a greater share of the estate than it would otherwise do. Nothing is further from the truth. What should be borne in mind by a Veterinarian is that apart from a small statutory provision contained in Act No. 13 of 1934, the surviving spouse of a deceased person does not succeed *ab intestato*. Consequently, if a person is married, it is very important that that person should have a Will providing for the surviving spouse and the children. If the estate is large a formation of a trust or trusts in favour of the children should

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be carefully considered. Here again the draftsmanship of a Will requires the attention of an experienced attorney. Consideration should be given for the disposal of the practice in the Will.

Guarantees:

No person should bind himself as surety or guarantor for another's obligations. Many a person's life has been ruined as the result of a hasty or ill-considered signature to a guarantee or suretyship.

Conclusion:

It has been said very wisely, a lawyer who acts for himself has a fool for a client — the layman who acts for himself will soon find that a stitch in time saves nine — timely legal advice may obviate disaster.

Engagement of a Locum:

The engagement of a locum tenens involves the entering into of a binding contract between the veterinarian and the locum. This is a contract which could be evidenced by letters, it could be verbal and, on the other hand, it might even be a properly drawn up legal document. If either party breaks the contract, what ever form it may take, the injured party has his remedy in the civil courts. If, for example, a locum failed to attend as he had undertaken to do, resulting in loss to the veterinarian, the veterinarian would be entitled in law to sue for the amount of such loss.

The breaking of a contract of this nature normally does not carry with it implications of professional misconduct. There may be a large number of reasons which would result in a locum failing to carry out his contract. Matters such as illness, unforeseen accidents and the like immediately come to mind. The South African Veterinary Medical Association must confine itself to dealing with matters of professional misconduct and not endeavour to sit in judgment of matters which concern the contractual relationship between two veterinarians. This is the policy which we believe has been adopted by the Medical Council and which we strongly recommend be adopted by the South African Veterinary Medical Association and, for that matter, the South African Veterinary Board.

INFORMATION ON WORM CONTROL.

A booklet entitled "A Guide for Veterinarians on Worm Control in Domestic Livestock" has been prepared to assist Veterinary Surgeons engaged in general practice to make use of the most up-to-date knowledge about both old and new anthelmintics; and to suggest how the drugs can be employed to the best advantage in relation to the information now available on the biology of parasites. Should any member of the Association wish to receive a copy, will he or she please communicate with the Technical Department, Cooper & Nephews, S. Af. (Pty.) Ltd., P.O. Box 2963, Johannesburg.

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XVIth INTERNATIONAL VETERINARY CONGRESS, MADRID — 1959.

RESOLUTIONS.

1. RADIO-BIOLOGY.

In view of the increasing impact of nuclear energy and radiobiology upon veterinary medicine and public health the XVIth International Veterinary Congress recommends that national and international veterinary authorities and institutions should undertake appropriate action with respect to the following fields:

(a) The utilization of radioisotopes as research, diagnostic and therapeutic agents.

(b) The use of ionizing radiation in food preservation and its public health implications.

(c) The measurement and evaluation of environmental contamination with radioactivity from fallout or nuclear accidents with respect to its effects on animals and, via the animal food chain, its effects on man.

(d) The undergraduate and postgraduate training of veterinarians to implement these recommendations.

II. VETERINARY MEDICINE.

The XVIth International Veterinary Congress draws the attention of national Governments to the importance of further researches into the practical application of blood grouping an similar procedures in animals in connection with animal husbandry and disease control.

III. VETERINARY MEDICINE.

"Attetion must be drawn to the increase in animal diseases due to the spread of industry and consequent contamination of the environment by toxic substances. Industrial and hygienic measures should be increased in order to reinforce prophylaxis of these diseases."

IV. EPIZOOTOLOGY.

"Recognizing the continued need for urgent action to establish further international standards for biological products for veterinary use, the XVIth International Veterinary Congress suggests that the Committee on Biological Standardisation of the World Health Organization, in collaboration with O.I.E. and F.A.O., be encouraged to extend, as soon as possible, its work towards the provision of international standards for additional suitable veterinary sub-

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stances. These meriting immediate attention are:

Antitoxins.—Cl. oedematiens alpha toxin.
Cl. Botulinum C and D.

Antiserums.—Swine fever.
Toxoplasmosis.

Vaccines.—Anthrax (Spore, type Pasteur).
Cl. welchii B and D.
Cl. botulinum.
Cl. chauvoei.
Cl. septicum.
Swine fever.
Canine distemper.
Newcastle disease.
Sheep Pox disease.

V. EPIZOOTOLOGY.

Since it is considered essential that opportunity for regular contact between research workers on foot-and-mouth diseases from all regions of the world should be provided it is recommended that, consistent with the existing arrangements between F.A.O. and O.I.E., every effort should be made to arrange for such regular contacts and distribution of the proceedings of the meetings."

VI. LIST OF DISEASES OF ANIMALS.

"The XVIth International Veterinary Congress authorizes the Permanent Committee to publish the List of Diseases of Animals as prepared in 1957 by its Subcommittee, subject to the inclusion of various subsequent amendments. It further resolves that the Secretariat should endeavour to secure firm orders for the List prior to its publication, but that meanwhile arrangements should be made for the printing of a minimum of 2,000 copies."

VII. PUBLIC HEALTH.

"Recognizing the fundamental contributions of veterinarians to human health through the prevention and control of zoonoses, and through studies in comparative medicine of human and animal disease and inter-relationship in general, the XVIth International Veterinary Congress *recommends* to national and international health and veterinary authorities:

(1) Intensification of activities for the control and where possible eradication of the zoonoses;
(2) Increased research efforts to clarify newly emerging communicable disease relationships of man and animals;
(3) Close collaboration in comparative medical studies on interrelated problems of human and animal health."

VIII. WORLD ANIMAL HEALTH YEAR, 1963.

"Be it resolved that the XVIth International Veterinary Congress proclaim 1963, which will be the 100th anniversary of initiation of the International Veterinary Congresses, as World Animal

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Health Year, in support of the 'Free the World from Hunger Campaign'. The XVVIth International Veterinary Congress urges all member countries and various international agencies, such as F.A.O., W.H.O. and O.I.E., to increase their efforts to control and eradicate animal diseases that threaten the health and economic well-being of man. The control and eradication of animal diseases is essential for raising the standard of living of man and should receive a high priority in the years preceding the World Animal Health Year."

IX. VETERINARY TEACHING.

"The XVIth International Veterinary Congress recommends that Veterinary Schools introduce and develop the training of specialists, particularly in: veterinary public health; inspection and technology of products of animal origin; zootechny and nutrition.

Each country will choose the method of specialist teaching most suitable (compulsory or optional, undergraduate or post-graduate) with a view to producing the greatest possible number of specialists."

OBITUARY.

Dr. E. J. PULLINGER.

Dr. Pullinger (Jimmy) died on 10th November, 1959, after a long illness. He graduated in 1929 and went to London to take his M.R.C.V.S. and Diploma in Bacteriology. In London he was employed by the United Dairies. He returned to Onderstepoort for a short while and then joined the Johannesburg Municipal Service in 1939. During the war he served with the South African Veterinary Corps attaining the rank of Major. After the war he completed his thesis on "Safe Milk Production" and he was an acknowledged authority on this subject. In the Municipal Service he rose to the position of Deputy Director of the Abattoir and Livestock Market Department..

His colleagues at the Abattoir will remember Jimmy for the unfailing courage and dignity with which he bore his long illness. To his wife (née Kathleen Crane) and his family, a daughter and three sons, the profession extends its sincere sympathy.

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IVth INTERNATIONAL CONGRESS ON ANIMAL REPRODUCTION

Physiology, Pathology and Artificial Insemination

To all who are active in the field of reproduction in animals, the undersigned take pleasure in sending you herewith the first particulars of the 4th International Congress on Animal Reproduction.

Place:

The congress will be held in The Hague, seat of the Netherlands Government. Most of the items on the programme will take place in Scheveningen, the seaside resort of international renown adjoining The Hague.

Dates:

During the 3rd International Congress on Animal Reproduction, which was held in Cambridge (England) in 1956, the Dutch delegates were asked if they would organise the next congress in the Netherlands in 1960. Although the Dutch said that in principle they were prepared to offer hospitality to the 4th Congress, it was decided after consultation with the International Committee for Animal Reproduction and Artificial Insemination to postpone this congress for one year, so that members would be able to attend the 8th International Congress of Animal Husbandry, in Hamburg, on June 13, 1961.

Consequently, the dates of the 4th International Congress on Animal Reproduction have been fixed for 5th-9th June, 1961, in The Hague.

Programme:

In the plenary sessions experts from all over the world will be invited to report on a number of subjects which are focussing international interest. Furthermore a considerable number of subjects will be dealt with in section meetings, such as the physiology and pathology of reproduction and artificial insemination. Whereas earlier congresses were devoted almost entirely to the subject of reproduction in mammals, this time room will be made for the problems which arise in the reproduction of poultry.

Papers:

The preliminary reports for the congress will be sent to participants in printed form; if possible, some time before the congress begins.

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CONGRESS LANGUAGES:

The official languages of the congress will be English, French and German. Should the number of participants from Spanish-speaking countries be great enough, Spanish will be added. A simultaneous translation system will be used during the working-sessions, enabling participants to listen directly to the spoken word in the congress language of their choice.

FOLDER:

A folder containing further details of the congress will be published in the spring of 1960. Those interested can apply to the: Secretariat of the 4th International Congress on Animal Reproduction, 14, Burgemeester de Monchyplein, The Hague (Netherlands).

On behalf of the Organising Committee.
Signed: Dr. L. Hoedemaker (Secretary),
Prof. Th. Stegenga (Chairman).

REGIONAL AND APPLIED ANATOMY OF THE DOMESTIC ANIMALS — Part II — THORACIC LIMB by John A Taylor, F.R.C.V.S.; Oliver and Boyd, Edinburgh and London; pp. 205 + viii; 52 figs. Price 27/6 net.

Anatomy, traditionally regarded as a musty subject, gains a stimulating freshness and vitality at the hands of the author of this series. This is due mainly to his approach to the subject matter, engendered, no doubt, by many years of good and sympathetic teaching. The succinct, lucid style is another contributing factor. Ons feels as if led by an enthusiastic guide who thoroughly appreciates both the importance of the features expounded and the requirements of his audience.

Without adopting a condescending tone, the author explains clearly a number of seemingly minor points, which are often omitted in more academic texts and thus prove stumbling blocks to the neophyte. The manner of presentation also allows the student to gain an overall picture, with emphasis on the clinically important aspects. The constant reference to surface anatomy is another excellent feature. Both for introductory reading, assistance during regional dissection, and final review work this book forms a superb teaching aid.

To practitioners, too, this book will prove a boon, especially to those who have neither the time nor the inclination to wade through ponderous texts, only to find themselves still without the essential information sought for at the end.

The dog and horse are adequately dealt with, but one would like to see more on the bovine species than just the brief consideration accorded the manus.

It is sincerely to be hoped that the following volumes — part three on the Pelvic Limb and part four on Thorax and Abdomen — will appear soon.

H.P.A. de B.

PRACTICAL FOOD INSPECTION by C. R. A. Martin, 1959.

This is a work which has been written especially for Inspectors. It covers a vast field including all aspects of food inspection. It is a pity it contains some serious technical errors such as: "Plasma minus its proteins is called serum"; in referring to the intradermal test, he says the tuberculin is injected subcutaneously; in dealing with redwater he describes the red colour of the urine as "haematuria"; in describing the Ziehl-Neelsen method of staining he uses absolute alcohol after counterstaining with methylene blue.

Although this is the case, this work contains a great deal of information of great practical value.

P. J. J. FOURIE.

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